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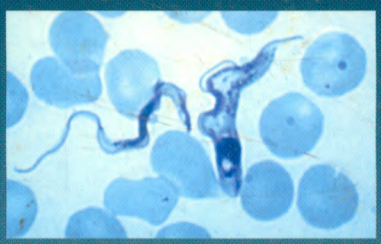
Johannes Kaufmann

Parasitic Infections of Domestic Animals



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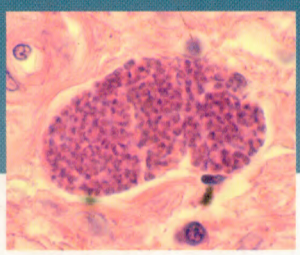

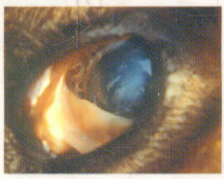

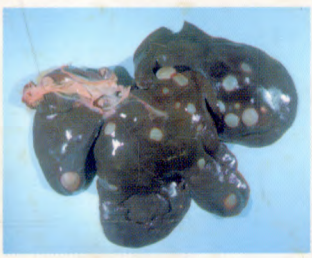

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JOHANNES KAUFMANN

Parasitic Infections of Domestic Animals

A Diagnostic Manual

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Basel · Boston · Berlin

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The author would like to dedicate this manual to his teacher and friend

Prof. Bernd Hörning

Bernd Hörning was Professor of Parasitology at the Veterinary Faculty of Berne, Switzerland. He retired in 1992. His extensive knowledge and commitment to Veterinary Parasitology and his amicable attitude towards students and colleagues should be honored with the present manual.

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Foreword

It is a great pleasure for me to introduce this manual for the diagnosis of parasites of domestic animals. It is a unique publication in many respects. Books concerning parasitic animal diseases – comprehensive textbooks, but also field manuals – abound. These generally deal with aetiology, clinical signs and pathology, as well as control and treatment. However, while methods for diagnosis are often mentioned, these are rarely sufficiently practical and specific to allow the personnel in field laboratories to reach a concise, rapid diagnosis of the parasite species involved in any particular case. *Parasitic Infections of Domestic Animals* succeeds in redressing these shortcomings.

That such a work ever came to fruition at all is to the credit of its principal author Dr. Johannes Kaufmann, head of the Diagnostic Section of the Institute of Parasitology at the University of Berne, Switzerland. In the late 1980s Dr. Kaufmann worked in a field laboratory of the International Trypanotolerance Centre in Gambia. While there he became acutely aware of the need for practical literature on parasite diagnosis. After returning to Switzerland he took on the gargantuan task of compiling such a work himself. Supplementing the material gleaned from his field work, Dr. Kaufmann has brought together a great deal of information from various institutes and universities.

The result is an impressively comprehensive, beautifully illustrated and, above all highly serviceable manual. It is easy to consult. Its systematic and colour-coded layout allows the user to steer swiftly and surely through a vast amount of information. Clinical pictures and figures show the parasite stage of diagnostic interest, facilitating a rapid and accurate diagnosis. The book has been designed to be used by veterinarians and technicians, by teachers and students – in fact, by anyone dealing with the diagnosis of diseases in domestic animals. On another practical level, it will enable meat inspectors and other public health officials to identify parasites of animals which may infect humans. While originally meant for use primarily in Africa, the book's relevance is by no means limited to this continent, since most of the economically important parasites considered in detail (e.g. coccidia of poultry, gastrointestinal nematodes of ruminants, strongyles of horses, *Ascaris* and trichinellosis of swine etc.) occur world-wide.

But what good is a well-conceived manual, intelligently and copiously illustrated if it fails to reach those workers in need of it most? The expense of publishing a work of this type,

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accompanied as it is by approximately 320 color and 400 black and white illustrations is close to prohibitive. That the price has nonetheless been set at a very reasonable level is only possible thanks to generous financial aid from *ciba* Basle, Switzerland, the Swiss Development Cooperation, Berne, Switzerland and the University of Berne, Switzerland. This is the final factor to assuring that *Parasitic Infections of Domestic Animals* will become a well-thumbed bench book in many laboratories world-wide.

Gerrit Uilenberg
Cargese, September 1995

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Introduction

The present manual is intended as a tool for the identification of cosmopolitan parasites affecting domestic animals. Special attention has been paid to imported or autochthonous parasitic infections occurring in African livestock, including dromedaries. In the course of compiling this work, a summary was made of the activities of some 300 institutions (national veterinary health departments, research institutes, universities, private enterprises etc.) dealing with the research, diagnosis and control of parasitic diseases in Africa. Both francophone and anglophone literature has been considered.

Laboratories dealing with the diagnosis of parasitic diseases need to rely on well-furnished libraries in order to cover the vast spectrum of parasites causing disease. Nevertheless, many laboratories, especially those in developing countries, do not have access to such resources.

It is for such situations – when technical information is needed but no library is available – that this manual was designed. It contains, in condensed form, all the information, necessary for a rapid and accurate diagnosis of parasitic infections of domestic animals.

Disease may be initiated either by *adult parasites* or by their *immature stages* and very often the identification of a particular parasite is based on *morphological details*, to which special attention has been paid in this manual. Histopathological sections have been included where pathological alterations need to be elucidated. Indirect diagnosis by immunological techniques is indicated where it plays a practical role. These techniques are described in the initial chapter, METHODS.

Molecular biological techniques have gained an increased significance in the detection of parasitic infections even under field conditions because of their high diagnostic specificity and sensitivity. Furthermore, samples may be collected in the field (e.g. squash blots on to filter papers) and transported easily (often without refrigeration) over long distances to laboratories, where the analysis can be carried out. A special section in the METHODS chapter has been devoted to this highly important and fascinating technology.

Sometimes the species diagnosis is relevant for the control, but very often a genus diagnosis is sufficient. This manual is not intended to serve as a textbook. It is meant to be a technical tool and located on the laboratory bench. The Standard Nomenclature of Animal Parasitic Diseases (SNOAPAD) taxonomy is used in order to allow access to specific literature,

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some of which is mentioned under the section FURTHER READING. Latin names of parasites which are no longer standard in the current scientific literature but still widely in use in certain publications and geographic areas are mentioned as synonyms in brackets.

In order to meet the requirements of laboratories operating under field conditions, a procedure for achieving a rapid and pragmatic diagnostic decision was established. Parasites of cattle, small ruminants, equine, swine, dromedaries and poultry are described in 6 chapters. Each chapter is divided into the 5 stages in which parasites may occur: stages found in the gut and faeces (1), stages found in the blood and circulatory system (■ 2), stages found in the urogenital system (■ 3), stages found in internal organs (■ 4) and stages found on the body surface (■ 5). Under each of these stages parasites are dealt with in a stereotypic manner: protozoa, helminths (trematodes, cestodes and nematodes) and arthropoda (arachnids and insects). Information relevant to each parasite is presented in the following order: scientific name of the parasite (taxon), synonyms, common name of the parasite or the disease caused by the parasite, species description containing biological data, host spectrum, geographic distribution, symptoms, significance of the parasite, diagnosis, therapy and prophylaxis. A numeric key of the source of each illustration is indicated in brackets [ ] at the end of each legend and refers to the BIBLIOGRAPHY. Parasites which are not species-specific are described in the chapter of that particular livestock species in which they play the most important role and a cross-reference (☞) is made to other animal species or organ systems in which they may also occur.

If a genus is described as a group (e.g. *Trichostrongylus* spp.) essential information is provided under "• General features". If several parasite species or genera can be controlled in a similar manner, therapy and prophylaxis may then be indicated for these species or genera collectively. If infections are arthropod-borne, information will be given on the biology of the respective vector.

Rickettsiaceae (*Ehrlichia* spp., *Anaplasma* spp., *Cowdria ruminantium*, *Eperythrozoon* spp. and others) form an important group of "parasite-like" pathogens and are often found in blood or tissue smears made for the detection of blood parasites. Some important species of Rickettsia are described in the present manual for differential diagnosis.

This manual is presented in the hope that it may assist all those colleagues struggling in their daily work with the diagnosis and control of livestock diseases all over the world. The manual wells from a great sympathy for those people "fighting at the front" under difficult field conditions, in regions where human survival is linked to the health of livestock often perilously endangered by a variety of deleterious conditions, among which parasitic infestation may be highly significant.

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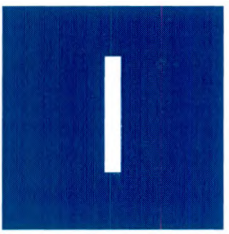

Methods

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1.1 Detection of helminth eggs, larvae, oocysts and sporocysts of protozoa in faeces

Table 1 Methods for the detection of parasite stages in faeces

Method	Detection
Sedimentation	
• in water	Eggs of *Fasciola* spp.; *Paramphistomum* spp.; *Dicrocoelium* spp.; oocysts of *Eimeria leuckarti* (equines)
• in 0.85% saline	Eggs of *Schistosoma* spp.
Flotation	
• with $ZnCl_2$ solution	Eggs of cestodes and nematodes; $ZnSo_4$, (*SG=1.30) oocysts and sporocysts of protozoa except *Eimeria leuckarti* (equines)
• with NaCl solution	Eggs of *Strongyloides* spp.; strongyles, (*SG=1.20) Ascaris spp., *Toxocara* spp., *Oxyuris* spp., *Anoplocephala* spp., *Moniezia* spp., oocysts of protozoa, *Trichuris* spp. and *Capillaria* spp.
Baermann apparatus	Larvae of lungworms, hatched larvae of strongyles and Strongyloides (in older samples)

*SG = specific gravity; concentrations of $ZnCl_2$- and NaCl solutions ☞ Table 2

General rule for the selection of solution for concentrating parasite stages

- saturated NaCl for floating strongyle eggs
- sedimentation in H_2O for trematode eggs
- saturated NaCl (SG=1.204) is a general purpose solution

Table 2: Flotation solutions

Chemical	g to be disolved in 1000 ml of water	SG* at 20°C
NaCl	360	1.2
$NaNO_3$	350	1.2
$ZnCl_2$	436	1.3
$ZnSO_4$ 7 x H_2O	703	1.3
Sugar	550	1.3

*SG= specific gravity

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1.2 Collection and examination of faeces

Faeces intended for parasitological examination should be collected from the rectum, using disposable plastic gloves. The sample is collected into the hand of the glove which is turned inside out, air is expressed and the glove is tied at the wrist. Freshly expelled faeces can also be used for examination as long as it is not contaminated with soil and dirt. This is especially important since many free living nematode larvae may occur in the environment, making a proper diagnosis difficult or impossible. Eggs may develop rapidly to a stage in which it is difficult to identify them.

The detection of parasite stages in faeces from animals suffering from diarrhoea may be markedly impaired. The diagnosis of parasitism by microscopic techniques is more reliable when repeated examinations are made. For example, negative results are sometimes obtained from faecal examinations if the parasites are not mature enough to deposit eggs. This might also occur during the encysted stage of certain parasites, such as strongyles or oesophagostomes. Tapeworm segments often leave the digestive tract without disintegrating, and negative results may be obtained unless segments are detected.

1.3 Direct smear method

It is possible to demonstrate the presence of parasite stages (eggs or larvae of helminths, oocysts of coccidia) by the examination of a thin smear of emulsified faeces. A small quantity of faeces is placed on a slide, mixed with some droplets of water and a cover slip is placed on the fluid. The slide is investigated using a magnification of 50 and 250×. This method, however, suffers from the drawbacks that (a) it is effective only where the concentration of parasite stages is high, (b) it is frequently difficult to identify them since they are partially covered by detritus and (c) quantitative results cannot be obtained.

Parasites found with the direct smear method:

- coccidia species and helminth eggs (only when high numbers of oocysts or eggs are present);
- cestode and trematode eggs (mainly in birds)

1.4 Flotation method

Separating the eggs from faecal debris by floating them on a variety of solutions. When faeces are emulsified in liquids of high specific gravity and either centrifuged or allowed to stand, the worm eggs and many protozoan cysts float to the top while the heavy coarse debris settles to the bottom. The top film can then be removed and examined. Nematode and cestode eggs float in a liquid with a specific gravity of between 1.10 and 1.20. Trematode eggs, which are much heavier, require a specific gravity of 1.30–1.35.

Procedure

1. Place a lump of faeces about the size of a walnut (5–10 g or a few faecal pellets of sheep or goats) in a shell vial, cup or glass.

2. Add approximately 50–100 ml of tap water and mix thoroughly with a spatula until all the faecal material is broken down.

3. The mixture is poured through a wire mesh screen with an aperture of 500–800 µm to remove large lumps. The strained fluid is caught in a bowl. The screen is rinsed with water and the debris left on the screen is discarded.

4. Transfer the suspension to a conical measure and fill with tap water to the top allow to settle for 30 min.

5. Discard the supernatant carefully to a remaining sediment of approx. 10 ml.

6. The sediment is stirred and a sample of 2 ml is poured into a centrifuge tube.

7. The tube is placed in the centrifuge. With a pipette, saturated NaCl is added until a convex meniscus stands above the top of the tube.

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8. A thick 19 x 19 mm square cover glass is placed on the tube, ensuring that no bubble is trapped under it. The cover glass may additionally be coated with a solution of egg white / glycerol (1: 1) to improve adherence of the eggs.

9. The tube is centrifuged at 2000 rpm for 2–3 min. (In the absence of a centrifuge let the sample stand for 30 to 60 min.)

10. Pick off the cover glass and place the sample on a slide and examine under a microscope by using a magnification of 32–100×.

Parasite stages found with the flotation method: eggs of cestodes and nematodes; larvae of lungworms; oocysts of coccidia (except *Eimeria leuckarti*). Trematode eggs are not satisfactorily detected with the flotation method (☞ Sedimentation method).

1.5 Sedimentation method

Heavy parasite eggs sink to the bottom of solutions used in the flotation techniques.

Procedure

1.–5.: ☞ Flotation method (1.4)

6. Add water to a point a little below the top of the measure and allow to settle again for 3 min.

7. After sedimentation the supernatant is again drawn off, a few drops of methylene blue (5%) added and the sediment screened in aliquots of 5 ml, using a low power stereomicroscope. Any trematode eggs are readily visible (brown or yellow colored) against the pale blue background.

Parasite stages detected with the sedimentation method:

- eggs of *Fasciola* spp., *Dicrocoelium* spp., *Paramphistomum* spp., *Schistosoma* spp., *Gastrodiscus* sp.;
- larvae of lungworms;
- oocysts of *Eimeria leuckarti* (equines)

1.6 McMaster method

This technique is used where the number of eggs or larvae per gram of faeces should be counted.

Procedure

1. Weigh 3 g of faeces and add 42 ml tap water.

2. Homogenize and pour suspension through a 250 µm aperture sieve, collecting the filtrate.

3. Collect the filtrate, agitate and fill a test tube of 15 ml volume.

4. Centrifuge at 2000 rpm for 2 min.

5. Pour off the supernatant, agitate sediment and fill tube to previous level with flotation solution.

6. Invert tube 6 times and remove fluid with pipette to fill both chambers of McMaster slide quickly.

7. Examine one chamber and multiply number of eggs or larvae under one etched area by 100, or two chambers and multiply by 50 to arrive at the number of eggs per gram (epg) of faeces.

Remark: It is impossible to calculate from the epg the actual worm population of the host. Nevertheless, egg counts in excess of 1000 are generally considered indicative of heavy infections. However, a low epg is not necessarily indicative of very low infections, since one part of the worm population may just have started to reach patency but may already cause disease. Alternatively, the epg may be affected by developing immunity. The eggs of some species, such as certain ascarids, *Strongyloides*, *Oxyuris*, *Trichuris* and *Capillaria*, can be easily recognized morphologically. However, with the exception of *Nematodirus* spp., the common trichostrongyle eggs require more detailed analysis (measurements) for differentiation. The diagnosis "strongyle egg" is usually sufficient for practical purposes.
(Figure 1)

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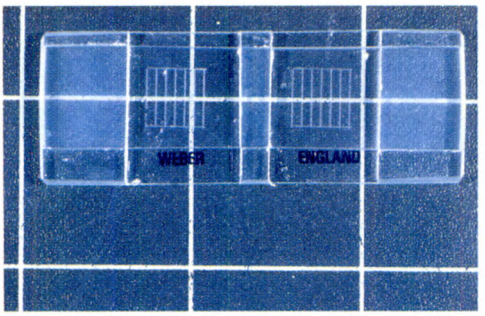

Fig. 1
McMaster chamber for the quantification of parasite eggs in faeces

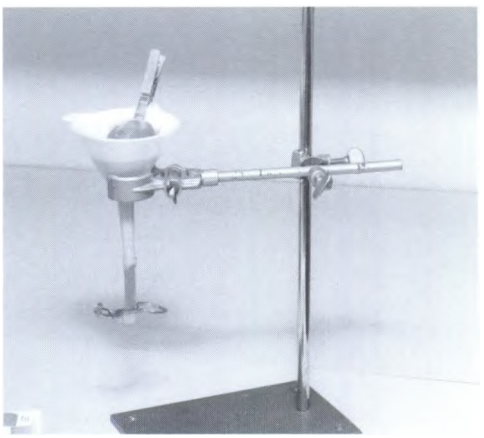

Fig. 2
The Baermann apparatus consists of a funnel held in a retort stand. A rubber tube attached to the bottom of the funnel is constricted by a clip. Faeces are wrapped in gauze and attached with a clothes-peg to the funnel. The larvae leave the faeces, migrate through the gauze to the sediment

1.7 Baermann method

Larvae of lungworms (third-stage larvae of gastrointestinal nematodes) migrate actively into the aqueous phase of the Baermann apparatus. This consists of a glass or plastic funnel held in a retort stand. A rubber tube attached to the bottom of the funnel is constricted by a clip. A sieve (aperture 250 µm) is placed on top of the funnel, which has been partially filled with water. A double layer of gauze is placed on top of the sieve. Faeces are placed on the gauze, and the funnel is slowly filled with water until the faeces are immersed. The apparatus is placed over night at room temperature, during which the larvae migrate out of the faeces and through the sieve to sediment in the neck of the funnel. The clip on the rubber is then opened and the water in the neck of the funnel collected in a small beaker for microscopic examination in a Petri dish.
(Figure 2)

A simple adaptation of the above method is to suspend the faeces enclosed in gauze in a urine glass filled with water and leave over night. The larvae will leave the faeces, migrate through the gauze and settle at the bottom of the glass. The sediment is examined under the low power microscope as above.

Parasite stages detected with the Baermann method:

- Larvae of lungworms (*Dictyocaulus* spp., *Muellerius* spp., *Cystocaulus* spp., *Protostrongylus* sp., *Metastrongylus* spp., *Crenosoma* spp.)
- larvae of strongyles

Cave: When faecal samples were collected from the ground, masses of free living nematodes may leave the faeces and appear in the sediment of the Baermann funnel.

1.8 Culture and recovery of third-stage larvae

Faecal culture provides an environment suitable for the hatching of helminth eggs and for their development to the infective stage. The general method described below is suitable for the culture of trichostrongylid or strongylid eggs in the faeces of ruminants, pigs and horses.

Procedure

The faeces should be moist and crumbly but not really wet. Very dry faeces should not be used for culture. If the faeces are very wet, then charcoal, peat moss or vermiculite should be added until the correct consistency is obtained.

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1. Faeces are broken up finely using a spatula.

2. Wide-mouthed glass or plastic jars are filled with the mixture, closed with the lid and incubated at 27°C for 7 or at 20°C for 10–20 days. Fungi are sometimes troublesome but indicate that the conditions in the culture are ideal. The growth of fungi can be reduced by stirring the culture each day.

3. Larvae are best recovered using the Baermann apparatus. The culture is removed from the incubator and the faeces are tipped out of the culture dish onto the double layer of gauze of the Baermann apparatus (+ above), distributing it as evenly as possible over the whole area.

4. The culture vessel (lid, jar, dish, etc.) is washed with a small quantity of water. The water is then tipped into the Baermann apparatus.

5. The Baermann apparatus is allowed to stand for 24 hours, or at least over night, during which time the larvae will have migrated through the screen into the bottom of the funnel from where they may be drawn for examination.

1.9 Identification of third-stage larvae of nematodes

Procedure

1. Larvae are killed by the addition of Lugol's iodine to the larval suspension on a microscope slide. Iodine kills the larvae and stains the free-living nematodes yellow while parasitic third-stage larvae remain unstained. A coverslip is placed on the prepared suspension before examination.

2. Microscopic examination at a magnification of 60–80 ×. Some details may only be seen at a magnification of 250 ×.

3. Parasitic larvae appear translucent and almost colourless while free-living nematodes are stained yellow with the iodine. Parasitic larvae show a well defined cuticular sheath (with few exceptions). Characteristics which permit differentiation of the different larvae are shape and internal structure of the head region, number and shape of the intestinal cells, relative size of the sheath tail and shape of the larvae's tail. Lungworm larvae, especially *Dictyocaulus viviparus*, are usually coiled.

1.10 Staining *Cryptosporidia* spp. in faecal smears

Procedure

1. Air-dry thin faecal smears and pass quickly through a flame.

2. Stain with Ziehl-Neelsen's carbol-fuchsin solution (Merck Diagnostica no. 9215) for 2 min.

3. Rinse with tap water.

4. Rinse for a few seconds with acid alcohol (3% hydrochloric acid in 70% ethanol).

5. Rinse again with tap water.

6. Counter stain with Brilliant Green (0.5%; Sigma no. B-6756) for 2 min.

7. Rinse again with tap water.

8. Air-dry the slide. Microscopic examination at 1000×, using immersion oil. Cryptosporidia appear as bright red granules on a blue-green background (☞ CATTLE, 1).

Alternative method

3 µl diarrhoeic faeces and 3 µl carbol-fuchsine (Merck no. 9215) are mixed, and a thin smear is made. After air-drying, the slide is examined at a magnification of 1000 ×, using immersion oil. The oocysts measure 4–5 µm in diameter and appear as thick-walled, red granules.

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1.11 Scotch tape method

Procedure

1. A transparent scotch tape, approximately 4 cm in length is pressed onto the skin of the anus and then placed on a microscopic slide.

2. Microscopic examination at a magnification of 32–100 ×.

Remark: This method is widely used to detect eggs of oxyurids (*Enterobius vermicularis*, *Oxyuris equi* etc.) in animals and man.

1.12 Sodium Acetate Formaldehyde (SAF) method

Procedure

1. Fixation of 2–5 g faeces in SAF solution (15 g sodiumacetate, 20 ml of a 100% acetic acid, 40 ml of a 40% formaldehyde and 925 ml water).

2. Mix thoroughly and pass suspension through a gauze (placed in a funnel) into a tube.

3. Centrifuge at 2000 rpm for 1 min.

4. Discard supernatant to a remaining sediment of 1 ml.

5. Resuspend the sediment with 7 ml of NaCl phys.

6. Add 2 ml of ether, cover the tube with a rubber and mix the contents vigorously.

7. Centrifuge at 2000 rpm for 3 min.

8. Discard the supernatant carefully and transfer some droplets of the sediment onto a microscopic slide. Place a coverslip onto the sample.

9. Microscopic examination at a magnification of 400 ×.

Remark: This method is widely used to detect protozoan parasites (*Entamoeba* spp., *Giardia* spp., *Balantidium* spp., *Eimeria spp.*) in the faeces.

2 Examination of material for *Trichinella spiralis*

2.1 Demonstration of encysted larvae of *T. spiralis* in muscle fibre (Compressorium method)

Procedure

0.5–1.0 g muscle, usually pillars of the diaphragm, are subdivided into small pieces approximately 3 × 10 mm and crushed firmly between pieces of plate glass. For this purpose a product called "compressorium" is available on the marked. The samples are compressed heavily with thumb screws. The samples should be almost transparent for the examination at a magnification of 50 × with a dissecting microscope. Larvae of *T. spiralis* are infective to man. Great care must be taken in handling infected or suspect material.
(Figure 3)

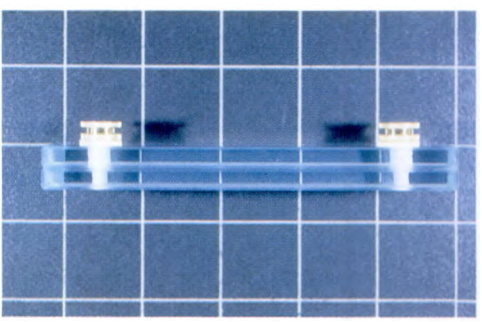

Fig. 3
Compressorium method: Samples of muscles are cut into small pieces and crushed between two glass plates. The samples are compressed heavily with thumb screws until they are almost transparent

2.2 Digestion of muscle for the release of encysted larvae of *T. spiralis*

Procedure

1. The 100 g sample is finely chopped in a chopper/blender or cut manually into small pieces.

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2. Transfer the chopped material to a 3 l beaker. Add 10 g of pepsin, dissolved in 2 l of tap water, to give an activity of 2000 FIP (= Fédération Internationale Pharmacie); 30,000 units FB (British Pharmacopoeia) or 10,000 NF (US Nat. Formula).

3. Add 16 ml hydrochloric acid (25%) and place a magnetic stirrer rod in the beaker.

4. Place the beaker on a heated magnetic stirrer which has been pre-set to maintain a temperature of the solution at 44–46°C.

5. After 30 min digestion transfer the contents of the beaker through a mesh screen (aperture of 180 µm) into a 2-l separating funnel. The debris in the screen is gently washed with a fine spray of water in order to remove larvae which may have been trapped on the screen.

6. Sediment for 30 min and run off 40 ml of the sediment into a 50 ml measuring cylinder. Allow to settle for 10 min, then siphon off 30 ml of the supernatant fluid. The sediment is resuspended with a further 30 ml of water and allowed to sediment for 10 min before 30 ml of the supernatant fluid is siphoned off.

7. Pour the remaining 10 ml into a ruled Petri dish (parallel lines 5 mm apart). Dilute the suspension with tap water if it contains a lot of undigested particles.

8. Examine the sediment using a dissecting microscope at a magnification of 20–50 ×.

Remar: Predilection sites are tongue, masseter, intercostal and diaphragm muscles. 10 g samples of each animal are collected and digested. When many animals need to be examined, it is recommended to take 10 g sample of diaphragm pillar tissue from each animal and bulk these to form composite samples with a maximum of 100 g.

Alternative method (for digestion if there is no heatable magnetic stirrer available):

The digested fluid and the tissue are placed in a 3l Erlenmeyer (conical) flask which is maintained at 37°C in a water bath or in an incubator. In this case the flask is shaken every 30 min for 4–5 h.
Remark: The use of pre-prepared and preheated pepsin-HCl solution may be easier and reduce the digestion period markedly.

3 Haematology

3.1 Bloodsmear and Giemsa-stain

Procedure

1. Apply a small drop of blood from a microhaematocrit capillary tube or a pipette to the slide approximately 20 mm from one end.

2. Place a spreader on the slide at an angle of 20–30°C. Draw it back to make contact with the blood.

3. Allow the blood to run to each end of the spreader. Spread the blood along the slide in a fairly rapid but smooth motion.

4. Quickly air-dry the slide and label it with a pencil or a diamond tipped pencil.

5. Fix the thin smear for 1 min in methyl alcohol.

6. Place it in an inverted position in Giemsastain (1.5 ml in 15 ml distilled water buffered to pH 7.2) for 30 min.

7. Wash the slide with water and drain in an upright position to dryness.

8. Examine with a microscope at a magnification of 100 and 1000 ×, using immersion oil. (Figure 4)

3.2 Diff-Quick®

This is a rapid staining method for blood smears.
The Diff-Quick® (Baxter Dade AG, 3186 Dü-

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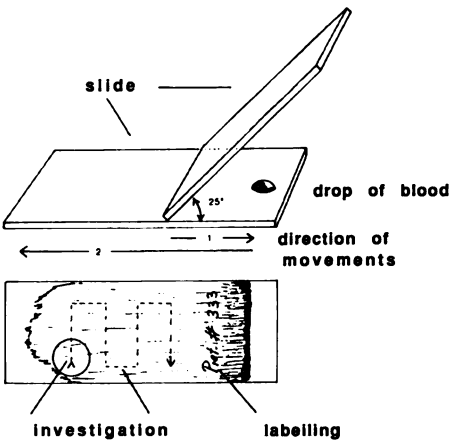

Fig. 4
Preparation of a bloodsmear. A small drop of blood is applied to the slide approximately 20 mm from one end. A spreader (or another clean slide) is placed on the slide at an angle of 20-30° and drawn back to make contact with the blood. The blood is allowed to run to each end of the spreader. The blood is spread along the slide in a fairly rapid but smooth motion [57].

Fig. 5
A good quality high power microscope is an important tool in every diagnostic laboratory. A standard equipment should contain the following magnifications: 32 × 100 ×, 400 × and for the investigation of blood smears 1000 × with oil immersion.
Rapid staining methods for bloodsmears are of great practical use in the routine diagnostic laboratory. The previously air-dried smears are fixed and stained in only 15 s by three consecutive brief immersions in the pre-prepared staining solutions.

dingen, Switzerland) staining set contains solutions for rapid staining of blood smears, yielding results comparable to the Pappenheim technique (Giemsa-May-Grünwald). By consecutive brief immersions in the Dade Diff-Quick Solutions, the previously air-dried smears are fixed and stained in only 15 s. As samples, freshly collected EDTA blood, but also smears from biopsies and other material may easily and quickly be stained with this method.
(Figure 5)

3.3 QBC®

A screening method for rapid detection of blood parasites (*Babesia* spp., *Trypanosoma spp.*, microfilariae etc.)
Originally developed for the diagnosis of malaria, the QBC® (Becton Dickinson, One Becton Drive, Franklin Lakes, N.J. 07417) is also a highly sensitive and rapid method for the diagnosis of blood parasites in animals. The QBC method concentrates the parasitized cells into a narrow zone of the micro blood tube that is examined under a fluorescence microscope. The test capillaries are internally coated with acridine orange and anticoagulants. Expansion of the centrifugally separated cell layers is achieved with a plastic float. With the QBC method, blood parasites may be detected and identified within a few minutes. The method is dependent on a micro capillary centrifuge and a fluorescence microscope.

3.4 Dark ground / phase contrast buffy coat method

Procedure
1. Determine the PCV by the microhaematocrit method.

2. Cut the microhaematocrit tube with a diamond tipped pencil one mm below the buffy coat to include the top layer of red cells.

3. Express the contents of the capillary tube onto a clean slide, mix and cover with a coverslip.

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4. Examine the preparation using phase contrast or darkground microscopy at a magnification of 100–400×.

Remark: A number of 10–20 fields should be examined. *Trypanosoma* spp. species may be identified using characteristics listed in Table 7. Giemsa-stained smears can be prepared from buffy coat. Microfilariae are commonly found with the dark ground buffy coat method.

4 Entomology

4.1 Skin scraping

Procedure

1. Scrapings are taken from the affected area by means of a scalpel or a "sharp-edged spoon". The area selected for scraping should be at the edge of a visible lesion and the hair over this area should be clipped away. It is useful to moisten the skin with liquid paraffin so that scrapings adhere to the scalpel. Scraping should be continued until a slight amount blood oozes from the surface.

2. The material is transferred to a slide and a drop of oil is added.

3. A coverslip should then be applied and the sample examined under a low magnification (100 ×).

Remark: If during this initial examination no mites are detected a further sample may be heated on a slide with a drop of 10% KOH. After allowing this preparation to clear for 5–10 min it should be re-examined.

5 Immunological methods

5.1 Introduction

Many parasites of veterinary importance stimulate the production of antibodies in the blood of the host which can be detected, using appropriate serological techniques. The sero-diagnosis is dependent on a specific antigen-antibody response.

A number of serological techniques are available for diagnosis of parasitic infections for routine diagnosis. The indirect fluorescent antibody test (IFAT) is among the most important and most simple techniques for the indirect detection of *Babesia equi*, *B. caballi*, *B. divergens*, *B. bovis*, *Toxoplasma gondii* and *Neospora* spp. infections. The IFAT will be described in detail below.

5.2 Indirect Fluorescent Antibody Test (IFAT)

The test depends upon a reaction between a particulate antigen and immune serum. The globulins of the latter are made visible under ultra-violet light after reaction with fluorescein-labelled anti-species serum (conjugate). The test may be carried out on sera from a variety of hosts for which suitable conjugates are available.
(Figure 6)

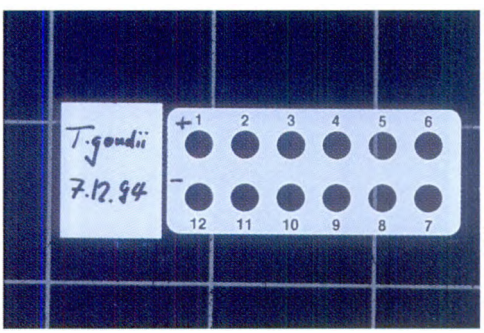

Fig. 6
Antigen is fixed onto defined areas of commercially available IFAT slides. For each individual test a positive and a negative control should be included. In addition, a PBS control may be used to indicate reaction of the conjugate with the antigen directly. Diluted test sera are applied in 10 µl spots onto the slide.

***Preparation of antigen slides for Babesia* spp.:**

1. Blood from a highly parasitaemic animal (between 5–20% of the erythrocytes should be infected) is collected into heparinised tubes.

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2. The blood is washed 3 times in phosphate buffered saline solution* (PBS; pH 7.2), centrifuging for 10 min at 1000 rpm between each wash. The supernatant and white cell layer are removed.

3. Two volumes of PBS are added to the packed red cells; the mixture is agitated.

4. If special IFAT slides are available 10 µl of erythrocyte suspension is applied to each hole. If normal slides are used smears are to be made which evenly cover the whole slide.

5. The smears are air-dried** and then wrapped carefully in aluminum foil, sealed in an airtight plastic bag and stored at –20°C (storage up to 1 year) or –80°C (storage for 2–3 years).

** Phosphate buffered saline solution (PBS) pH 7.2: 87.8 g NaCl, 10.91 g Na_2HPO_4 anhydrous, 2.77 g NaH_2PO_4. Make this up to 1 l with distilled water. Dilute 1/10 with distilled water before use.*

*** Some antigens such as* Toxoplasma gondii *and* Neospora *spp. tachyzoites should be fixed in acetone for 5 min before freezing.*

Procedure (IFAT)

1. Allow antigen slides to reach room temperature.

2. Mark out the slides (if necessary), using a diamond pencil.

3. Prepare serum dilutions (1/20 up to 1/2560) in PBS (pH 7.2).

4. Apply 10 µl spots to the slide, including a positive, a negative and a PBS control.

5. Incubate in a moist chamber at 37°C for 30 min.

6. Rinse with PBS and wash twice with PBS, 5 min each. Allow to dry.

7. Apply fluorescein-labelled conjugate at correct dilution* (1/100 –1/1000). Incubate (in dark) in a moist chamber at 37°C for 30 min.

8. Rinse with PBS and wash twice with PBS, 5 min each. Allow to dry.

9. Mount in 90% glycerol in PBS.

10. Examine under UV illumination at a magnification of 400 ×. Sera dilutes at 1/80 (depending on the particular antigen) and above showing strong fluorescence of the parasites are usually considered to be positive. Special attention should be paid to the pattern of fluorescence in the test sera compared with the controls. This demands experience of the observer!

The following conjugates should be used according to the host species:

- Horse: anti-horse immunoglobulin
- Cattle: anti-bovine immunoglobulin

Individual batches of antigen and conjugate should be titrated against each other using control sera to optimise the conjugate dilution.

5.3 Enzyme linked immunosorbent assay (ELISA)

The antigen is attached to the plastic wells. The antibodies in the test serum when present attach to the antigen. The retained antibody is detected by an anti-species serum conjugated to an enzyme (mostly alkaline phosphatase) which in the presence of a suitable substrate produces a colour reaction, the intensity of which is proportional to the antibody titer. The ELISA is one of the most commonly used serological tests today. The test is dependent on well defined antigens and controls. For the precise reading of the intensity of the colour reaction may be assessed by a spectrophotometric determination (ELISA reader). In veterinary parasitology the ELISA technique is used for *Trichinella spiralis*, *Hypoderma bovis* and *Toxoplasma gondii*.

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5.4 Immunoblotting (Western blot)

Antigen samples are separated in an analytical gel, for example an SDS polyacrylamid gel. The resolved molecules are transferred electrophoretically to a nitrocellulose membrane in a blotting tank. The blot is then treated sequentially with an antibody to the specific antigen, and washed, and then a radiolabelled conjugate to detect antibodies is bound to the blot. The principle is similar to that of ELISA. After washing again, the blot is placed in contact with an X-ray film in a cassette. The autoradiograph is developed and the antigen bands which have bound the antibody are visible. The technique can be modified for use with an enzyme coupled conjugate, and the bound material can be detected by treatment with a chromogen which deposits an insoluble reagent directly onto the blot.
(Figure 7)

5.5 Complement Fixation Test (CFT)

Complement is fixed during the reaction between some antigens and antibodies. By conducting this reaction in the presence of measured amounts of complement and then detecting the remaining unfixed amount with a separate antigen-antibody reaction, a quantification

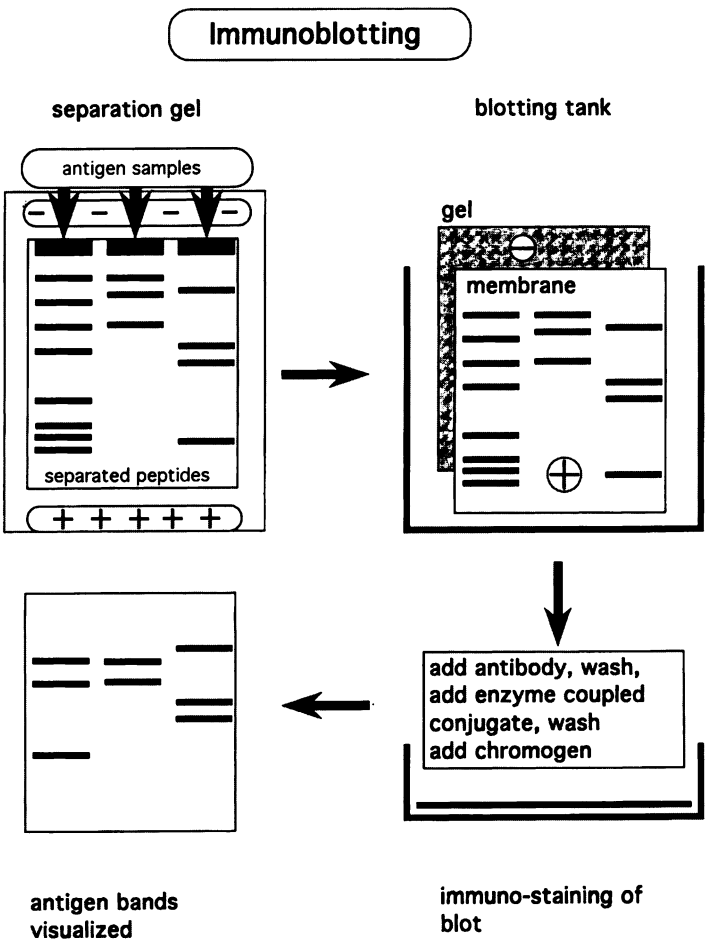

Fig. 7
Immunoblotting:
schematic procedure

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of the complement fixing activity of the original serum is obtained. The CFT is required by many national authorities as an import control of horses with regard to *Babesia equi*, *Babesia caballi* and *Trypanosoma equiperdum*, although it is less sensitive than the IFAT. More and more, the CFT is replaced by IFAT and ELISA.

6 Molecular biological techniques

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6.1 Introduction

A microscope, a well-trained eye and the application of immunodiagnostic tools adapted for the field will, by and large, cover the needs of the person in the field involved in the routine diagnosis of parasitic infections. If easy techniques fulfill diagnostic requirements, then there is indeed every reason to avoid more sophisticated technology. Fieldworkers have established valuable connections to local authorities and animal owners and are well aware of the factors that influence parasite infection and transmission in this area. In this capacity, they function more and more as an important interface between the field and research laboratories. However, epidemiological studies are often called for and it is especially in this area that molecular biology techniques have started to find widespread applications. For this reason, acquiring an elementary insight into recent developments in molecular diagnostics is of interest to all who deal with the diagnosis of parasitic infections.

Sensitivity and specificity are the two most important criteria that define the quality of a diagnostic technique. We are faced with several situations where parasite detection can be very difficult. The parasite often eludes detection by microscopy because the organisms, or the cells infected by them, have been sequestered to parts of the body from which samples cannot easily be taken without invasive manipulations; the number of parasites in circulation can therefore be extremely low. In eradication programs or epidemiology studies, it is important to monitor carrier-status, the presence of reservoirs and the infection rates of transmitting vectors. This requires the direct demonstration of parasites, since the presence of antibodies does not allow a distinction between past and current infections. New DNA technologies now provide valuable tools for solving these problems since they can both detect and identify parasites.
Specificity becomes crucial whenever a distinction needs to be made between pathogenic and non-pathogenic parasites that are closely related. Cross-reactivity in diagnostic tests with antigens of unrelated parasites can also occur. Considerable efforts have gone into developing new techniques that allow the problems of sensitivity and specificity to be addressed. The rapid spread of hybridoma technology over the last two decades has allowed considerable progress in fine-tuning serological techniques and monoclonal antibodies have become the cornerstone of many diagnostic kits. Over the last decade, molecular biology has also become a routine technology and its application has stimulated rapid progress in the development of reagents that are capable of identifying a single unicellular parasite.

6.2 Recombinant antigens

In many diagnostic tests, the detection of anti-parasite antibodies in the serum of infected or immune animals depends on the availability of specific antigens that have to be purified from the organism. The parasite from which the antigen is to be purified must be available in large quantities, which is often difficult to achieve, and expensive in vitro culture or large numbers of experimental animals are often required to generate sufficient starting material. In addition, significant differences in yield, purity and quality can arise from batch to batch. Using the technique of gene cloning, it is now possible to isolate parasite genes encoding a single antigen and to introduce these genes into bacteria, yeast

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or mammalian cells, where they can be expressed in large quantities as a recombinant antigen. It is also possible to "engineer" these antigens and remove those parts that are responsible for cross-reactivity with other organisms. This way, a continuous and reliable source of diagnostic antigens is guaranteed and increased specificity can also be achieved. Importantly, this technique also applies for antigens of parasites which can never be maintained in vitro or collected in large enough quantities for antigen production.

6.3 Detection and identification of parasites using nucleic acid probes

The use of immunodiagnostic methods for the detection of parasites has certain limitations. Demonstrating antibodies against a parasite provides little information on whether the parasite is still present in the animal's body and in primary infections, disease will mostly develop well before antibodies become detectable. For this reason there is a need to be able to detect parasites directly. Several highly sensitive and specific techniques have been developed that rely on the demonstration of parasite nucleic acid sequences. These techniques depend either on the use of DNA probes or on the polymerase chain reaction (PCR). Both will be discussed below.

6.3.1 Techniques based on DNA probes

The genetic material (genome) of parasites that are considered distinct species show significant differences, which one could call their "genomic signature", allowing them to be distinguished from each other. Technologies now exist that enable the isolation and identification of such specific sequences, resulting in the generation of DNA probes which can be used in a variety of assays. DNA probes can be long, consisting of several thousands of nucleotides or be limited to as little as 20 or 30 nucleotides, also called oligonucleotides.

A deciding factor contributing to sensitivity is the number of times that the target sequence occurs in the genome. Ribosomal genes are usually present in large numbers and for this reason the development of DNA probes and investigations on phylogenetic relationship between parasites have often relied heavily on finding species-specific differences in the ribosomal genes. Repetitive sequences which are not derived from the ribosomal genes, however, are equally useful and have also been used for species identification.

In order to allow detection, parasite DNA needs to be immobilised on a nitrocellulose or nylon membrane. Alkaline denaturation causes the separation of the DNA strands, making the sequences accessible and allowing the DNA probe to bind to the target sequence by a process called hybridisation. Binding of the probe to parasite sequences can be demonstrated by labelling it prior to hybridisation.

In past years, the detection of nucleic acids used to depend almost entirely on the use of radioactively labelled probes. More recent developments, however, have led to the appearance of a wide range of labelling kits in which colour reactions or chemiluminescence are used to demonstrate specific hybridisation. This new technology offers many advantages. The expensive laboratory set-up required for the safe handling, storage and disposal of radioactive substances has become unnecessary. Importantly, the problem of radioactive decay of probes is now eliminated and specific reagents can be stored and used over a long period of time in a standardised way. Indeed, with a minimum of equipment and imagination, DNA probes could be used under the most primitive field-laboratory conditions.

There are several easy ways in which parasite nucleic acids can be transferred to membranes. Droplets of fluid to be tested for the presence of parasites (such as blood, lymph, spinal fluid or stool samples) can simply be applied to membranes and left to dry in a process called "dot-blot". In many cases, drying is sufficient to expose the DNA. Parasites that are surrounded by a strong cyst wall, however, will require processing prior to application to a membrane. Parasite-containing vectors, such as infected tsetse flies, ticks, snails etc., can also be squashed onto membranes ("squash-

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blots"). "Touch-blots" made from lesions have also been used for the diagnosis of *Leishmania* infection, where parasite strain-identification is important for deciding on the strategy of treatment which is often associated with severe side-effects. Using recombinant DNA technology, probes have now been developed which also allow different *Leishmania* strains or geographical isolates to be distinguished from each other.

Membranes on which material has been blotted can be dried and stored for a long time before analysis. This makes it possible to collect many samples during separate field trips and then to process them all together upon return to the laboratory. Figure 8 shows a squash blot prepared from snails in which different larval stages of *Fasciola hepatica* could be detected using a DNA probe which hybridises to repetitive DNA fragments present in the genome of *F. hepatica*. This example illustrates another advantage of using techniques that depend on detecting genomic sequences. Whereas the morphology and the antigenic repertoire of the different life-cycle stages of a parasite can vary extensively, the parasite genome is very stable. Morphological identification, especially when mixed infections occur or when many samples need to be examined,

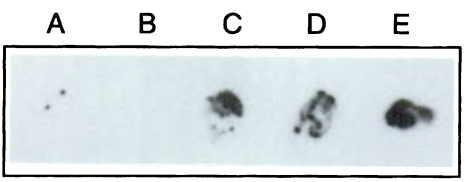

Fig. 8
Detection of isolated larvae of *Fasciola hepatica* and of larval stages in snail 'squash blots'. In position A, two *F. hepatica* rediae, collected from an infected snail, were deposited onto the filter; in position B, an uninfected snail was squashed onto the filter (no signal detectable) and in C to E, whole snails infected with *F. hepatica* were crushed onto the filter. The filter was hybridised with a ^{32}P-labelled DNA probe (Fhr-IV) which hybridises to repetitive DNA fragments present in the genome of *F. hepatica*. The filter was exposed overnight to X-ray film.

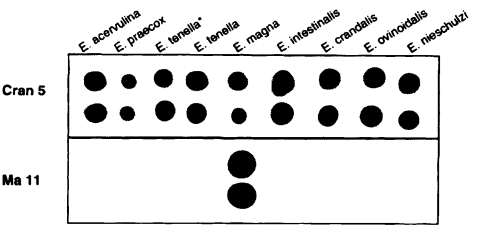

Fig. 9
Specific detection of *Eimeria magna* DNA by 'dot blot analysis'. DNA was isolated from different *Eimeria* species and spotted in duplicate onto two filters. Each dot contains approximately 10 nanograms of genomic DNA. One filter was hybridised with the 32P-labelled DNA probe, Cran 5, which detects repetitive sequences of all *Eimeria* species. The second filter was hybridised with the probe, Ma11, which hybridises exclusively with *E. magna*. The *E. tenella* isolate labelled with (*) also contained *E. acervulina*.

can be difficult and time-consuming and may require expertise which is not always available. Species-specific probes can thus often help to circumvent problems associated with the identification of parasites by light microscopy. An example for the use of a species-specific DNA probe which is capable of distinguishing *Eimeria magna* from several other *Eimeria* species by 'dot blot' analysis is shown in Figure 9.

When treated with a minimum of care, parasite nucleic acids are more stable than most antigens on which immunodiagnostic techniques depend. For a more detailed analysis of field samples and the characterisation of new strains, especially within the context of epidemiological surveys, it is often necessary to collect and store parasites. Most samples can be stored indefinitely in 70% ethanol/1mM EDTA and intact DNA can easily be analysed at a later point. Such analysis involves the extraction of DNA from samples, digestion with a number of empirically chosen restriction enzymes, separation of the DNA fragments by electrophoresis, transfer by capillary blotting to a membrane and hybridisation with specific probes (-called southern blot hybridisation). Figure 10 summarizes this procedure and Figure 11 demonstrates how a specific probe is capable of dis-

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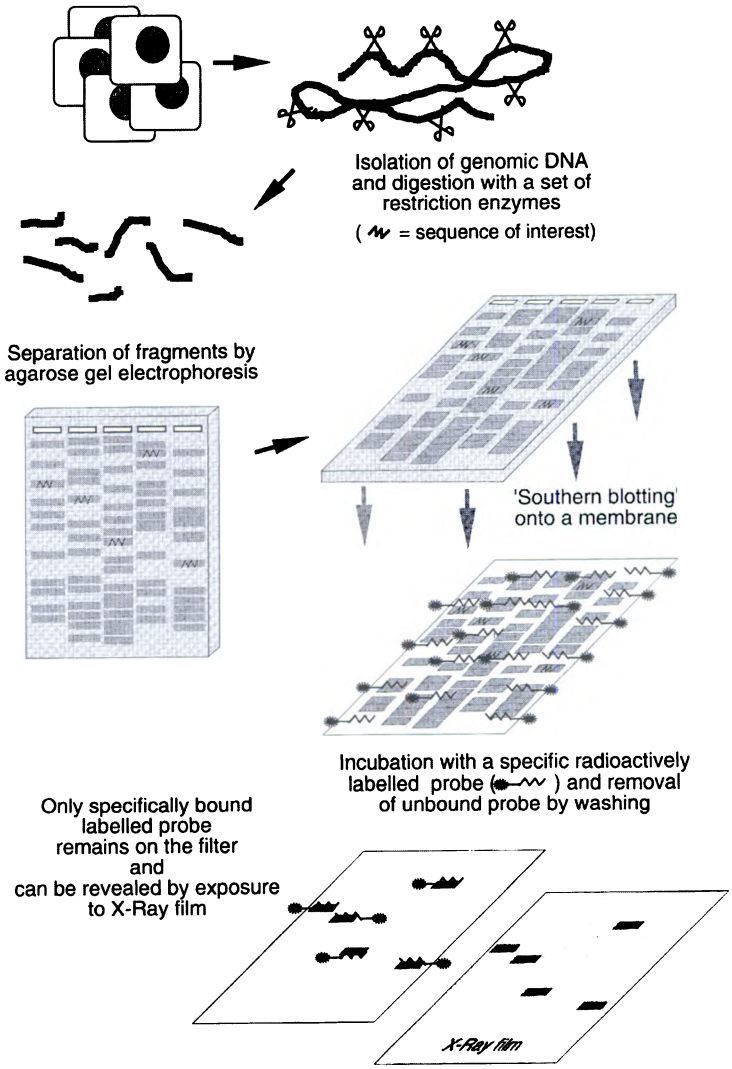

Fig. 10

The principle of Southern blot analysis. Genomic DNA is extracted from an organism (here shown as a collection of cells) and digested with a restriction enzyme that cuts the genomic DNA into many fragments of different sizes. These DNA fragments can be separated, based on their different sizes, by electrophoresis in an agarose gel. The separated fragments are then blotted onto a nylon or nitrocellulose filter by a procedure called southern blotting. The DNA is fixed onto the filter by baking the filter or by UV-cross-linking. The filter is subsequently incubated with a radioactively labelled DNA probe, specific for the sequence of interest, which will hybridise to homologous sequences present on the filter. Unbound probe is washed away and only specifically bound probe remains on the filter. Specific hybridisation of the radioactive probe can be visualised by exposing an X-ray film to the filter (autoradiography). Black bands will appear where the probe hybridised to the filter.

Remark: Probes can now also be labelled enzymatically; incubation with the enzyme's substrate then results in the generation of either a colour reaction on the filter or chemiluminescence, which can be detected by X-ray film.

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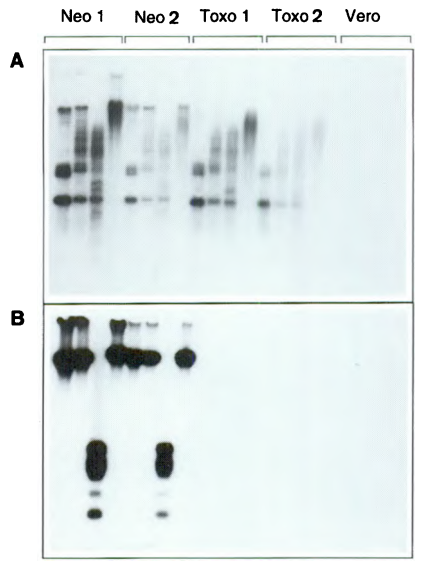

Fig. 11
Southern blot analysis of DNA isolated from two Neospora and two Toxoplasma isolates. Genomic DNA was extracted from two different Neospora isolates (Neo 1 and Neo 2) and two different Toxoplasma strains (Toxo 1 and Toxo 2). Each DNA sample was digested with four different restriction enzymes, BamHI, HindIII, HaeIII and PstI. As a control, DNA was also isolated from the Vero cell line in which the parasites were propagated in culture. The DNA fragments were separated by agarose gel electrophoresis and blotted onto a filter. The filter was hybridised first with a DNA probe which detects both Neospora and Toxoplasma DNA sequences and exposed to an X-ray film (Panel A). The similarity in the pattern of hybridisation demonstrates the close relationship between the two organisms. The probe was then removed from the filter by process called 'stripping' and the filter subsequently rehybridised with a Neospora-specific probe (Panel B). The specificity of this probe is demonstrated by the fact that no hybridisation can be observed where the Toxoplasma DNA was blotted onto the filter. Note that none of the probes hybridises to the DNA isolated from the Vero cells.

tinguishing *Neospora* from the morphologically virtually identical *Toxoplasma*.

6.3.2 Detection of parasites by the polymerase chain reaction (PCR)

Despite the fact that species-specific probes tend to detect highly repeated sequences in the genome, the limits of detection are still in the nanogram range (for *Eimeria* spp., this would correspond to approximately 10^3-10^4 oocysts). The development of the polymerase chain reaction (PCR), however, has opened up extensive possibilities for the detection of parasites and pathogens in general. This technique was originally made possible by the discovery of a temperature-resistant DNA polymerase (Taq polymerase) which was isolated from *Thermus aquaticus*, an organism found in hot springs. The PCR requires a special piece of equipment known as a "thermocycler". A PCR reaction mixture consists of the DNA sample to be tested, two specific oligonucleotides, called primers, a temperature resistant DNA polymerase and a mixture of nucleotides which form the building blocks for DNA synthesis. The oligonucleotide primers are designed to match and bind at two well-defined sites of a known parasite DNA sequence. The distance between the oligonucleotide binding sites is exactly known (e.g. 800 bases), The principle of a PCR reaction is summarised in Figure 12. The double-stranded DNA in the sample is first made single stranded (melted) by incubation at high temperature (about 95°C). The reaction is then cooled down to a lower temperature (e.g. 55°C) at which the primers can bind to the specific target sequences in the DNA sample. The temperature is then raised again to 72°C at which the polymerase synthesizes two new strands of DNA using the specific primers as the starting point and the specific target DNA fragment as a template. These three events, melting, primer annealing and DNA synthesis constitute one cycle. By continuously repeating this cycle, the polymerase synthesizes increasing numbers of copies of the target sequence and the fragment is thus "amplified" so that it can easily be detected by agarose gel electrophoresis. Starting with

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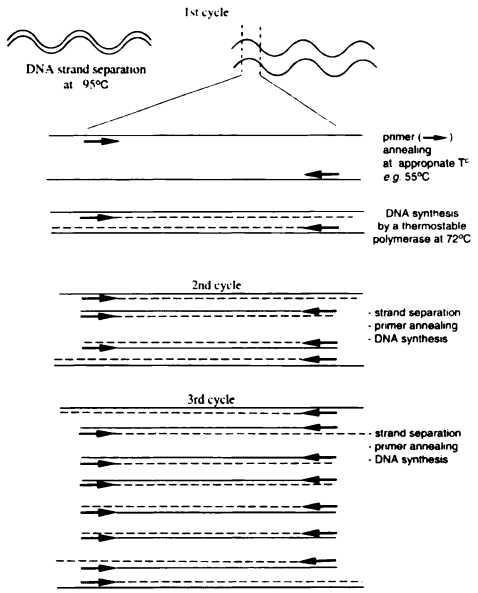

Fig. 12
The principles of PCR

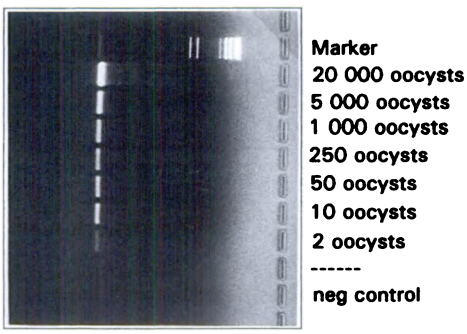

Fig. 13
Detection of *Eimeria tenella* oocysts by PCR. DNA was extracted from decreasing numbers of E. tenella oocysts ranging from 20,000 oocysts to 2 oocysts. Sequences corresponding to the intergenic region between the 5S ribosomal genes were amplified by PCR. The amplification reactions were loaded onto an agarose gel. After electrophoresis, the amplified DNA fragments (expected size: 560 base pairs) were visualised by ethidium bromide staining. No amplification product can be observed when PCR was performed on a sample without DNA (negative control) (reproduced from Stucki et. al. *Experimental Parasitology* Vol. 76: 68-75).

one molecule, 10^9 molecules can theoretically be generated in 30 cycles. This way, using a minimal amount of parasite DNA as starting material, specific parasite DNA fragments can be amplified and little as 10 picograms of parasite DNA are now sufficient to detect the parasite (in the case of *Eimeria* this would be less than 10 oocysts). Figure 13 shows how an *E. tenella*-specific DNA fragment can be amplified from as few as two oocysts.

6.3.3 Random amplification of polymorphic DNA (RAPD)-PCR

A further method which is presently being developed in several parasite systems and which also depends on PCR is known as RAPD analysis (random amplification of polymorphic DNA). In this procedure, a single oligonucleotide primer with an arbitrary sequence can be used to amplify a set of characteristic fragments from genomic DNA by PCR. The principle of this method is that the oligonucleotide will, by chance, bind to the DNA template. If a second binding site also occurs on the complementary DNA strand within a reasonable distance (usually not more than 2-3 kilobase pairs) the stretch of DNA between them will be amplified. To increase the chances of obtaining amplification products, the PCR is performed under conditions which allow imperfect matches between the primer and the template DNA. Because there is no bias towards repetitive sequences, this method requires considerably more genomic DNA (approximately 50 ng) than when repetitive sequences are the target, but it has the advantage of being able to detect and distinguish between different strains of a particular parasite as well as different species.

Certain precautions are essential, however, when PCR technology is applied. Because of its extreme sensitivity, the risk of contamination and generating false positives are extremely high. For this reason the use of PCR for diagnostic purposes requires a special infrastructure, the implementation of strict rules and the continuous monitoring of possible contamination by including controls in all experiments.

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6.4 Concluding remarks

Molecular biology has contributed considerably to the development of new diagnostic tools, often bringing sensitivity to a level where a single parasite can be detected. Molecular cloning and the production of recombinant proteins has led to an expansion of the range of parasites for which diagnostic antigens can be produced. As is the case for all diagnostic tests, however, it is important to realise that the use of specific probes or specific primers for PCR implies that they only provide a positive identification of the parasite for which the probes or the primers were designed. They provide no information on the presence of other parasites, whether related or not, which could be present in the sample. RAPD-PCR meets this requirement to some extent in that it allows the detection of different parasites or parasite strains, provided the organism is presents in the sample in sufficient numbers. Therefore, however sensitive and specific the technologies used, all results need to be interpreted with care and considered in the right context.

6.5 Glossary of terms

Cloning
Insertion of a DNA sequence into a plasmid or viral DNA vector and propagation of the recombinant DNA in a bacterial host.

Genomic DNA
The combined genetic material of an organism composed of DNA.

Hybridisation
Binding of labelled DNA (probe) to target DNA or RNA because of homology in sequence.

Polymerase chain reaction (PCR)
A process that rapidly amplifies DNA sequences; the technique relies on the separation of the DNA strands at 95°C, the annealing of specific primers to the DNA at lower temperature and the synthesis of new strands by a temperature sensitive DNA polymerase; this cycle is repeated 20 to 30 times.

Primer
A short stretch of nucleotides (also called oligonucleotide) which is capable of binding to a sequence containing matching complementary bases.

Probe
Radio- or chemically labelled DNA fragments that hybridise to homologous target DNA sequences and permit their visualisation.

Restriction enzyme
Endonuclease that reproducibly cleaves double-stranded DNA at specific, well-defined recognition sites.

Southern blot analysis
A procedure (developed by E. Southern) whereby target DNA fragments are analysed. DNA is transferred from an electrophoresis gel to a membrane by capillary action and visualised by hybridisation with a labelled DNA fragment (probe). The same technique performed with RNA is called Northern blotting.

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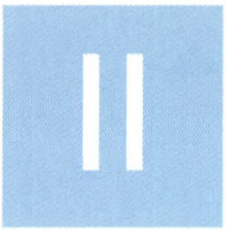

Parasites of Cattle

CONTENTS

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PROTOZOA

- Protozoa oocysts found in the faeces

Eimeria spp. Bovine coccidiosis

Location: Terminal ileum, caecum, colon
Hosts: Cattle
Species description: Of the 21 species of *Eimeria* that infect cattle, *E. bovis* and *E. zuerni* are most often associated with clinical disease. Coccidiosis is commonly a disease of young cattle and occurs especially in management systems (night holding place, limited water source, paddocks) which concentrate the hosts and the parasites within a restricted area. Bloody diarrhoea, progressive weight loss and death, especially in animals after weaning can result from heavy infections. Ruminants do normally excrete a few *Eimeria* spp. oocysts. The condition called coccidiosis includes both severe dysentery and excretion of a high number of oocysts.
Geographic distribution: World-wide
Symptoms: Diarrhoea lasting for a few hours, followed by a self cure or development of severe dysentery accompanied by a haemorrhagic and viscous diarrhoea, dehydration and paralysis. Death can occur rapidly, mainly in calves. Another form of coccidiosis is characterized by persisting, non-haemorrhagic diarrhoea with continuous weight loss until cachexia. This condition may last for several weeks. Animals that survive severe illness can have significant weight loss that is not quickly regained, or can remain permanently stunted.
Significance: *E. bovis* and *E. zuerni* are most commonly involved in clinical coccidiosis of cattle.
Diagnosis: Clinical signs and extremely high numbers of oocysts per gram of faeces (50,000–500,000).
Therapy: The drugs that are commonly used to treat clinical coccidiosis in ruminants are listed in Table 1. These include sulfonamids, nitrofurazone, amprolium (10 mg/kg po. > 10 days), monensin (1 mg/kg po. for 10–30 days) and lasalocid (0.5–1 mg/kg po. for up to 6 weeks). Sulfadimidine (33% solution for intravenous injection), sulfamethazine (50–100 mg/kg, po. daily for 4 days), sulfaquinoxaline (15 mg/kg, po. for 3 days) may be used to treat sick animals. Sulfaguanidine is less effective than the soluble sulfonamides which can be administered orally or parenterally. Sulfonamides, combined with trimethoprime can also be used to treat clinical coccidiosis. Toltrazuril (1 × 20 mg/kg) is highly effective to treat clinical coccidiosis in ruminants.
Prophylaxis: Young animals should be kept in clean and dry quarters, and watering devices should be protected from faecal contamination. Decoquinate, amprolium and ionophorous antibiotics can be used in cattle for prophylaxis (Table 3). Neonates should receive colostrum within 6 hours after birth and stress (e.g. weaning, sudden change in feed, etc.) should be minimized.
(Figures 14, 15, 16, 17, 18, 19, 20, 21, 23, Table 3)

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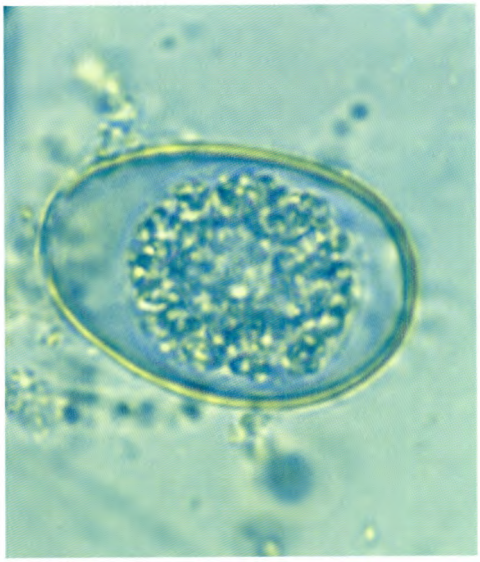

Fig. 14 Oocyst of *Eimeria bovis*
(26–32 × 18–21 µm)

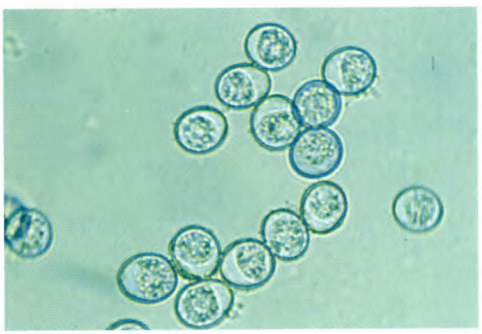

Fig. 15 Oocysts of *Eimeria zuerni*
(16–20 × 15–18 µm)

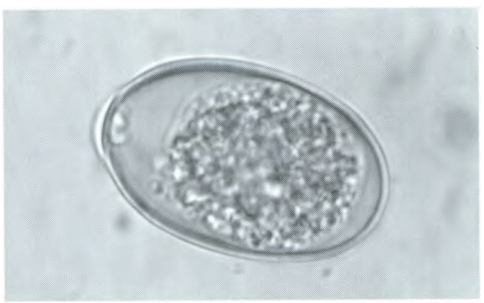

Fig. 16 Oocyst of *Eimeria brasiliensis*
(33–42 × 23–30 µm)

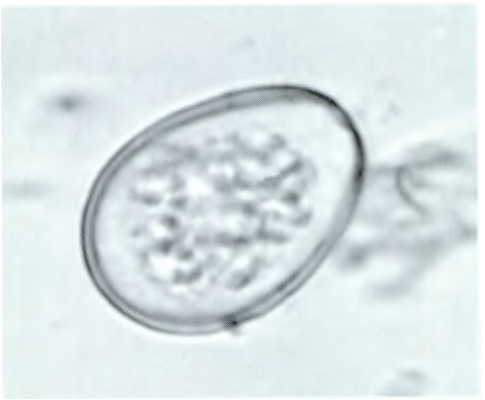

Fig. 17 Oocyst of *Eimeria alabamensis*
(16–24 × 12–16)

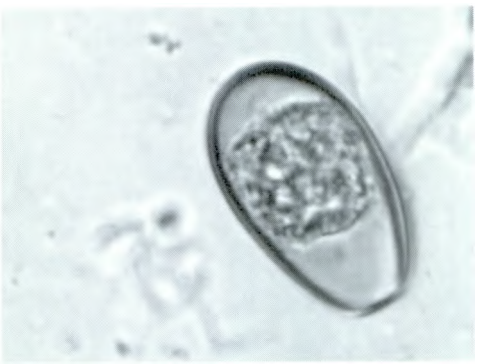

Fig. 18 Oocyst of *Eimeria auburnensis*
(36–42 × 19–26 µm)

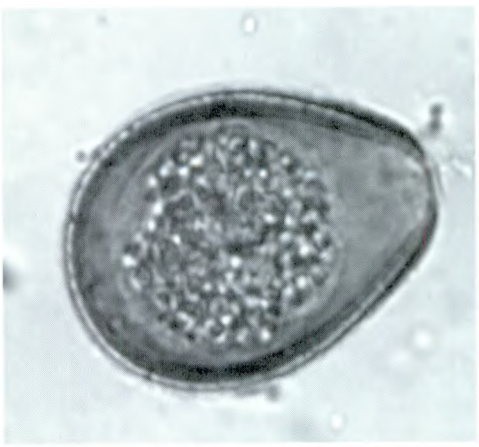

Fig. 19 Oocyst of *Eimeria bukidnonensis*
(44 × 32 µm)

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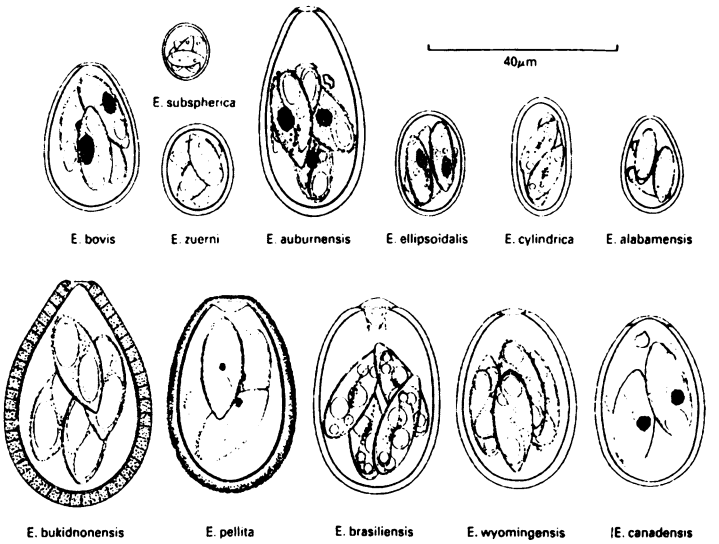

Fig. 20 Coccidia found in cattle [1]

Fig. 21 Bull with an acute coccidiosis

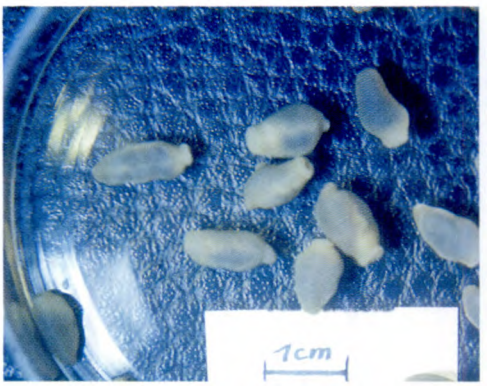

Fig. 22 Rumen flukes found in the rumen of N'Dama cattle

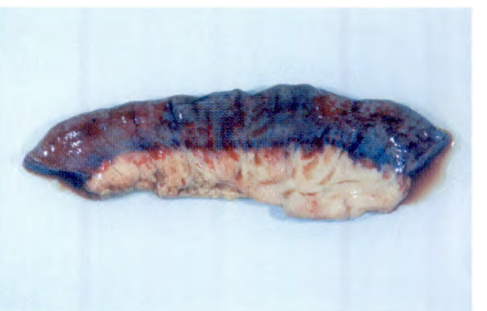

Fig. 23 Haemorrhage in the small intestine of a calf with an acute coccidiosis (*Eimeria bovis*)

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Table 3 Recommended use, dosage and dosage form of anticoccidial drugs used in cattle, goats, sheep, and swine

Drug[1]	Use	Animal	Dosage	Dosage forms
Amprolium	Therapeutic	Cattle	10 mg/kg daily for 5 days	20 % soluble powder
		Sheep	50 mg/kg daily for 5 days	9.6 % solution
		Goats	100 mg/kg daily for 5 days	Feed additive crumbles
		Swine	25-65 mg/kg once or twice daily for 3-4 days	
	Prophylactic	Cattle, sheep, goats	5-10 mg/kg daily for 21 days	
		Swine	25 mg/kg in piglets for first 3-4 days of life	
Sulfonamides				
Sulfamethazine	Therapeutic	Cattle, sheep, goats	50-100 mg/kg daily for 4 days	Soluble powder or bolus
Sulfaquinoxaline	Therapeutic	Cattle, sheep, goats	15 mg/kg, po.[2] daily for 4 days	9.6 % liquid soluble powder and feed additive
Sulfaguanidine	Prophylactic	Sheep	0.5-3 g per lamb per day for 20 days	
		Swine	60 mg/kg for 7-10 days	
Ionophorous antibiotics				
Monensin	Prophylactic	Cattle, sheep & goats	1 mg/kg for 30 days	Feed additive
Lasalocid	Prophylactic	Cattle, sheep & goats	0.5-1 mg/kg per day for up to 6 weeks	Feed additive
Other compounds				
Nitrofurazone	Therapeutic	All	10-20 mg/kg daily for 5 days	88.9 % m/m
Decoquinate	Prophylactic	Cattle	0.5 mg/kg in feed for at least 28 days	6 % premix for addition to feed
Toltrazuril	Therapeutic	Sheep	20 mg/kg, single treatment	25 % liquid
Diclazuril	Therapeutic	Sheep, goats	20 mg/kg, po.	2.5 % suspension

[1] Several trade names, [2] po. = orally; [2]

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Cryptosporidium parvum (syn. *C. bovis*)

Location: Small and large intestine
Hosts: All domestic animals
Species description: Spherical oocysts 4.5–5 µm in diameter are passed in the faeces. Especially calves younger than 8 weeks may be affected, showing different degrees of diarrhoea, associated with oedema and hyperplasia of the mesenteric lymph nodes. Cryptosporidial diarrhoea in both immunodeficient and non-immunodeficient human beings have been reported.
Geographic distribution: World-wide
Symptoms: Persistent diarrhoea in calves 5–35 days old that does not respond to therapy.
Significance: *Cryptosporidium parvum* is pathogenic for calves, sheep, pig, rat and man. Bovine cryptosporidiosis is a zoonosis. Diarrhoea due to *Cryptosporidium parvum* alone is often mild and self-limiting, although the severity may be related to the general strength of the calf and the density of the pathogen in the environment and the intensity of the exposure to the organism. Combination of the infection with rota- and/or coronavirus are common and result in persistent diarrhoea, emaciation and death.
Diagnosis: Oocysts may be demonstrated in Ziehl-Neelsen's carbol-fuchsin stained smears of diarrhoeic faeces (☞ METHODS, 1.10).
Therapy: No effective specific treatment known. Supportive therapy, such as rehydration and antibiotics may help in mild cases. Chemoprophylaxis of bovine cryptosporidiosis can be made experimentally with halofuginone lactate (60–120 µg/kg/day, po. for 7 days) or by paromomycin (100 mg/kg/day for 11 days).
Prophylaxis: Animals with a massive oocyst excretion should be separated from other animals and man. Contamination with faeces should be avoided. Oocysts are resistant to many disinfectants (exception 5% formalin). Emphasis should therefore be on regular removal of contaminated faecal material.

Note: Trophozoites of *Giardia bovis* (small intestine) and *Entamoeba bovis* (rumen, intestines) occur world-wide in the faeces of cattle, but are considered to be non-pathogenic.
(Figures 24, 25)

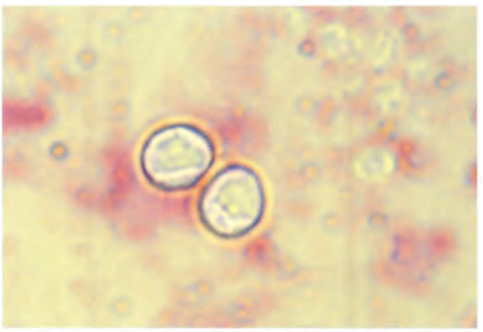

Fig. 24 Oocysts of *Cryptosporidium parvum* (4.5–5 µm) stained with carbol-fuchsine (☞ METHODS 1.10)

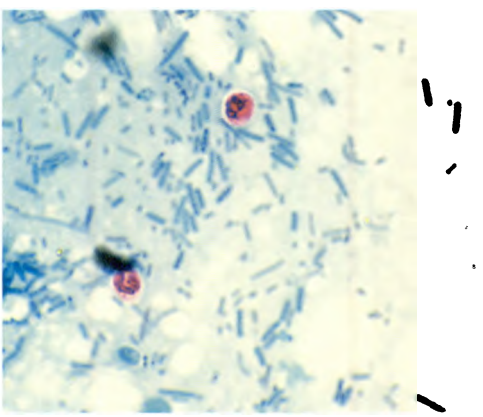

Fig. 25 Oocysts of *Cryptosporidium parvum* (4.5–5 µm) stained with the modified Ziehl-Neelsen's carbol-fuchsine (☞ METHODS, 1.10)

Buxtonella spp.

Remarks: This is a protozoan parasite found occasionally in the large intestine of cattle. Oocysts are found in the faeces. The parasite is non-pathogenic.
(Figure 26)

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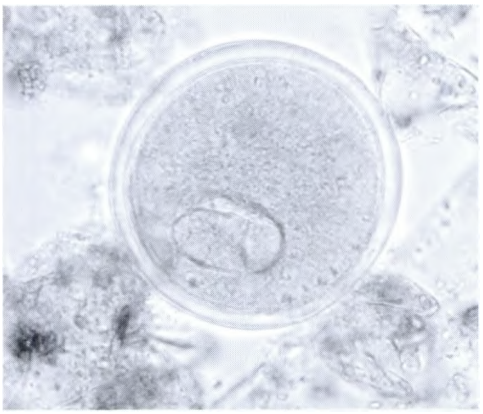

Fig. 26 Cyst of *Buxtonella* sp. (50–131 µm)

HELMINTHS

- **Trematoda eggs found in the faeces and adult trematodes living in the gastrointestinal tract**

PARAMPHISTOMATIDAE

Location: Adult flukes in the rumen; immature flukes in the small intestine

Hosts: Domestic and wild ruminants

Species description: A great number of species of this family has been described in Africa. Indirect life cycle with development in freshwater snails (*Bulinus* spp.; *Planorbis* spp.). The cercariae encyst on herbage which is in contact with water and develop into the infective stages (metacercariae). Infection is acquired by ingestion of herbage contaminated with metacercariae, especially around water holes. The adult flukes are generally non-pathogenic (>80% of adult cattle are infected in endemic areas). Disease occurs when masses of immature worms attach to the intestinal mucosa after excystment, causing destruction and inflammation. Peaks usually at the end of the dry season. Game animals, using the same water holes, may be a source of reinfection. Standing water, troughs and other water bodies are preferred habitats of the intermediate hosts.

Geographic distribution: World-wide

Symptoms: Enteritis with extensive diarrhoea, hypoproteinaemia, weakness during the intestinal phase, when immature worms irritate the small intestinal mucosa. Severe symptoms mainly in young stock. Irregular rumination and progressive degeneration of the animal's condition may be found. Infections with adult rumen flukes are generally inapparent, despite the high numbers of adult parasites in the rumen. Rumenitis may occur due to adult flukes, especially if *Carmyerius* spp. are involved. Adult flukes are generally non-pathogenic.

Significance: Severe diarrhoea and weakness, generally caused by the immature intestinal flukes, may lead to death.

Diagnosis: This is based on the demonstration of immature flukes in the diarrhoeic faeces and the presence of previous cases in the area.

Cave: In acute paramphistomidosis the large, clear, operculated eggs may not be found in the faeces. In many areas > 80% of the adult ruminants pass paramphistome eggs in the faeces. The presence of eggs without clinical signs does not necessarily indicate paramphistomidosis.

Therapy: Niclosamide (90 mg/kg po.), resorantel (65 mg/kg po.) and closantel are active against immature forms. Triclabendazole (12 mg/kg) and albendazole (10 mg/kg) may also be used against immature flukes. Resorantel is active only against mature flukes (☞ Table 4).

Prophylaxis: Avoiding infection by supplying the herds with clean water, e.g. bore holes or raised, snail-free troughs.

Table 4 Recommended drugs against *Paramphistomum* spp. infections in cattle

Drug	Dosage (mg/kg)	Immat. flukes	Mature flukes
Niclosamide	160.0*	+	
Oxyclozanide and Levamisole	18.7 19.4*	+	
Oxyclozanide	15		+
Resorantel	65		+

*at 3-day intervals

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Remarks: Rumen flukes in Africa belong to the families of Paramphistomatidae (*Bothriophoron bothriophoron*; *Stephanopharynx compactus*; *Cotylophoron cotylophorum* and other *Cotylophoron* spp.; *Paramphistomum* spp. [8 species] and *Cotylophoron* spp.; [*C. daubneyi* and *C. microbothrium* are the most frequent species]) and Gastrothylacidae (*Carmyerius* spp. [*C. spatiosus*; *C. papillatus*; *C. dollfusi* [Zebu only in Madagascar]).
(Figures 27, 28, 29)

Fig. 27 Rumen flukes found in the rumen of N'Dama cattle

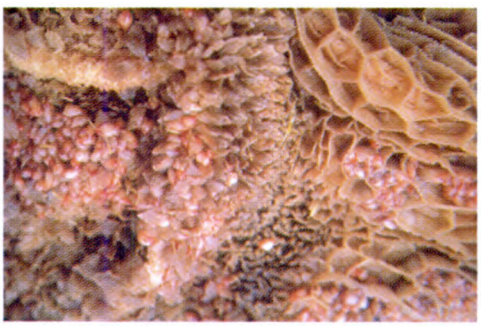

Fig. 28 *Paramphistomum* sp. attached to the rumen mucosa

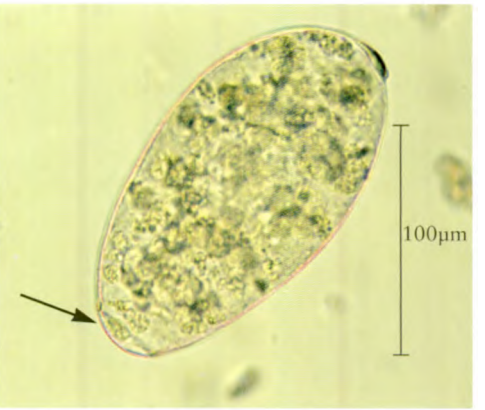

Fig. 29 Egg of a rumen fluke (Paramphistomatidae)

F. giganticca Tropical large liver fluke
(☞ CATTLE, ■ 4.2)

F. hepatica Large liver fluke of temperate areas and high altitude regions in East Africa

Location: Adult flukes in biliary ducts; eggs in the faeces (☞ CATTLE, ■ 4.2)
(Figures 30, 31)

Dicrocoelium hospes African small liver fluke
(☞ CATTLE, ■ 4.2)

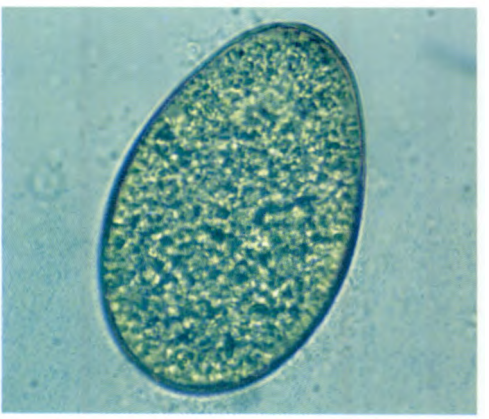

Fig. 30 Egg of *Fasciola hepatica* (130–150 × 63–90 µm)

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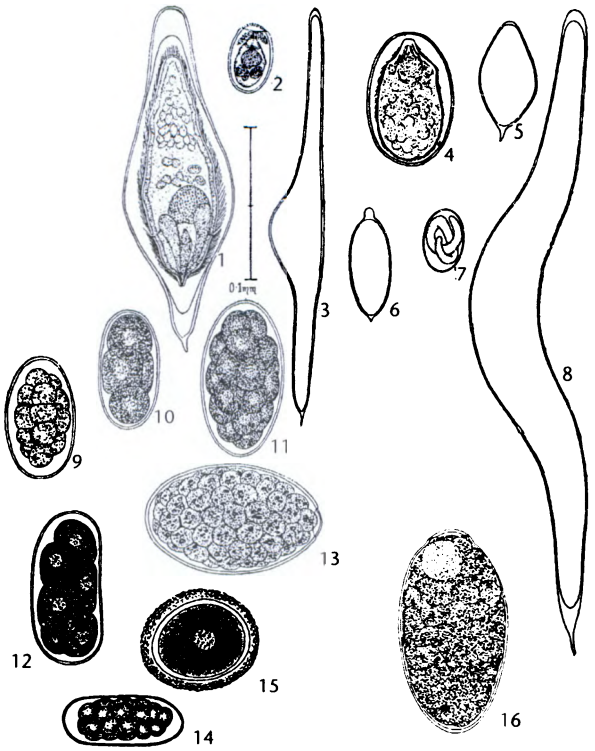

Fig. 31 Eggs of helminth parasites of cattle (not drawn completely to scale) [3]
(1) *Schistosoma bovis*, (2) *Eurytrema pancreaticum*, (3) *Schistosoma spindale*, (4) *Schistosoma japonicum*, (5) *Schistosoma indicum*, (6) *Ornithobilharzia turkestanicum*, (7) *Thelazia rhodesi*, (8) *Schistosoma nasalis*, (9) *Oesophagostomum radiatum**, (10) *Mammomonogamus laryngeus*, (11) *Mecistocirrus digitatus*, (12) *Bunostomum phlebotomum**, (13) *Carmyerius spatiosus*, (14) *Cooperia pectinata**, (15) *Toxocara vitulorum* and (16) *Fasciola hepatica*.
* Strongyle-type eggs are difficult to differentiate in routine examinations

D. dentriticum Small liver fluke

Location: Adult flukes in biliary ducts; eggs in the faeces (☞ CATTLE, ■ 4.2)
(Figures 32, 33)

Fig. 32 Egg of *Dicrocoelium dendriticum* (36–45 × 25 µm)

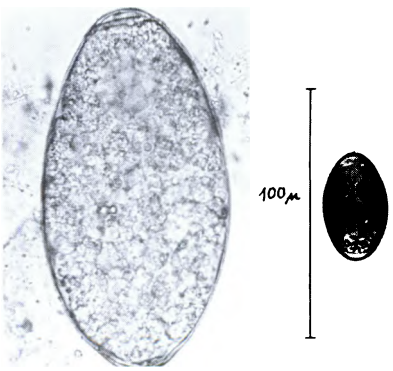

Fig. 33 Egg of *Fasciola hepatica* (a) and *Dicrocoelium dendriticum* (b) [4]

Eurytrema pancreaticum Pancreas fluke

Location: Adult flukes in pancreatic ducts; eggs in the faeces (☞ SHEEP AND GOATS, ■ 4.5)
(Figure 34)

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Fig. 34 Egg of *Eurytrema pancreaticum* (40–50 × 23–34 µm)

Schistosoma spp. (S. bovis, S. mattheei, S. curassoni) Blood flukes

Location: Adult flukes in mesenteric veins; eggs in the intestinal wall and faeces (☞ CATTLE, ■ 2)

(Figures 35, 36)

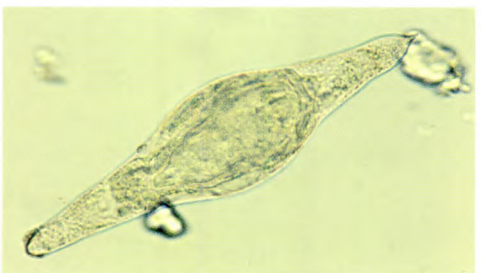

Fig. 35 Egg of *Schistosoma bovis* (180 × 60 µm)

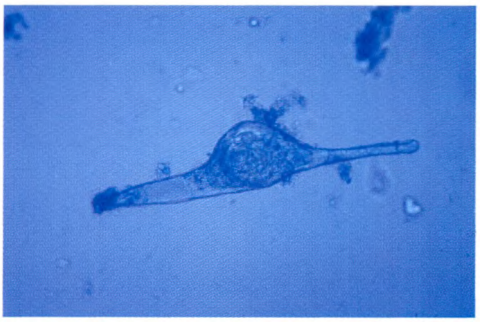

Fig. 36 Eggs of *Schistosoma spindale* (300 × 80 µm) [4]

- **Cestoda eggs found in the faeces and adult cestodes living in the gastrointestinal tract**

(Figure 37)

Moniezia expansa and *Moniezia benedeni* Common tapeworms

Location: Small intestine

Hosts: Cattle, sheep, goat and many other ruminants

Species description: Worms are up to 6 m long and 1.6 cm wide (*M. expansa*). *M. benedeni* which occurs more often in cattle is broader (up to 2.6 cm). The life cycle of *Moniezia* is indirect, including many species of oribatid mites as intermediate hosts. Ruminants are infected by ingestion of the

Characteristics	*Moniezia expansa*	*Moniezia benedini*	*Avetellina spp.*	*Thysaniezia giardi*	*Stilesia hepatica*
Length	1–6 m	26 mm	3 m	2 m	20–50 cm
Width	16 mm	26 mm	3 mm	12 mm	2 mm
Egg	Semi-triangle	Diamond-shaped	Oval	Oval	Round
Genitalia	Double	Double	Single	Single	Single
Interproglottid glands	Follicular	Compact	-	-	-
Paruterine organs in gravid segment	-	-	One: lemon-shaped	Numerous: onion-shaped	Two: round

Fig. 37 Morphological identification of common cestodes in ruminants [5]

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infected mites with herbage. The prepatent period is 35–40 days. Infections with *Moniezia* spp. may be found in more than 50% of a particular population. *M. expansa* eggs are triangular, contain a pyriform apparatus and measure 56 × 67 µm. *M. benedeni* eggs are cuboid, with a thick, ornamented shell and measure 75 µm in diameter.

Geographic distribution: World-wide (Figures 38, 39, 40, 41, 42)

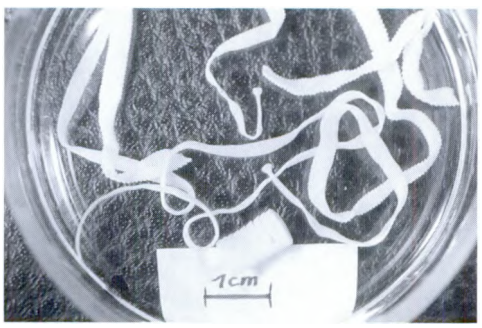
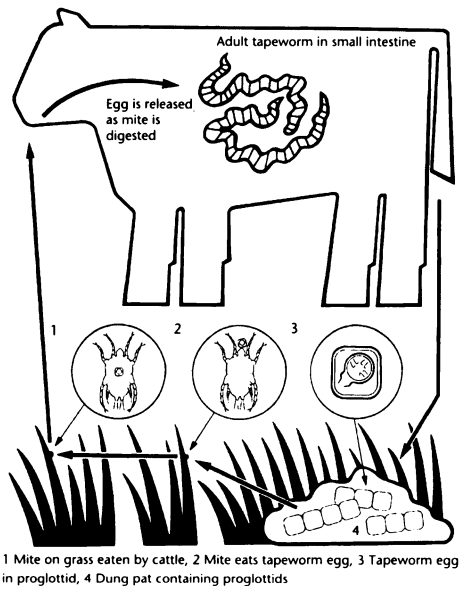


Fig. 38 *Moniezia* sp.



1 Mite on grass eaten by cattle, 2 Mite eats tapeworm egg, 3 Tapeworm egg in proglottid, 4 Dung pat containing proglottids

Fig. 39 Life cycle of *Moniezia* spp. [6]

Fig. 40 *Moniezia* sp.; segments with double genitalia [4]

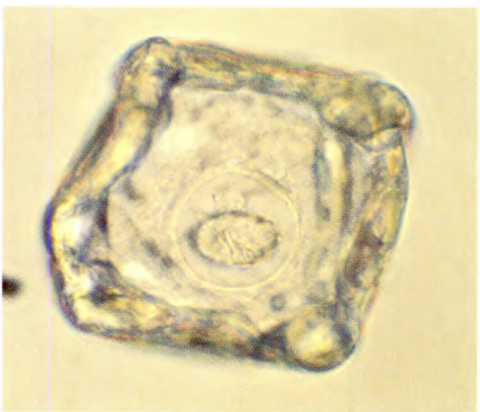


Fig. 41 Egg of *Moniezia* sp. (60–80 µm in diameter)

Fig. 42 *Moniezia* sp. in the small intestine of a calf

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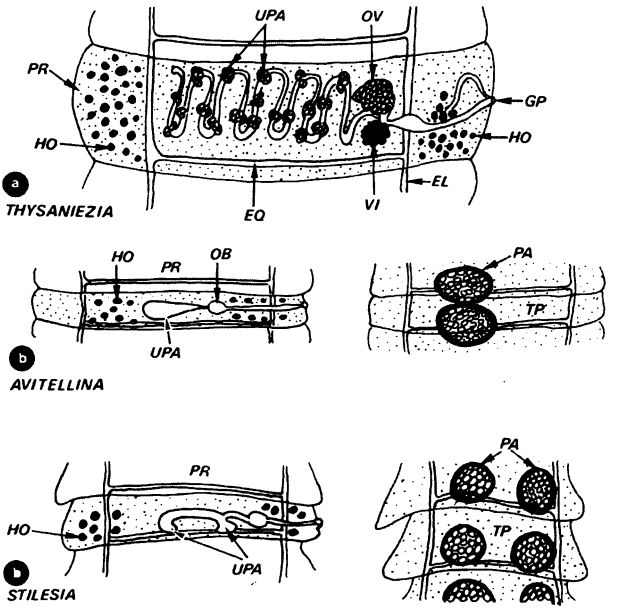

Fig. 43 Morphology (schematic) of proglottids of different tapeworms found in ruminants; EL, EQ = excretion pores, GP = genital pore, HO = testicular vesicles, OB, OV = ovary, PA = paruterine organ with eggs, PR = internal proglottis, TP = terminal proglottis, UPA = uterus with paruterine organ and VI = vitellarium [7]

Avitellina centripunctata

Location: Small intestine
Hosts: Sheep, goat, dromedary and rarely cattle
Species description: Adults reach 3 m in length and are about 3 mm wide. Proglottids appear not segmented. The uterus and the paruterine organs show an opaque line in the medial portion of the proglottids. The eggs measure 21–45 µm. This genus is non-pathogenic. Oribatid mites but also bark lice and dust lice are suspected to act as intermediate hosts.
Geographic distribution: Arid areas of Africa (Figure 37, 43)

Thysaniezia ovilla (syn. *T. giardi*)

Location: Small intestine
Hosts: Sheep, goat and cattle, dromedary and wild ruminants
Species description: Adults reach 2 m in length and are 12 mm wide. The side of the segment bulges out, giving the margin of the worm an irregular appearance. Eggs are found in groups of 10–15 in elongated paruterine organs (about 100 µm long), with a thick gray shell and a protuberance at one end. Oribatid mites and psocids (bark lice, dust lice) are the intermediate hosts. This species is not clinically important but frequently encountered, especially in southern parts of Africa.
Geographic distribution: Arid areas of Africa (Figures 37, 43, 44)

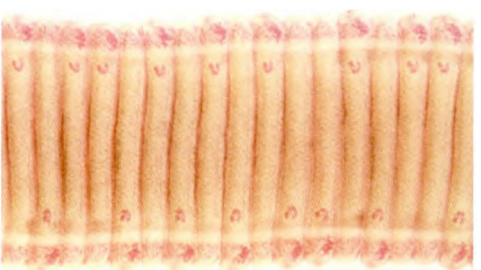

Fig. 44 *Thysaniezia ovilla*; segments with single genitalia which alternate irregularly [8]

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Stilesia globipunctata

Remarks: This tapeworm occurs in the small intestine mainly of sheep, goat, dromedary and rarely of cattle and other ruminants in Europe, Africa and Asia (☞ Sheep and Goats, 1).
(Figures 45, 46)

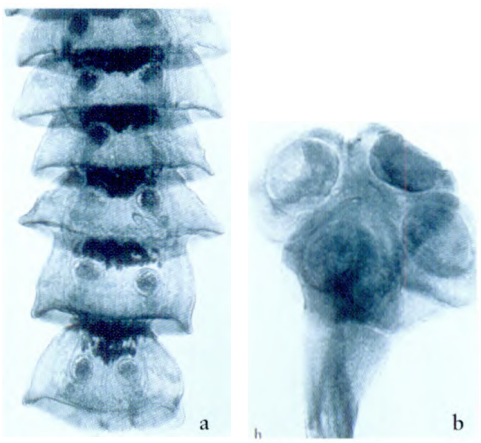

Fig. 45 *Stilesia* sp.; proglottids (a) and scolex (b); PA = paruterine organ [4]

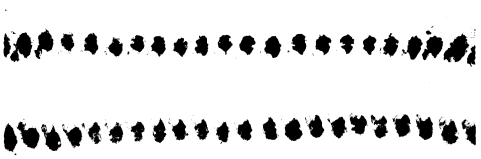

Fig. 46 *Stilesia globipunctata*; immature segments [8]

- **General features of intestinal tapeworms (*Moniezia* spp., *Thysaniezia* spp., *Avitellina* spp. and *Stilesia* spp.) of ruminants**

Symptoms: Generally inapparent infections; heavy infections are found only in young animals and can cause reduced weight gain. Masses of *Moniezia* can cause obstruction of the intestine.

Cave: Because of the large size of the tapeworms their presence is obvious and the underlying cause of parasitism, the small trichostrongylids, easily overlooked.

Significance: Tapeworms are widespread in ruminants, but their pathogenicity has not been proved conclusively and it seems that they are relatively low-pathogenic.

Diagnosis: Proglottids which look like cooked rice grains, containing typical thick-shelled or imperfectly rounded eggs, appear in the faeces. Eggs may also appear isolated in the faeces. The presence of tapeworms in the small intestine at slaughter is conclusive.

Therapy: Niclosamide (75–90 mg/kg) and praziquantel (5 mg/kg, sheep only) are specific cestodicidal drugs. In addition the following benzimidazoles are effective against tapeworms: albendazole (7.5 mg/kg), fenbendazole (5–10 mg/kg), oxfendazole (5 mg/kg), mebendazole (15–20 mg/kg, po.), netobimin (7.5–20 mg/kg, po.), febantel (5–7.5 mg/kg, po.). Special attention should be given to the control of tapeworms in lambs to avoid losses in heavily infected populations.

Prophylaxis: Reduction of the mite population is not feasible and emphasis should be given to anthelmintic treatments when losses due to tapeworms occur.

- **Nematoda eggs found in the faeces, adult nematodes living in the gastrointestinal tract and first-stage larvae of *Dictyocaulus viviparus***

Gongylonema pulchrum
Gullet worm, zigzag worm

Location: Mucosa of oesophagus and forestomachs

Hosts: Sheep, goat and less frequently cattle

Species description: Eggs laid by adult worms are passed in faeces and hatch into larvae when swallowed by manure-eating beetles or cockroaches. Larvae in cattle may be liberated in the stomach and migrate towards

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the oesophagus. Adult worms are 6 to 14 cm in length and a number of oval cuticular thickenings appear in longitudinal rows at the anterior end. The worms are embedded in the oesophageal lining in a zigzag pattern.

Geographic distribution: World-wide

Symptoms: Rarely observed

Significance: This is a harmless parasite and mainly found at autopsy. Irritation of the oesophageal and gastric mucosae may occur in infected animals.

Diagnosis: Mainly at necropsy; eggs occur in the faeces.

Therapy: Generally not indicated

Prophylaxis: Keeping the animals on dry, well-drained grounds or concrete floors has been described to be effective in controlling infection.

(Figures 47, 48, 49)

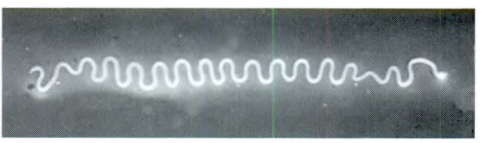

Fig. 47 *Gongylonema pulchrum*; typical zigzag pattern [4]

Fig. 48 *Gongylonema pulchrum*; anterior end with cuticular plaques [8]

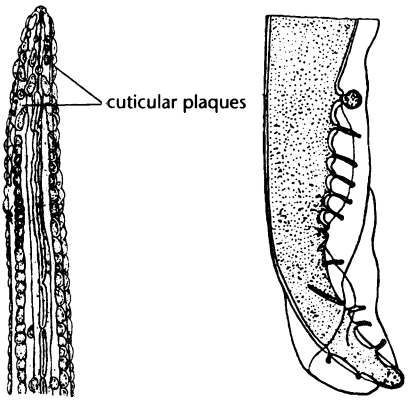

Fig. 49 *Gongylonema pulchrum*; anterior end with cuticular plaques [5]

Gongylonema verrucosum
Rumen gullet worm

Location: Mucosa of rumen, reticulum, omasum

Hosts: Cattle, sheep and goat

Species description: Not well known; dung beetles are intermediate hosts. The worms have a reddish colour when fresh. Males are 32–41 mm long and females 70–95 mm. A festooned cervical ala as well as cuticular bosses on one side only are typical.

Geographic distribution: South Africa, India, USA

Symptoms: Inapparent

Significance: Non-pathogenic

Diagnosis: Casually at necropsy

Therapy: Unknown

Prophylaxis: ☞ above *G. pulchrum*

Parabronema skrjabini

Location: Abomasum

Hosts: Cattle, sheep, goat and dromedary

Species description: This parasite belongs to the Spiruridae. *Stomoxys* spp. and *Lyperosia* spp. are intermediate hosts. Infective third-stage larvae develop in the flies and are deposited on the final host where the larvae are ingested. Males are 15–18 mm long, with one spicule measuring 600–710 µm and the other one 290–310 µm. Female

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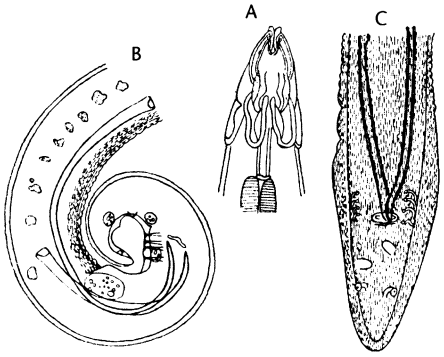

Fig. 50 *Parabronema rhodesiense*; anterior end (a), lateral view of the tail end of a male (b), ventral view of the tail of a male (c) [9]

worms are > 36 mm long and the vulva is situated at 5 mm distance to the hind end. The anterior end is characterised by pseudo-lips, which are wound around the buccal opening. Four papillae are found on these lips. The tail end of the male is reeled to a spiral and four pairs of preanal papillae are found. A group of terminal papillae is present as it is in all the spirurids.

Geographic distribution: Asia, Central and East Africa

Symptoms: Unspecific; abomasitis may be found.

Significance: Unknown; *P. skrjabini* may play a role together with other gastrointestinal nematodes

Diagnosis: At necropsy, adult worms may be found in abomasal scrapings.

Therapy and Prophylaxis: A specific therapy and prophylaxis is not described and probably also not necessary. For further information ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figure 50)

Haemonchus contortus Large stomach worm, twisted wire worm, barber's pole worm

Location: Abomasum

Hosts: Cattle, sheep, goat and other ruminants

Species description: *H. contortus* is known as the "red stomach-worm" or "wire worm" of ruminants and one of their most prevalent and most pathogenic parasites. Adults are 10 to 30 mm long. Males are shorter than females and have a reddish colour and a bursa with an asymmetrical dorsal lobe and barbed spicules. Females are identified as "barber's pole worms" because their white uteri are wound around their red blood-filled intestine. The vulva flap is prominent in female worms. The life cycle is direct and typical for the strongyle nematodes. The prepatent period is 19–21 days but it can be shortened by immunosuppressive pathogens such as concomitant trypanosome infections and stress factors. In arid areas, the parasite survives the dry season as inhibited fourth-stage larvae within the abomasal mucosa of the host. The inhibited larvae resume their development a few weeks before the onset of the new rainy season. This phenomenon is accompanied by a drastic increase of the egg output of infected animals before the wet season starts ("rains rise").

Geographic distribution: World-wide

Symptoms: Anaemia, oedema ("bottle jaw"), rough coat, weight loss or retarded growth

Significance: *H. contortus* is a very common parasite and one of the most pathogenic nematodes of ruminants. Heavy infections cause death in young animals, whereas chronic infections cause anaemia, hypoproteinaemia, progressive emaciation. These effects are generally compensated during the rainy season and only appear progressively during the following dry season when previously heavily infected animals may die.

Diagnosis: Eggs of strongyle-type appear in the faeces. In acute infections anaemia and death may occur before the worms reach maturity. No eggs are found in this case in the faeces and only the examination of the abomasum at necropsy allows an exact diagnosis.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 51, 52, 53, 54, 55, 56, 57, 58, 59, 60)

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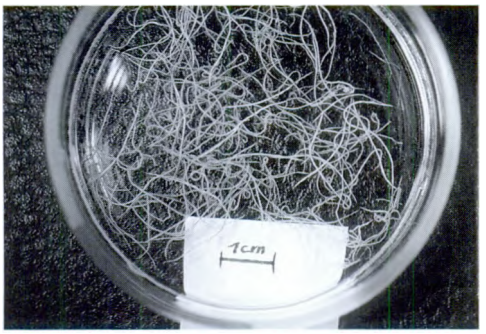

Fig. 51 *Haemonchus contortus*; adult parasites

Fig. 54 *Haemonchus contortus*; bursa copulatrix (schematic) with barbed spicules (490–540 µm long) and asymmetrical dorsal lobe [3]

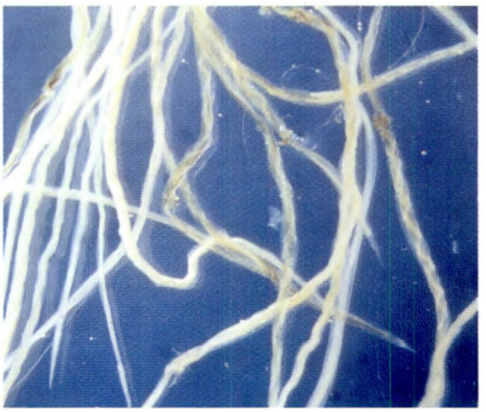

Fig. 52 *Haemonchus contortus*; twisted unteri and intesine [10]

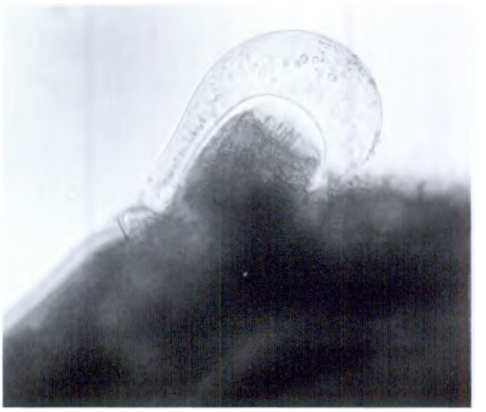

Fig. 55 *Haemonchus contortus*; vulva flap

Fig. 53 *Haemonchus contortus*; prominent cervical papillae (50×)

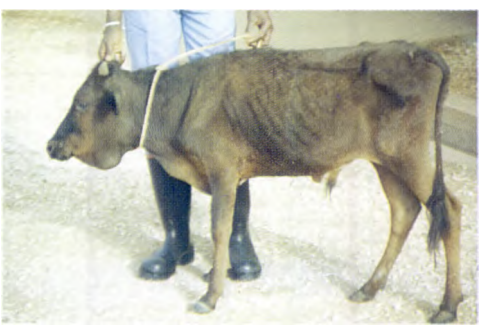

Fig. 56 N'Dama calf showing emaciation and "bottle jaw" due to a chronic haemonchosis

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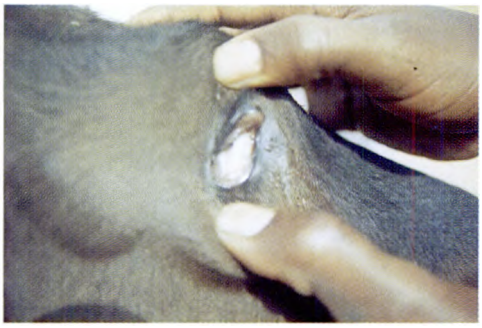

Fig. 57 Pale mucosa membranes following a heavy *Haemonchus contortus* infection

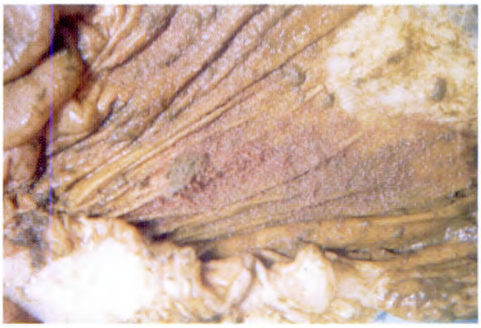

Fig. 59 *Haemonchus contort* on the abomasal mucosa of a calf

Fig. 58 Oedematous abomasal mucosa due to hypoproteinaemia [10]

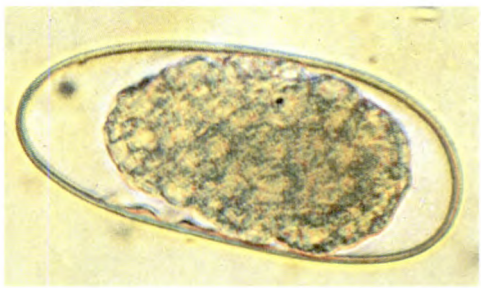

Fig. 60 Strongyle-type egg [11]

Mecistocirrus digitatus

Location: Abomasum of ruminants, stomach of pigs, rarely small intestine

Hosts: Cattle, buffalo, sheep, goat, pig and very rarely man

Species description: This nematode resembles *Haemonchus contortus*. The females also have the ovaries wound spirally around the intestine but they differ from the latter by the position of the vulva which is situated about 0.6–0.9 mm from the tip of the tail and the absence of a vulvar flap. The male bursa has a symmetrical dorsal lobe, which is very small. The spicules are long (3.8–7 mm) and united together for almost their whole length. The eggs measure 95–120 × 56–60 µm. This nematode is a blood sucker.

Geographic distribution: Widespread in Central and South America, Asia, Africa (including island of Mauritius)

Symptoms: Acute infections are associated with anaemia, weakness, poor growth and sometimes death. Chronic infections are associated with oedema, bottle jaw and progressive weight loss.

Significance: *M. digitatus* is an important parasite in endemic areas and young animals are particularly at risk. The effects are similar to those of *Haemonchus contortus*. This parasite generally occurs together with other gastrointestinal nematodes.

Diagnosis: Strongyle-type eggs appear in the faeces. Adult worms may be identified at necropsy.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 61, 62, 63)

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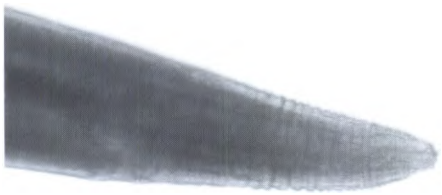

Fig. 61 *Mecistocirrus digitatus*; anterior end [8]

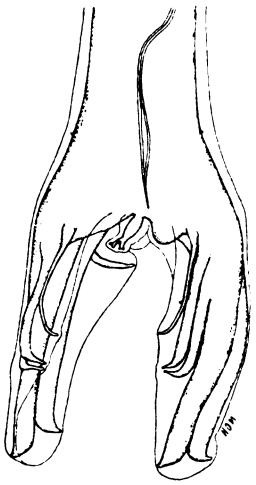

Fig. 62 *Mecistocirrus digitatus*; bursa copulatrix [3]

Trichostrongylus axei Stomach hair worm

Location: Abomasum and occasionally small intestine

Hosts: *T. axei* occurs in the abomasum of sheep, goat, cattle, wild ruminants and in the stomach of pig, horse donkey and man.

Species description: Direct nematode life cycle; males are 2.5–6 mm long and females are 3.5–8 mm long. The spicules are dissimilar: one measures 74–98 µm and the other 96–128 µm. The gubernaculum length is 50–61 µm. The eggs measure 79–92 × 31–41 µm.

Geographic distribution: World-wide

Symptoms: Weight loss and poor growth occur when animals are heavily infected especially in mixed infections with other trichostrongyles.

Significance: Serious weight loss and poor growth occur when animals are heavily infected especially in mixed infections with *Haemonchus*, *Ostertagia* and heavy *Cooperia* burdens.

Diagnosis: Detection of strongyle-type eggs in the sediment or flotation of faeces. An accurate diagnosis of the species can only be obtained by microscopic examimination of adult specimens at necropsy.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 64, 65, 66)

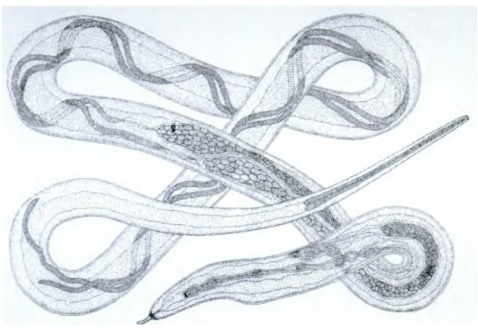

Fig. 63 *Mecistocirrus digitatus*; female worm (19–43 mm long; 470 µm in diameter) [9]

Fig. 64 *Trichostrongylus axei*; spicula (74–98µm and 96–128 µm) and gubernaculum (50–61 µm)

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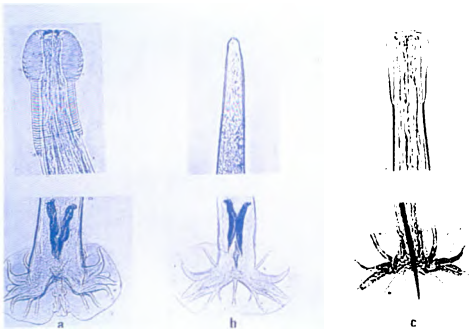

Fig. 65 (Berlin) *Cooperia* spp.; (a), *Trichostrongylus* spp. (b) and *Nematodirus* sp. (c); anterior ends (above) and bursa copulatrix (below) [4]

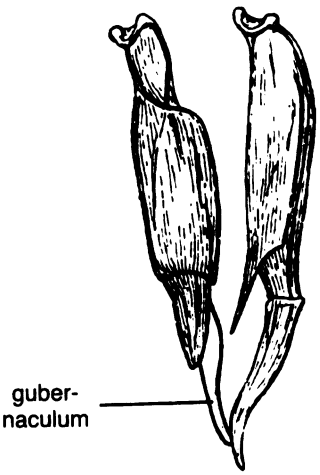

Fig. 66 *Trichostrongylus axei*; spicula (74–98 µm and 96–128 µm) and gubernaculum (50–61 µm) [5]

Trichostrongylus colubriformis and other *Trichostrongylus* spp. Hair worms, black scours worms

Location: Small intestine

Hosts: Cattle

Species description: ☞ above *T. axei;* the male worms are 4–4.5 mm and the females 5–7 mm long. The spicules of *T. colubriformis* are equal (135–156 µm long) and show a prominent distal end. The gubernaculum is present. Infection is acquired by ingestion of infective third stage larvae which burrow superficially into the crypts of the intestinal cells.

Geographic distribution: World-wide

Symptoms: *Trichostrongylus* spp. infections may be difficult to diagnose. The symptoms they produce are similar to those of other trichostrongylid infections. Weakness, diarrhoea or constipation and in chronic infections anaemia due to emaciation and malnutrition may be seen.

Significance: *T. colubriformis* and other small intestinal *Trichostrongylus* spp. are commonly present in mixed-species infections, so their effect is additive.

Diagnosis: Strongyle-type eggs appear in the faeces. Adult worms can be identified at necropsy.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 67, 68)

Fig. 67 *Trichostrongylus colubriformis*; bursa copulatrix with spicula (135–156 µm)

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Fig. 68 *Trichostrongylus colubriformis*; spicula (135–156 µm) and gubernaculum (65–75 µm) [5]

Ostertagia spp. (*O. ostertagi, O.* [syn. *Skrjabinagia*] *lyrata* and other species)

Brown stomach worms

Location: Abomasum, upper small intestine

Hosts: Cattle, sheep and goat

Species description: *O. ostertagi* is the most important trichostrongylid of cattle world-wide. In Africa it plays a role in imported cattle and autochthonous infections are reported from East Africa. *O. circumcincta* was reported from sheep and goats in East Africa (Kenya, Ethiopia, Uganda, Zambia) and South Africa, and *O. pinnata* was only reported from sheep in Kenya. *O. lyrata* (syn. *Skrjabinagia lyrata* or *Grosspiculagia lyrata*) occurs in cattle in Africa. It is very similar to *O. ostertagi* which accounts for enormous losses world-wide in cattle rasing areas. Adult worms of the genus *Ostertagia* are brownish, thread-like, and grow to 9 mm in length. They all have a restricted and small cephalic vesicle, small cervical papillae projected from the body surface and pronounced longitudinal, cuticular ridges. The life cycle is direct and typical for roundworms. Environmental conditions of cold or excessive dryness may trigger a condition known as *hypobiosis*, in which larval development in the abomasal mucosa is arrested and maturation is resumed several months later. Ingested larvae enter the glands of the abomasum, causing erosion of the cells, maldigestion, protein losses with the consequence of weight loss, diarrhoea and hypoproteinaemia.

Geographic distribution: World-wide

Symptoms: Severe diarrhoea, oedema (bottle jaw or ascites), weight loss leading to emaciation.

Significance: *Ostertagia* spp. are widespread parasites of cattle. Affected cattle not only lose weight but often die of clinical ostertagiosis. *Ostertagia* spp. infections are generally accompanied by other gastrointestinal nematodes.

Diagnosis: Strongyle-type eggs appear in the faeces. Eggs per gramm > 1000 in calves indicate a harmful condition. It should be controlled with an anthelmintic treament.

Therapy and Prophylaxis: Avoid overstocking, use pasture management to avoid accumulation of infective larvae on herbage and soil; regular or strategic use of anthelmintics ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 69, 70, 71, 72)

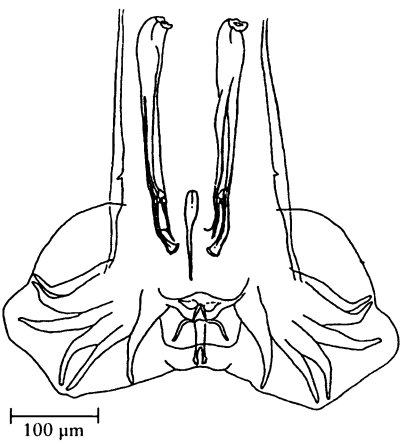

Fig. 69 *Ostertagia ostertagi*; bursa copulatrix with spiculum (200 – 280 µm) [12]

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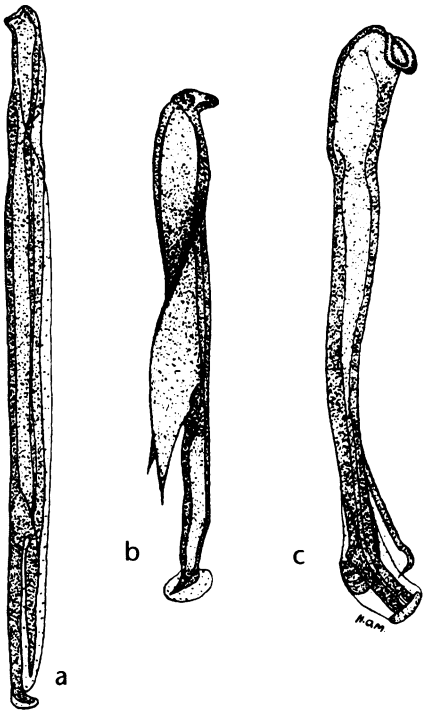

Fig. 70 Spicules of: (a) *Ostertagia circumcincta* (up to 450 µm long), (b) *Ostertagia trifurcata* (150–180 µm long) and (c) *Ostertagia ostertagi* (200–280 µm long) [3]

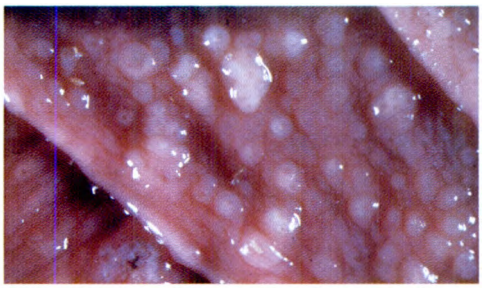

Fig. 71 *Ostertagia ostertagi*; nodules in abomasal mucosa (2–4 mm in diameter)

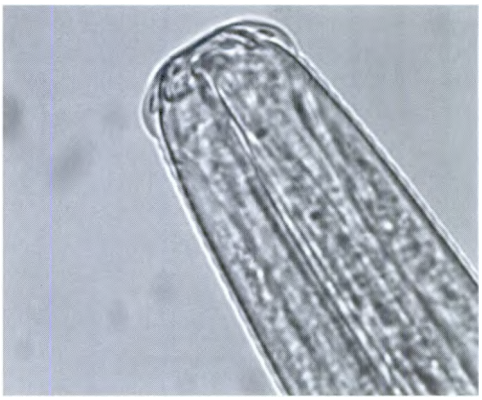

Fig. 72 *Ostertagia* sp.; anterior end with cephalic vesicle

Cooperia spp. (*C. pectinata, C. punctata, C. spatulata* and other species)

Cattle bankrupt worms

Location: Small intestine

Hosts: Cattle

Species description: Direct life cycle; infective larvae develop within 4 days after faecal deposition. Typical for all *Cooperia* spp. the "swollen" head because of a pronounced cephalic vesicle. The body cuticle bears 14–16 longitudinal ridges which appear punctuated. There are no cervical papillae. The spicules are stout, pigmented brown and usually have a ridged, wing-like expansion in the middle. When present in large numbers calves may show markedly reduced weight gains. *Cooperia* spp. are generally accompanied by other trichostrongylids.

Geographic distribution: World-wide; widespread in Africa

Symptoms: Reduced weight gain, especially in calves; diarrhoea can occur, causing dehydration which can severely affect young stock.

Significance: Heavy burdens with *Cooperia* spp. result in poor utilization of feed, weight loss and transient diarrhoea. Especially in calves they can significantly contribute to the overall damage of gastrointestinal nematode infections.

Diagnosis: Strongyle-type eggs occur in the faeces. Species diagnosis only with adult specimens at necropsy.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 73, 74, 75, 76, 77, 78)

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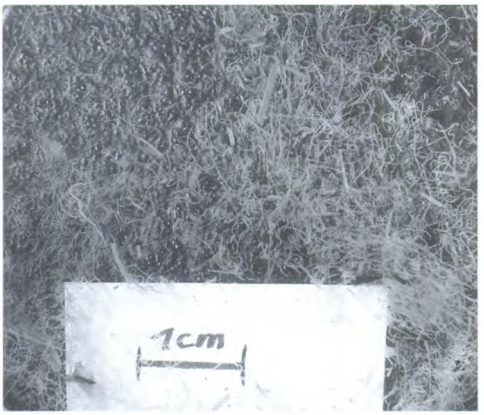

Fig. 73 *Cooperia* spp.; adult parasites [10]

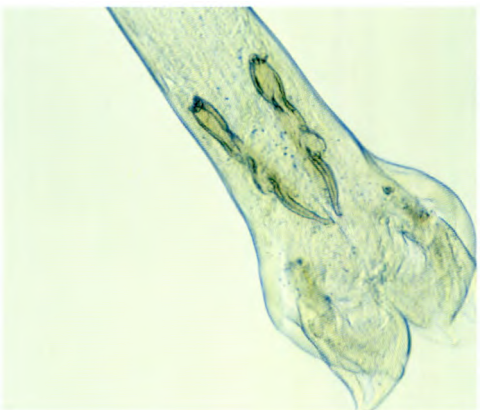

Fig. 74 *Cooperia punctata*; bursa copulatrix with spicules (123–145 µm)

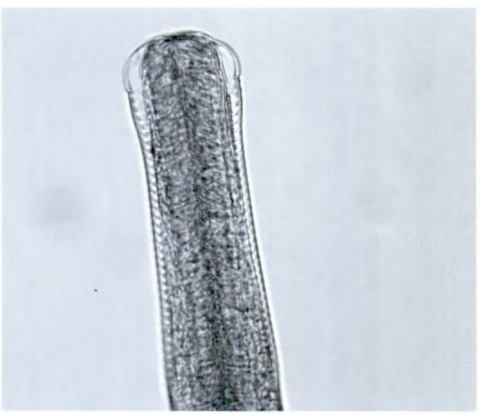

Fig. 75 *Cooperia pectinata*; anterior end with enlarged cephalic vesicle

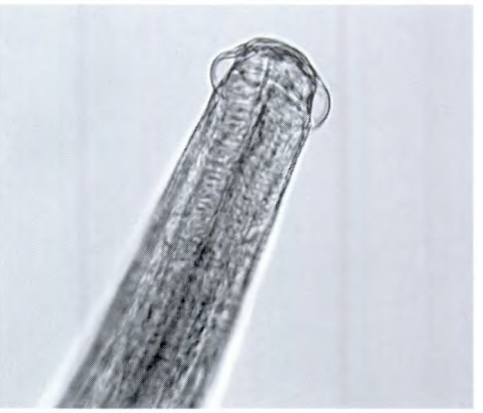

Fig. 76 *Cooperia punctata*; anterior end with enlarged cephalic vesicle

Fig. 77 *Cooperia pectinata*; spicules (240–390 µm)

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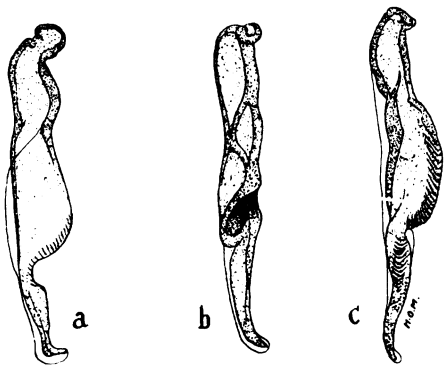

Fig. 78 Spicules of (a) *Cooperia curticei* (135–145 µm long), (b) *Cooperia punctata* (123–145 µm long) and (c) *Cooperia pectinata* (240–390 µm) [3]

***Nematodirus* spp. (*N. filicollis, N. spathiger, N. helvetianus, N. battus*) Thin-necked intestinal worms, thread-necked strongyles**

Location: Small intestine

Hosts: Cattle (*N. helvetianus, N. filicollis*, seldom *N. spathiger*); sheep and goat (*N. spathiger, N. filicollis, N. battus*); and dromedary (*N. spathiger*)

Species description: *Nematodirus* species have an inflated cuticle (bell-glass) around their anterior end. The anterior end is clearly thinner than the posterior part. The bursa of the males has elongate lateral lobes and the spicules are long and slender and their tips are fused together. The tail of the females is short with a slender terminal appendage. The eggs are large (150–260 × 65–110 µm) and can be distinguished from those of other trichostrongylids by their large size. The life cycle is direct. Infective third-stage larvae develop within the eggs which are resistant to adverse environmental conditions. Infection occurs when infective larvae are ingested. The prepatent period is 15–28 days.

Geographic distribution: World-wide; 5–6 *Nematodirus* species are known to occur in North Africa (Maghreb) and certain regions of South Africa. Little is known about the tropical regions of Africa but it seems that *N. spathiger, N. helvetianus* and *N. filicollis* occur in tropical areas.

Symptoms: Intestinal symptoms, diarrhoea, inappetence may be associated with heavy *Nematodirus* infections.

Significance: Heavy infections together with other gastrointestinal nematodes may cause poor growth, emaciation and death. The host is harmed mainly by the larvae which may cause significant destruction of the intestinal epithelial cells.

Diagnosis: Typical eggs which are larger than most strongyle eggs appear in the faeces.

Cave: The eggs of the small ruminant hookworm *Gaigeria pachyscelis* are also large and should be differentiated from those of *Nematodirus* spp. During the prepatent period damage and symptoms of infection may be seen without the typical eggs being excreted in the faeces. Mucosal scrapings may demonstrate the immature worms in the mucosa.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 79, 80, 81, 82)

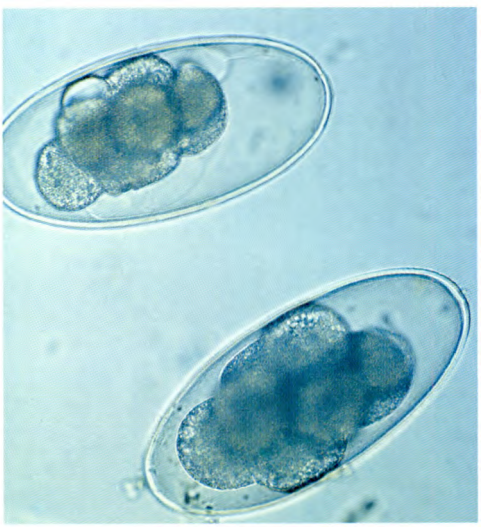

Fig. 79 Eggs of *Nematodirus* spp. (150–260 × 65–110 µm)

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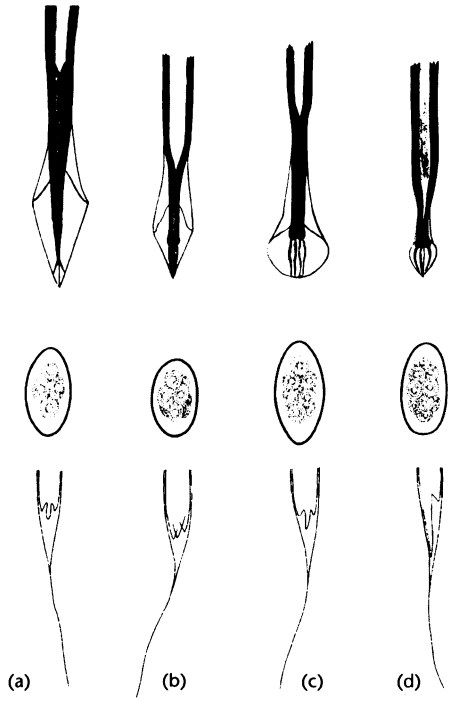

Fig. 80 Posterior end of spicules of: (a) *Nematodirus helvetianus*, (b) *N. filicollis*, (c) *N. spathiger* and (d) *N. battus* [5]

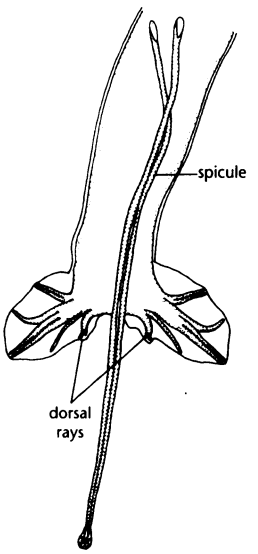

Fig. 81 *Nematodirus spathiger*; bursa copulatrix with spicules (950 µm long; tips fused together) [5]

Fig. 82 *Nematodirus helvetianus*; anterior end with enlarged cephalic vesicle (schematic)

Impalaia tuberculata

Remarks: This is mainly a parasite of the dromedary, rarely of zebu, cattle and sheep in many parts of Africa. It occurs in the abomasum and upper small intestine (☞ DROMEDARIES, ■ 1).

Dictyocaulus viviparus Cattle lungworm

Remarks: The first-stage larvae of *Dictyocaulus viviparus* (☞ Cattle, ■ 4.3) is found in the faeces of infected animals. The larvae are recovered from the faeces by means of the Baermann technique (☞ METHODS, 1.7). (Figure 83)

Fig. 83 First-stage larvae of *Dictyocaulus viviparus* (390–490 × 25 µm) [4]

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Oesophagostomum radiatum Nodular worm

Location: Adult worms are found in the large intestine (caecum and colon ascendens). Larvae occur in nodules between small intestine and rectum.

Hosts: Cattle

Species description: Direct life cycle; ingested larvae penetrate the intestinal wall, forming nodules anywhere between the small intestine and the rectum. The head end of the adult worms is characterised by a large cephalic vesicle which is constricted behind its middle. Eggs appear in the faeces about 40 days after ingestion of the third-stage larvae.

Geographic distribution: World-wide

Significance: One of the most damaging worms to cattle when present in high numbers (> 200 adults in calves; > 1000 adults in adult cattle). Young stock may die from nodular worm infections.

Symptoms: Heavy infections are accompanied by anaemia, oedema (hypoalbuminaemia) and diarrhoea (reduced fluid absorption). *Oesophagostomum* infections occur generally with other gastrointestinal nematodes.

Diagnosis: Thin-shelled strongyle-type eggs appear in the faeces and pea-shaped nodules in the intestinal wall at necropsy indicate infection with the nodular worm. The diagnosis can be confirmed by demonstrating the typical large worms at necropsy.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 84, 85, 86, 87)

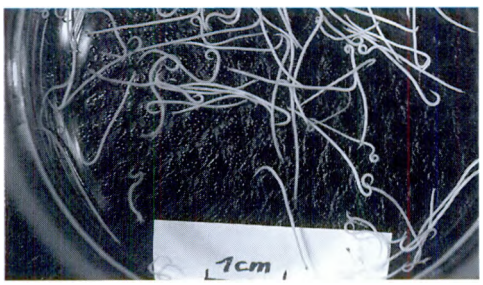

Fig. 84 *Oesophagostomum* spp.; adult parasites

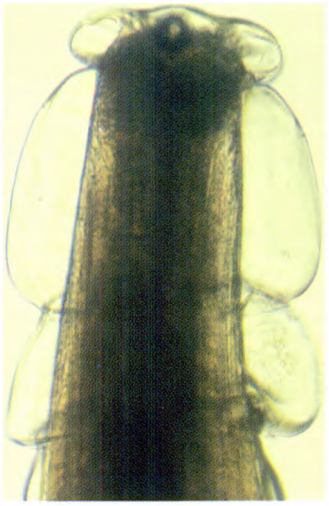

Fig. 85 *Oesophagostomum radiatum*; anterior end with large, constricted cephalic vesicle

Fig. 86 Multiple nodules caused by *Oesophagostomum radiatum* in the caecal mucosa of a calf

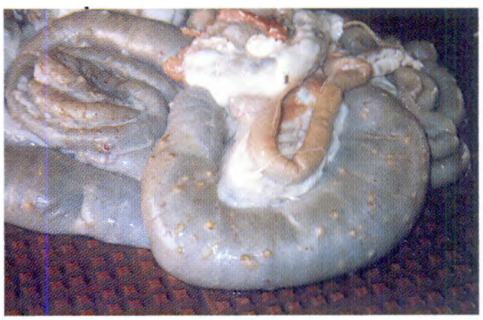

Fig. 87 Multiple nodules in the wall of the large intestine caused by *Oesophagostomum radiatum*; view from the serosa side

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Oesophagostomum multifoliatum

Remarks: This is mainly a large intestinal parasite of sheep, goats but also zebu. Its occurrence has been reported from Nigeria, Kenya, Tanzania and Zimbabwe (☞ SHEEP AND GOATS, 1).

Oesophagostomum columbianum

Remarks: This parasite occurs in the colon of sheep, goats, camel, a number of antelopes and occasionally of zebu. It occurs worldwide and is particularly reported from tropical Africa (☞ SHEEP AND GOATS, 1).

Bunostomum phlebotomum

Cattle hookworm

Location: Small intestine

Hosts: Cattle

Species description: Adults are 10–28 mm long. There is a prominent buccal capsule with two pairs of subventral lancets. The spicules of the male measure 3.5–4 mm. The life cycle is direct. Infective larvae usually enter cattle by ingestion or by skin penetration.

Significance: *B. phlebotomum* is one of the most pathogenic helminths of cattle in warm and humid areas. Especially for suckling and freshly weaned calves, during the rainy season it can be a major pathogen causing severe anaemia. Only 50 adult specimens in the small intestine can cause severe anaemia in calves. This parasite is generally accompanied by other gastrointestinal nematodes. *Bunostomum* was found to occur focally only in certain herds of a region.

Geographic distribution: World-wide, especially in warm, humid zones

Symptoms: Penetration of the skin by larvae may cause irritation of the host: itching of the legs and feet results in cattle stamping their feet. Iron-deficiency anaemia and hypoproteinaemia, accompanied by oedma ("bottle jaw") are the predominant symptoms of cattle hookworm infections.

Significance: *B. phlebotomum* may be a serious threat to cattle, especially to calves. Because of its skin-penetrating infection mode, it can also affect suckling calves, killing them within a few days. Heavy infections cause acute anaemia and death. Chronic infections result in poor growth.

Diagnosis: Strongyle-type eggs in the faeces and severe anaemia especially in young calves during the rainy season may suggest hookworm infection.

Therapy and Prophylaxis: ☞ below THERAPY OF NEMATODE INFECTIONS, p. 53

(Figures 88, 89)

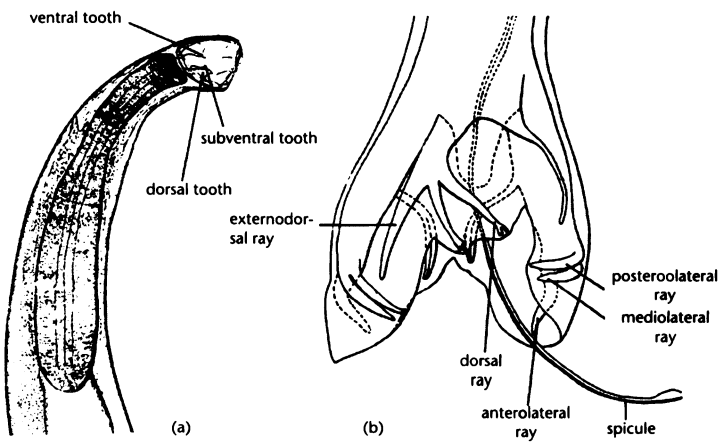

Fig. 88 *Bunostomum phlebotomum*; anterior end (a) and bursa copulatrix (b) [5]

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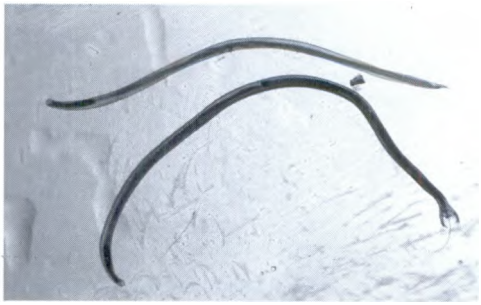

Fig. 89 *Bunostomum phlebotomum*; adult male (10–18 mm) and female (25–28 mm)

Toxocara (syn. *Neoascaris*) *vitulorum*
Large cattle roundworm

Location: Small intestine
Hosts: Cattle
Species description: Large white worms with blunt ends, up to 30 cm long and 5 mm in diameter. The head end is characterised by three lips without papillae. The cuticle is thin and these worms therefore have a soft, translucent appearance. Adults are almost exclusively found in young calves. Adult females produce enormous numbers of eggs (8×10^6 eggs per day).

The patency is low and 4–6 months after birth no adults remain in calves. Eggs become infective within 15 days and may survive for extended periods. Infection is acquired by ingestion of embryonated eggs (containing second-stage larvae). The larvae hatch in the intestine of the host and penetrate the intestinal wall and either migrate via liver, lungs, trachea, oesophagus to the intestines where they develop to adults or they migrate to several tissues, including mammary glands. These larvae may remain dormant until they are mobilized during the late pregnancy to pass via the milk to the calves. These larvae may reach maturity about three weeks after the calf is born. *Toxocara* larvae can also cross the placenta. *T. vitulorum* is found almost exclusively in calves and prenatal and trans-mammary infections constitute the major sources of infection of young calves.

Geographic distribution: World-wide
Symptoms: Unspecific; intermittent diarrhoea, steatorrhoea, colic due to intestinal obstruction, weight loss and death.
Significance: In tropical and subtropical regions of Africa *T. vitulorum* is considered as a serious parasite with high mortality rates among neonatal calves. In temperate areas this parasite occurs without producing great losses.
Diagnosis: Eggs with thick, pitted shells (75–95 × 60–75 µm) appear in the faeces of calves which are around 4–6 weeks old. Sometimes adult, almost transparent worms appear on the faeces. An increased antibody titer of the pregnant cow close to birth may indicate a risk of prenatal infection of the foetus.
Therapy: Several of the anthelmintics commonly used for trichostrongyle infections in cattle are also effective against *T. vitulorum* in calves. These include albendazole, fenbendazole, oxfendazole, febantel, mebendazole and levamisole. Piperazine (70–150 mg, po.) also eliminates *T. vitulorum* (☞ below THERAPY OF NEMATODE INFECTIONS).
Prophylaxis: In endemic areas pregnant cows and neonatal calves should be treated with compounds which are effective especially against immature stages such as levamisole (7.5 mg/kg po. or pour on), pyrantel (10–20 mg/kg po.) or fenbendazole (7.5 mg/kg po.).
(Figures 90, 91)

Fig. 90 *Toxocara vitulorum*; adult worms (up to 30 cm long) found in the small intestine at slaughter [8]

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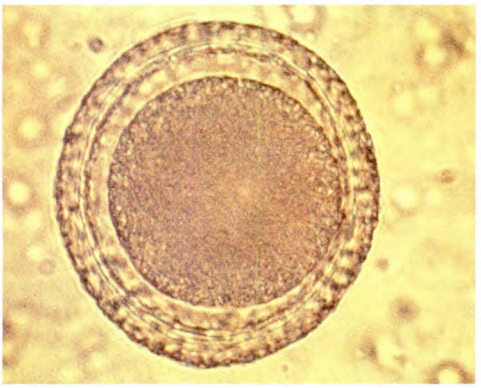

Fig. 91 Egg of *Toxocara vitulorum* (69–95 × 60–77 µm) [11]

Trichuris ovis, Trichuris globulosa and *Trichuris discolor* Whipworms

Location: Caecum and colon

Hosts: Cattle, sheep, goat and many other ruminant species

Species description: Whip worms are 3–8 cm long and easily identified by their long filamentous anterior portion and a thick shorter posterior portion. The male posterior end is usually tightly coiled and there is a single spicule. Direct roundworm life cycle. Infective larvae (second-stage larvae) develop within the eggs after at least 3 weeks on pasture. Eggs may remain infective for several years. Animals become infected by ingesting embryonated eggs, and the larvae penetrate the anterior small intestine for 2–10 days before they move to the caecum where they develop to adults. Prepatent period is 50–84 days and varies markedly among the species. High numbers of preadult and adult worms cause irritation and inflammation of the caecum and colon.

Geographic distribution: World-wide

Symptoms: Mild infections (up to 50 adult *Trichuris* spp. in cattle or small ruminants) do not cause symptoms. Heavy infections (> 500 adult *Trichuris* spp.) may cause colitis, diarrhoea, progressive weight loss. Oedema may occur in the neck and thoracic region.

Significance: Whipworms are widespread but the naturally acquired infections in cattle, sheep and goats usually do not cause clinical disease. Sheep older than 8 months show an age resistance, resulting in a resistance to reinfection 2–3 weeks after a primary infection. However, animals kept in poor condition and carrying multiple infections (e.g. trypanosomes and trichostrongylids) are often heavily parasitised by *Trichuris* spp.

Diagnosis: Demonstration of the characteristic, brown, barrel-shaped eggs with a transparent plug at either pole.

Therapy: Some of the modern benzimidazoles (at increased dose rates) may be used to treat *Trichuris* spp. and *Capillaria* spp. infections in ruminants. These include albendazole, fenbendazole, netobimin and oxfendazole. Ivermectin (200 µg/kg, sc.) is effective.

Prophylaxis: Difficult because of the tenacity of the infective eggs. Regular removal of faeces in the surroundings of the animals may drastically reduce the infection risk.

(Figures 92, 93, 94)

Fig. 92 *Trichuris* spp.; adult parasites

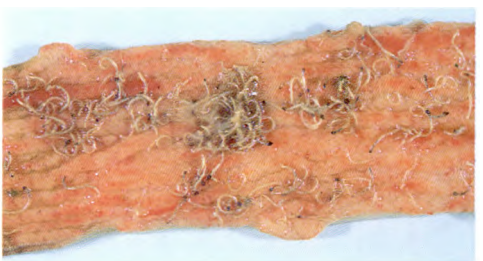

Fig. 93 *Trichuris* spp.; masses of worms in the large intestine of a calf

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Fig. 94 Egg of *Trichuris* spp. (70–80 × 25–40 µm) [4]

Fig. 95 *Capillaria bovis*; adult parasite containing eggs

Trichuris skrjabini

Remarks: This parasite occurs in dromedaries and may occasionally be found in cattle (☞ DROMEDARIES, 1).

Capillaria bovis

Location: Small intestine
Hosts: Cattle, sheep, goat and other ruminants
Species description: *C. bovis* occurs in the gastrointestinal tract of cattle, sheep and goats. The males are 8–13 mm and the females are 12–20 mm in length. The eggs measure 45–50 µm × 22–25 µm. The life cycle is direct.
Geographic distribution: Temperate climates but via imports also in Africa
Symptoms: Low or no pathogenic effect in ruminants
Significance: Little is known about *Capillaria* spp. except that they occur frequently.
Diagnosis: Detection of *Capillaria* eggs by the flotation or sedimentation method.
Therapy and Prophylaxis: ☞ *Trichuris* spp.
(Figures 95, 96)

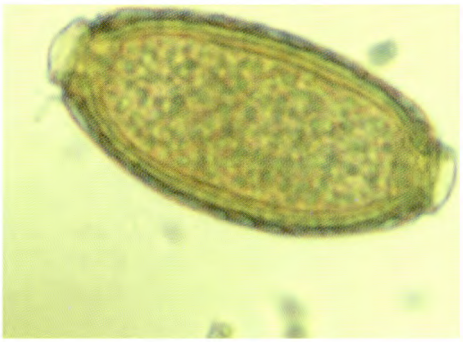

Fig. 96 Egg of *Capillaria bovis* (40–50 × 22–25 µm)

Strongyloides papillosus
Intestinal threadworm

Location: Small intestine
Hosts: Cattle, sheep, goat, dromedary and other ruminants
Species description: *S. papillosus* is a hair-like nematode which is 3.5–6 mm long and only 0.05–0.06 mm wide. Only the parthenogenetic females are parasitic. They are embedded in the mucosa of the upper small intestine. Free-living males and females reproduce sexually outside the host. Infection is acquired by ingestion of infective third-stage larvae and skin penetration.

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S. papillosus can cross the placenta and infect calves before birth. This parasite can also pass via the colostrum to newborn calves.

After ingestion or skin penetration of infective larvae they migrate via blood to the lungs. After penetration of the alveoli they are coughed up and swallowed, and develop within 9 days into adults. There are two possible ways of development; one is a homogonic cycle involving adult females in the host producing eggs that do not require fertilisation to develop. These eggs are passed in the faeces and then develop to infective third-stage larvae.

In the heterogonic cycle adult threadworms in the intestine lay eggs which develop into a different type of larvae. These larvae can develop to adult males and females which can live outside the host. The fertilised eggs of this population produce infective larvae that are ingested by the host. The prepatent period is about 10 days!

Geographic distribution: World-wide

Symptoms: The migratory phase is associated with coughing, fever and pneumonia which may be followed by secondary infections, especially in calves kept under poor conditions. Adult parasites cause irritation and severe inflammation of the small intestine. Catarrhal enteritis, digestive disturbances and intermittent diarrhoea may be seen. Death may occur in suckling calves.

Significance: Intestinal threadworms are widespread in warm and humid areas. They may cause great losses, particularly in young calves.

Diagnosis: Small, embryonated, thin-shelled eggs appear in the faeces. The adult, parasitic females are difficult to recognise at necropsy. Microscopical examination of mucosa scrapings is necessary to discover them.

Therapy: Not all the anthelmintics are effective against the intestinal threadworms. Albendazole, febantel and fenbendazole, oxfendazole are effective and proven against *S. papillosus*. Most of the modern benzimidazoles are effective against *S. papillosus* (Table 5). Thiabendazole (75 mg/kg, po.) is very effective. Ivermectin (200 µg/kg, sc.) is effective against the adult worms. Levamisole and mebendazole are insufficiently effective against intestinal threadworm infections.

Prophylaxis: Special attention must be paid to pasture management. The night holding areas should be rotated regularly to reduce the infection risk in the surroundings of the herds. Suckling calves should be kept on clean, dry areas to avoid infection by skin penetration. In heavily infected herds, the pregnant cows should be treated with an anthelmintic to avoid transplacental infection of the unborn calf. Treatments must be repeated more frequently than with other nematodes because of the extremely short prepatent period.

(Figures 97, 98)

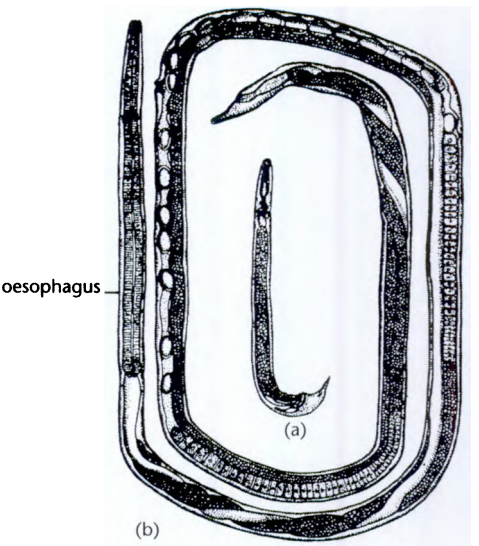

Fig. 97 *Strongyloides papillosus*; free living male (a) and parthenogenetic parasitic female (3.5–6 mm long) with elongated oesophagus (b) [5]

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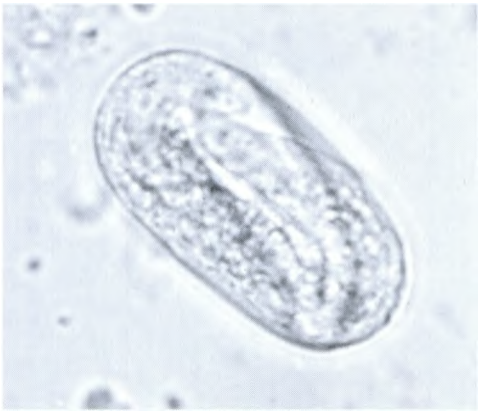


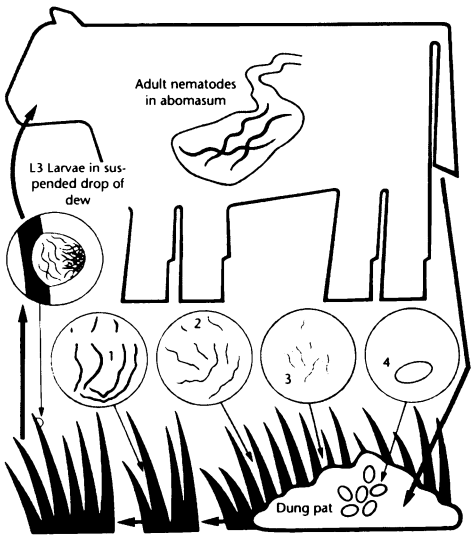
Fig. 98 Embryonated egg of *Strongyloides papillosus* (45–65 × 25 µm)

- **Therapy of nematode infections**

Species of *Haemonchus*, *Mecistocirrus*, *Parabronema*, *Trichostrongylus*, *Ostertagia*, *Cooperia*, *Impalaia*, *Nematodirus*, *Bunostomum*, and *Oesophagostomum*

Gastrointestinal nematodes occur wherever ruminants are raised. Attempts to control nematode infections should concentrate on a reduction of the worm load rather than on an eradication of these parasites. The risk to acquire a harmful infection is high where the stocking rate is high (fenced paddocks, correos, night holding places, etc.) or when the daily grazing time is short at the time when animals should put on body weight (e.g. rainy season in mixed farming-systems of sub-Saharan regions). Priority should be given to strategic control rather than to a regular dosing of anthelmintics. Animals at risk (weaned calves) should be treated repeatedly during the first grazing season. Strategic control programs of first season grazing animals in temperate areas are described extensively (☞ FURTHER READING). In semiarid areas first season calves should be treated at least twice during the rainy season, 4 weeks after the onset of the rains and at the end of the rains with an anthelmintic listed below, in order to make animals use the great food resources of the wet season. An additional treatment at the culmination of the wet season may markedly increase the growth rate. Regular rotation of the heavily contaminated pastures or night holding places and long daily grazing periods during the rainy season are effectively reducing the risk of nematode infections. Rainy season treatment is more effective than dry season treatment.
(Figures 99, 100, 101, Table 5)

Fig. 99 N'Dama cattle tethered on a "correo". The night holding areas may represent an important source of infections with gastrointestinal nematodes



1 L3 Larvae in grass, 2 L2 Larvae in dung, 3 L1 Larvae in dung pat, 4 Nematode egg in dung pat

Fig. 100 Direct life cycle of a typical round worm

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Table 5 Anthelmintics recommended for cattle and sheep

Anthelmintics	Spectrum	Dosage (mg/kg)		
		Cattle	Sheep	AM
Albendazole	Nematodes, cestodes, trematodes	7.5	5.0	po.
Coumaphos	*Trichostrongylid* spp.	2 (6x)		po.
Febantel	Nematodes	7.5	5	po.
Fenbendazole	Nematodes, cestodes	7.5	5.0	po.
Ivermectin	Nematodes, arthropods	0.2	0.2	po. or sc.
Ivermectin and Clorsulon	Nematodes, trematodes, arthropods	0.2/7		po.
Ivermectin	Nematodes, trematodes, arthropods	0.5		pour on
Levamisole	Nematodes	5 0	5.0	sc. or im.
Levamisole	Nematodes	7.5	7.5	po.
Levamisole	Nematodes	10	10	pour on
Mebendazole	Nematodes	20		po.
Netobimin	Nematodes, cestodes	7.5	7.5	po.
Morantel tartrate	Nematodes	10	10	po.
Morantel tartrate	Nematodes	SRB*		ir.
Oxfendazole	Nematodes, cestodes	4.5	5	po.
Phenothiazine	Nematodes	220[1]		po.
Pyrantel tartrate	Nematodes	12.5	25	po.
Thiabendazole	Nematodes	44–75		po.
Moxidectin	Nematodes, arthropods	0.2	0.2	po.

*Slow Release Bolus (rumen retention device releases about 200 mg/day for 60 days)

[1] safety index very low (= 1); AM = application method, po. = orally, sc. = subcutaneously, im. = intramuscularly and ir. = intraruminal

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2 Stages in the blood and circulatory system

PROTOZOA

TRYPANOSOMATIDAE

Trypanosoma congolense, Trypanosoma vivax, Trypanosoma brucei Tsetse-transmitted trypanosomosis, Nagana, sleeping sickness

Hosts: Cattle, sheep, goat and many other domestic and wild animal species

Vector: Several species of tsetse flies: *Glossina morsitans* (savanna areas), *Glossina palpalis* (areas around rivers and lakes) and *Glossina fusca* (high forest areas). All three species transmit trypanosomosis and feed on a wide spectrum of mammals.

Species description: The most important trypanosomes affecting cattle, sheep and goats are as follows (in order of importance): *T. congolense*, *T. vivax* and *T. brucei*. These three species belong to the Salivaria (Table 6). The differentiation and the morphological characteristics of these pathogenic trypanosomes are listed in Table 7. All three species are transmitted by tsetse flies. Most tsetse-transmission is cyclic and begins when blood from a trypanosome infected animal is ingested by the fly. The trypanosomes lose their surface coat, multiply in the fly, re-acquire a new surface coat and become infective. The life cycle within the tsetse fly varies among the different trypanosome species. The infective form for animals in the tsetse fly is referred to as the metacyclic form. The development in the tsetse flies may be as short as 1 week with *T. vivax* or extend to a few weeks for *T. brucei*. Mechanical transmission requires blood containing trypanosomes being transferred from one animal to another. Tsetse flies inoculate metacyclic trypanosomes into the skin of animals, where the trypanosomes multiply and cause swellings (chancres). They enter the blood stream either directly or through the lymph nodes, then the bloodstream where they divide rapidly by binary fission. *T. congolense* attach to endothelial cells and localize in capillaries. *T. brucei* and *T. vivax* invade tissues and cause tissue damage in several organs. The necropsy findings vary and are not specific. Extensive petechiae of the serosal membranes, especially in the peritoneal cavity may occur in acute, fatal cases. The lymph nodes and the spleen are usually swollen. Chronic cases are associated with atrophy of body and organ fat, severe anaemia and swollen lymph nodes.

The immune response of infected animals is vigorous, and immune complexes cause inflammation, which contributes to the clinical signs and lesions of trypanosomosis. Trypanosomes have multiple genes that code for different surface-coat glycoproteins. The number of different antigenic types of glycoprotein that can be made is unknown but exceeds several hundred. The antigenic variation results in persistence of the organisms in the host and is a way of the parasite to evade the host's immune system. Antigenic variation has prevented the development of a vaccine and permits reinfections when animals are exposed to a new antigenic type of the same trypanosome species. Animals infected with trypanosomes show some degree of immunodepression and are more susceptible to a.o. gastrointestinal helminth infections. *Haemonchus contortus* shows a reduced prepatent period and causes a markedly increased mortality in animals that are chronically infected with trypanosomes.

Some *bos indicus* (Zebu) are very sensitive to trypanosomosis and they are generally

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Table 6 Pathogenicity[1] of salivarian trypanosomes in livestock

Trypanosome subgenus	Trypanosome species	Cattle	Sheep and Goats	Pigs	Camels	Equines
Trypanozoon	*T. brucei*[2]	+	+ +	+	+ + +	+ + +
	T. evansi[3]	+ +	+	+ +	+ + +	+ + +
	T. equiperdum[4]	-	-	-	-	+ + +
Nannomonas	*T. congolense*	+ + +	+ +	+	+ +	+ +
	T. simiae	-	+	+++	+++	-
Duttonella	*T. vivax*	+ + +	+ +	-	+ +	+ +
Pycnomonas	*T. suis*[5]	-	-	+ +	-	-

- = not pathogenic; + = mildly pathogenic; + + = moderately pathogenic; + + + = severely pathogenic;
[1]under usual field conditions, but which is modified by many factors; [2]*T. brucei gambiense* and *T. brucei rhodesiense* cause human sleeping sickness in West and East Africa, respectively, and have animal reservoirs, in which pathogenicity is low; *T. brucei brucei* is not infective to humans; [3]mechanical transmission by biting flies other than tsetse; [4]venereal transmission; [5]rarely encountered

Table 7 Morphological characteristics of trypanosomes

Species	Free flagellum	Kinetoplast	Undulating membrane	size in µm	Size and motility in dark ground
T. vivax	present	large, terminal	not prominent	20-26	large,extremely active, transverses the whole field quickly
T. brucei	present in all but stumpy forms central	small, subterminal	prominent	12-35*	large, rapid movements in confined areas
T. congolense	absent	medium, subterminal, marginal	not prominent	9-18	small, adheres to red blood cells by anterior end

* polymorphic: slender, intermediate and stumpy forms; *T. theileri* is frequently found in cattle and may be distinguished by its large size; *T. theileri* is more than twice the size of the pathogenic African trypanosomes

not raised within the tsetse-belt. The most pathogenic species for cattle is *T. congolense*, which causes an acute, lethal disease in zebu cattle. Some pure taurine cattle breeds are less susceptible and succumb less to the disease. *T. vivax* causes a less acute disease than *T. congolense*. In East Africa, however, *T. vivax* can cause an acute hemorrhagic syndrome. Development of clinical symptoms is slower but remissions are frequent. *T. brucei* causes a chronic infection without distinct symptoms.

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A number of indigenous African taurine cattle breeds (e.g. N'Dama, Baoulé, West African Shorthorn and many others) are less susceptible to trypanosomosis (often referred to as "trypanotolerance"). These breeds are adapted to a certain environment and tsetse-challenge and may be an alternative way to control losses due to trypanosomosis. However, "trypanotolerance" is not an absolute trait and refers to a lower susceptibility of an indigenous breed to its innate environment. If such animals are kept under heavy trypanosomosis challenge they may equally succumb like other non-tolerant breeds. Many indigenous breeds of sheep and goats from western and Central Africa are also resistant to trypanosome infections.

Geographic distribution: *T. congolense* and *T. brucei* occur mainly within the tsetse-belt (area from latitude 15°N to 29°S) of Africa. *T. vivax* occurs in Africa within the tsetse-belt where it is transmitted by tsetse flies but also in non-tsetse areas (Central and South America, West India, Mauritius) where it may be transmitted by Tabanids and biting flies.

Symptoms: The primary clinical signs are intermittent fever, anaemia and weight loss. Cattle usually have a chronic course with a high mortality, especially if there are other stress factors such as poor nutrition or other diseases. Mortality rates are increased if concomitant infections with gastrointestinal helminths are present. Blood sucking nematodes (e.g. *Haemonchus contortus, Bunostomum* spp. and *Gaigeria* spp.) may severely aggravate the anaemia. Ruminants generally recover gradually if the number of infected tsetse flies is low. However, stress results in relapse.

Significance: Tsetse-transmitted trypanosomosis is one of the major constraints for cattle rearing within the tsetse-belt of Africa. Trypanosomes render their host more susceptible to other diseases (e.g. worm infections) which then may contribute to the high mortality.

Diagnosis: A presumptive diagnosis is based on finding an animal in poor condition in an endemic area. Confirmation depends on demonstrating trypanosomes in a wet mount of the buffy coat area of a PCV tube after centrifugation (☞ METHODS, 3.4). Other infections that cause anaemia and weight loss, such as gastrointestinal nematodes, babesiosis, anaplasmosis and theileriosis should be eliminated by further examinations. Various serological tests are suitable for herd and area screening rather than for individual diagnosis. Tests for the detection of circulating trypanosome species-specific antigens in peripheral blood may be useful for both individual and herd diagnosis.

Therapy: Most of the drugs commonly used to treat trypanosomosis (Table 8) have a narrow therapeutic index. Therefore the administration of the correct dosing is essential. Resistance to trypanocidal drugs is widespread and should be considered in refractory cases. The following compounds may be used in ruminants: Diminazene aceturate (3.5–7 mg/kg, im.), homidium bromide (1 mg/kg, im.), isometamidium (0.5–1 mg/kg, im.) and quinapyramine sulfate (5 mg/kg, sc.) (☞ Table 8). Concomitant gastrointestinal nematode infections should be treated at the same time and supportive treatment (e.g. iron dextrane) may help recovery (☞ CATTLE, 1).

Prophylaxis: Control can be carried out at several levels. Attempts to eradicate tsetse flies have failed and are no longer justifiable. Tsetses can be partially controlled in the surrounding of a herd by spraying or dipping of animals. The strategic use of insecticide-impregnated screens may drastically reduce the tsetse population in the surrounding of herds and thus the infection risk. Pour-on application of insecticides on a herd level is an effective way of controlling both tsetse flies and fly nuisance (☞ CATTLE, ■ 5). Losses due to trypanosomosis are often drastically increased by other opportunistic infections (mainly gastrointestinal nematodes) and other stress fac-

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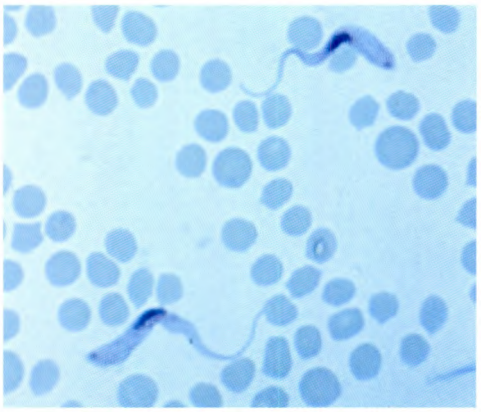

Fig. 101 *Trypanosoma vivax* (20–26 µm); Giemsa-stained bloodsmear [4]

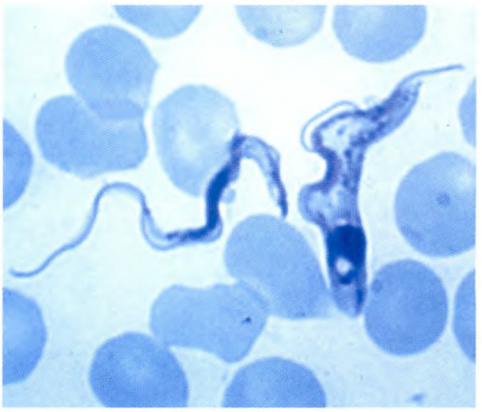

Fig. 102 *Trypanosoma brucei* (12–35 µm); Giemsa-stained bloodsmear; this species is polymorphic: slender form (left), stumpy form (right) [13]

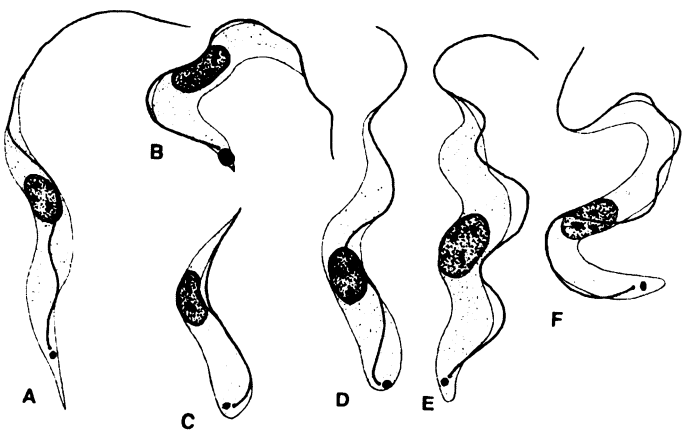

Fig. 103 Important *Trypanosoma* species of livestock:
(a) *Trypanosoma theileri* (60–70 µm),
(b) *T. cruzi*,
(c) *T. congolense* (9–18 µm),
(d) *T. vivax* (20–26 µm),
(e) *T. equiperdum* (25 µm) and
(f) *T. brucei* (12–35 µm, polymorphic) [14]

tors. Control measures should also include these factors. The prophylactic use of trypanocidal drugs requires a regular application of the drug and includes the risk of creating resistance. It should therefore be limited to emergencies, such as cattle trekking through infested areas and herds seasonally exposed to high trypanosomosis risk. Isometamidium (1–2 mg/kg, im.) protects cattle for 2 to 6 months. Prophylactic use of trypanocidal drugs should be avoided in cattle prior to slaughtering, since drug residues may be detrimental to human health. Proper nutrition is essential in all cases and the strategic application of crop residues at the late dry season may significantly improve the nutritional status and thus the resistance of animals in endemic areas. The exploitation and propagation of indigenous breeds can be a promising way of animal production in endemic areas.

(Figures 101, 102, 103, 104, 105, Table 6, Table 7, Table 8)

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Table 8 Generic and trade names of trypanocides for the the treatment and prevention of animal trypanosomosis

Compound generic name	Trade name	Manufacturer	Action	Range of dosage Rates (mg/kg)	Route of administration	Remarks
Diminazene aceturate	Berenil	Hoechst AG, Germany	T	3.5-7.0	IM SC	Also babesiacidal; toxic to horses, donkeys, dogs, and camels
	Ganasag	Squibb, USA	T	3.5-7.0	IM or SC	
Homidium bromide	Ethidium	CAMCO Animal Health, UK	T(P)	1.0	IM	
Homidium chloride	Novidium	Rhône-Merieux, France	T(P)	1.0	IM	
Isometamidium	Samorin	Rhône-Merieux, France	P/T	0.25-1.0	IM, IV[3]	Toxic above 2 mg/kg; highly irritant; avoid SC administration
	Trypamidium	Rhône-Poulenc Sante France	P/T	0.25-1.0	M, IV[3]	
Quinapyramine sulfate[1]*	Antrycide	Coopers Animal Health Ltd, UK	T	3.0-5.0	SC	Rest animals before and after treatment
Quinapyramine prosalt[1]	Antrycide[2] R.F.	Coopers Animal Health Ltd, UK	PT	3.0-5.0	SC	Dosage calculated as sulfate
Suramin*	Naganol Antrypol	Bayer AG, Germany	T	10.0	IV	Severe local reactions by other routes
Melaminophenylarsine dihydrochloride	Cymelarsan	Rhône-Merieux, France	T	0.25-0.5	IM, SC	The IM route is preferred in equines. *T. evansi* and *T. brucei*

Many of these preparations are sold under a variety of trade names. Consult publications such as *Veterinary Pharmaceuticals and Biologicals*, 6th ed. Lenexa, KS, Veterinary Medicine Publishing Co, 1989/1990;

[1]Reintroduced in 1985 to treat mainly *T. evansi* infections;

[2]Prosalt. This is a mixture of sulfate and chloride salts of quinapyramine;

[3]Given by very slow injection of 1% W/V solution at 0.5 mg/kg;

T = therapeutic action; P = prophylactic action; (P) = short prophylactic activity; IM = intramuscular; IV = intravenous; SC = subcutaneous;

* no longer commercially available

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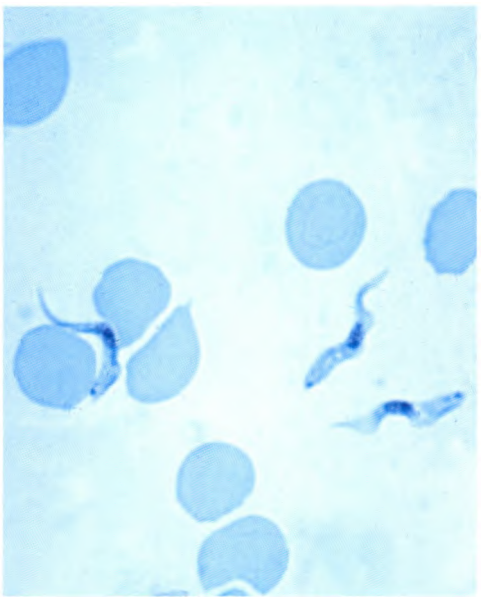

Fig. 104 *Trypanosoma congolense* (9–18 µm); Giemsa-stained bloodsmear [15]

Fig. 105 Comparison of a trypano-tolerant N'Dama cattle (left) and susceptible zebu (right) after 15 weeks' exposure to a middle-grade tsetse challenge. Both animals were chronically infected with *Trypanosoma congolense* [36]

Trypanosoma evansi (syn. *T. brucei evansi*) Surra

Remarks: *T. evansi* occurs in dromedaries, equines, buffaloes, carnivores and many other mammalian species. It occurs in North Africa, Asia, Middle and South America. The pathogenicity of *T. evansi* strains varies considerably but the highest pathogenicity is commonly observed in camels and equines (☞ DROMEDARIES, ■ 2). Several species of blood-sucking flies act as vectors. *T. evansi* is not transmitted by tsetse flies. No developmental stages have been demonstrated in the vectors which differentiate the parasite from *T. brucei*. *T. evansi* is 15–35 µm long and 1.5–2.5 µm wide. *T. evansi* is morphologically identical with *T. brucei* and other members of the subgenus Trypanozoon (☞ DROMEDARIES, ■ 2).

Trypanosoma theileri

Hosts: Cattle

Vector: Tabanid flies (*Tabanus* spp. and *Haematopota* spp.)

Species description: This is a large species (60–70 µm, sometimes up to 120 µm in length=twice the size of the pathogenic trypanosomes) and belongs to the Stercoraria. The undulating membrane is well developed and the free flagellum is well defined. Although the parasite is considered as non-pathogenic it may assume increased significance when stress conditions arise or when concurrent infections with other pathogens are present.

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Geographic distribution: World-wide
Symptoms: Infections are often asymptomatic.
Significance: This species may be very prevalent but it seems to be nonpathogenic.
Diagnosis: ☞ above *T. congolense*, *T. vivax*, *T. brucei*
Therapy and Prophylaxis: Generally not indicated
(Figure 106)

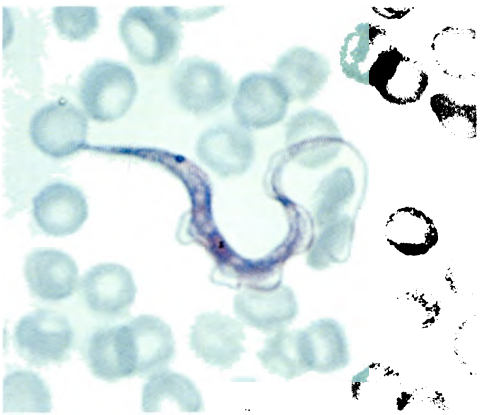

Fig. 106 *T. theileri* (60–70 µm, sometimes up to 120 µm); Giemsa-stained bloodsmear [4]

BABESIIDAE

Babesia bovis

Redwater, tropical bovine babesiosis

Hosts: Cattle
Vector: *Boophilus* spp. (*B. microplus*, *B. annulatus*) and possibly *Rhipicephalus bursa*.
Species description: This is a small form, 2.4 × 1.5 µm and slightly larger than *B. divergens*. The organisms (merozoites) are usually found in the centre of the erythrocytes. Vacuolated "signet-ring" forms are especially common. Transovarian transmission occurs in the ticks.
Geographic distribution: Tropical Africa, Madagascar, Mediterranean basin and southern Europe
Significance: The disease caused by *B. bovis* is similar to that caused by *B. bigemina*. Affected animals may die. The strains vary in virulence. In some regions *B. bovis* is more virulent than *B. bigemina*.
(Figures 107, 108)

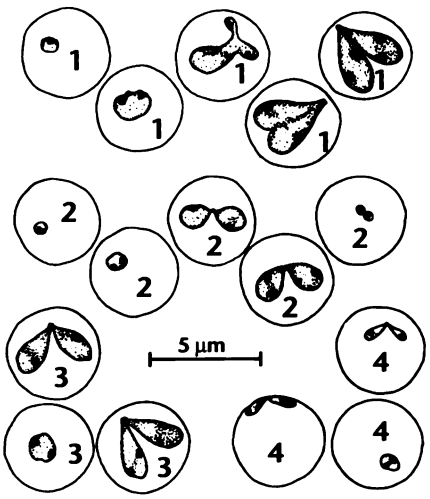

Fig. 107 Morphology of bovine *Babesia* spp. 1 *Babesia bigemina*, 2 *Babesia bovis*, 3 *Babesia major* and 4 *Babesia divergens* [16]

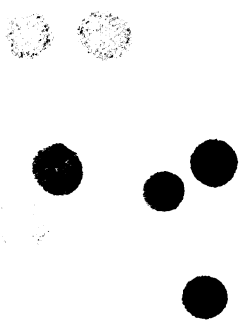

Fig. 108 *Babesia bovis* (2.4 × 1.5 µm, "small form"); Giemsa-stained bloodsmear [4]

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Babesia divergens
European bovine babesiosis

Hosts: Cattle
Vector: *Ixodes ricinus* and possibly other ticks
Species description: This species is smaller than *B. bovis*. The merozoites usually occur as paired, club-shaped organisms about 1.5 × 0.4 µm. The angle between the pair is relatively large, so that they diverge more from each other than those of *B. bovis*. The organisms tend to lie along the circumference of the host erythrocyte. Other forms may be stout and pyriform, measuring 2 × 1 µm; some may be circular and others may be vacuolated and up to 2 µm in diameter. Transovarian transmission occurs in the vector. Highest parasitaemia levels are found during the period of high fever.
Geographic distribution: Northwestern Africa in woodlands at altitudes above 1000 m. It also occurs in northern and central Europe.
Significance: The disease entity produced by *B. divergens* is less severe than that caused by *B. bigemina*. In severe infections death may occur.
(Figure 109)

Babesia bigemina (syn. *Piroplasma bigeminum*), Redwater, tropical bovine piroplasmosis, tick fever, "Texas fever"

Hosts: Cattle
Vector: *Boophilus* spp. (*B. microplus, B. decoloratus, B. calcaratus*), *Rhipicephalus* spp. (*R. evertsi, R. bursa* and *R. appendiculatus*), and *Haemaphysalis* spp. (*H. punctata*)
Species description: This is a large form, 4–5 µm by 2 µm wide. Round forms are 2–3 µm in diameter. The organisms are characteristically pear-shaped and lie in pairs forming an acute angle in the erythrocyte. Round, oval or irregularly shaped forms may occur, depending on the stage of development. Transovarian transmission occurs in the ticks. *B. bigemina* is one of the most pathogenic *Babesia* species known.
Geographic distribution: Tropical Africa, Madagascar, Mediterranean basin and many other parts of the world
Significance: *B. bigemina* is one of the most important diseases of cattle in some tropical and subtropical areas.
(Figure 110)

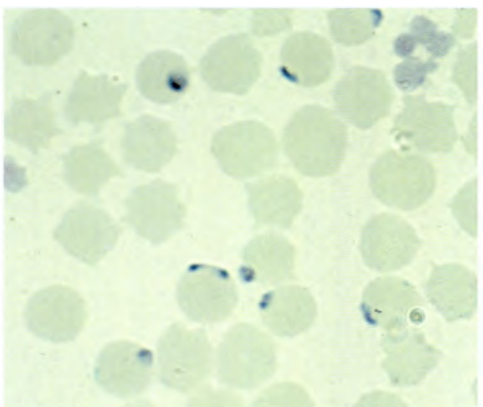

Fig. 109 *Babesia divergens* (1.5 × 0.4 µm); Giemsa-stained bloodsmear [4]

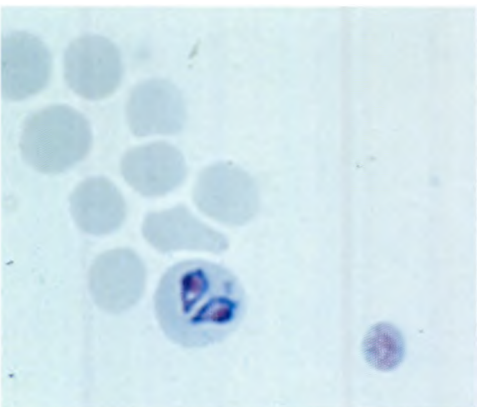

Fig. 110 *Babesia bigemina* (4–5 × 2 µm, "large form"); Giemsa-stained bloodsmear [4]

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Babesia major (syn. *Piroplasma major*)
European bovine piroplasmosis

Hosts: Cattle

Vector: *Haemaphysalis punctata*

Species description: This species resembles *B. bigemina* except that it lies in the centre of the erythrocyte. The pyriform bodies are 2.6 × 1.5 µm and the angle formed by the organisms is less than 90°. Round forms are about 1.8 µm in diameter. Transovarian transmission may occur in the tick vector.

Geographic distribution: North Africa, Europe, UIS, South America

Significance: *B. major* is less pathogenic than *B. bovis*. Clinical signs are moderate and sometimes infections are inapparent.

(Figure 111)

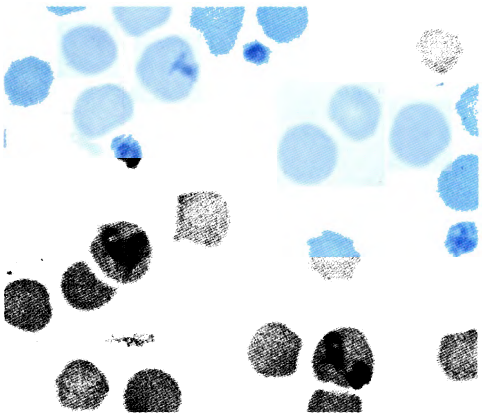

Fig. 111 *Babesia major* (2.6 × 1.5 µm); Giemsa-stained bloodsmear [15]

- **General features of bovine babesiosis**

Symptoms: Infections with *B. bigemina*, *B. major* and *B. divergens* are characterised by a haemolytic syndrome (haemolytic babesiosis), including continuous fever (40–41°C), high parasitaemia (2.5–10%), anaemia, icterus (intense yellow-brownish ocular, gingival and vaginal mucosae) and haemoglobinuria (dark brown, frothy urine) and general depression. Abortion and agalactia in dairy cows are early signs. The symptoms are less pronounced in the subacute form. *B. bovis* infections are dominated by a shock syndrome, leading to death following a steep rise in temperature in the peracute form. Acute disease is accompanied by fever, ataxia, pedalling movements and overt agressivity (attacks). Haemolysis, icterus and haemoglobinuria are less apparent than in other babesioses.

Significance: Widespread throughout the tropics, causing heavy losses in non-resistant (often imported) livestock. Without treatment mortality rates are very high (30% for *B. bigemina*, 70–80% for *B. bovis*). *B. bigemina* is one of the most important diseases of cattle in some tropical and subtropical areas.

Diagnosis: Clinical diagnosis is difficult and can be confused with leptospirosis (peracute disease), anaplasmosis (chronic in nature) or cowdriosis (no movements). Splenomegaly, icterus and haemoglobinuria and thickened bile in the gall bladder are characteristics of haemolytic babesiosis. Petechiae and microinfarcts suggest infections with *B. bovis*. The presence of Babesia parasites in the blood smears of animals without clinical symptoms does not necessarily support the diagnosis of babesiosis. It can be a resurgence of a chronic infection following an immunosuppression due to another disease. In *B. bigemina* infections the severity of the disease can be deducted from the percentage of parasitized red blood cells (< 0.1% mild, 0.5–1% subacute, 5–10% severe infection), whereas in *B. bovis* infections the presence of parasites indicates babesiosis, because parasite concentration in the peripheral blood is considerably lower than in the organs. In acute *B. bovis* infections impression smears of congested organs show punctiform babesial bodies in agglutinated erythrocytes. Serological techniques (IFAT, ☞ METHODS, 5.1) are available and should be used especially in epidemiological studies. For the individual

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Table 9 Drugs used in the treatment of babesiosis in cattle

Drug	Dosage	AM	Parasite	Remark
Trypan blue*	2-3 mg/kg	IV	*B. bigemina*	Not effective against small babesias; discouloration of animal's flesh; can cause severe tissue sloughing if not given IV
Acridine derivatives				
Euflavine[1]*	4-8 ml/100 kg as 5% solution	IV	*B. bigemina* *B. bovis* *B. divergens*	Highly irritant if not given IV
Diamidine derivatives				
Amicarbalide Diampron[1]	5-10 mg/kg	IM	*B. bigemina* *B. divergens* *B. bovis*	
Diminazene Berenil[2]	3-5 mg/kg	IM	*B. bigemina* *B. bovis* *B. divergens*	
Imidocarb	1-3 mg/kg Imizol[3]	SC or IM	*B. bigemina* *B. bovis* *B. divergens*	Prophylactic activity up to 8 weeks depending on dose and *Babesia spp.* involved; nephrotoxic; cholinesterase inhibitor; slowly metabolized and eliminated–tissue
Phenamidine Lomadine[1]	8-13.5 mg/kg	SC or IM	*B. bigemina* *B. bovis*	Cholinesterase inhibition
Quinoline derivatives				
Quinuronium Babesan[4]	1 mg/kg	SC	*B. bigemina* *B. bovis* *B. divergens*	Low therapeutic index Slow effect against *B. bovis* Side effects associated with the stimulation of the parasympathetic nervous system

Many of these preparations are sold under a variety of trade names; * no longer commercially available;
IM = intramuscular; IV = intravenous; SC = subcutaneous; AM = application method;
[1] May & Baker Ltd., Dagenham, England. [2] Hoechst AG, Frankfurt, Germany. [3] Burroughs Wellcome & Co., London, England.
[4] ICI-, Macclesfield, Cheshire, England

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diagnosis the demonstration of the parasite in the bloodsmear or with the QBC method (☞ METHODS, 3.1 and 3.3) is more adequate.

Therapy: Diminazene aceturate (3.5 mg/kg, im.), Imidocarb dipropionate (1.2–2.4 mg/kg, sc.), quinuronium sulfate (1–2 mg/kg, sc. or im.) and amicarbalide diisethionate (5–10 mg/kg, im.) can be used against the pathogenic *Babesia* species of cattle (☞ Table 9).

Two points should be considered: 1) For serious clinical attacks the dose should be split to avoid shock due to massive destruction of *Babesia*. 2) Most babesicidal drugs are toxic to the host, so caution is required in their use. The commonly used babesicidal compounds are listed in Table 9. In endemic areas it is preferable to support immunity within the population, as such animals can withstand repeated infections. The aim of therapy is therefore to reduce but not to eradicate the parasite concentration within the host. In severely affected animals emphasis should also be given to supportive treatment (electrolytes, rehydration, iron-dextran, etc.) and rich nutrition during the convalescence.

Prophylaxis: Several methods are used in endemic areas to control or prevent babesiosis.

- Controlled immunisation, using babe sicidal-drugs:
  Application of a babesicidal drug (e.g. diminazene aceturate, 3.5 mg/kg, im.) following inoculation of a non-attenuated *Babesia* strain.
- Imidocarb (2 mg/kg) is administered to an animal prior to the introduction onto an infected pasture. The delayed action of imidocarb controls clinical infection (up to 60 days for *B. bigemina* and up to 21 days for *B. divergens* and *B. bovis*) without blocking the immune response.
- Immunization with attenuated *B. bovis* and *B. bigemina* strains:
  Inoculation of a attenuated *Babesia* strains. Several research teams are presently working on the development of *Babesia* vaccines in cattle. In some countries vaccines are available.
- Prophylaxis through tick control:
  In tropical Africa and the Mediterranean region tick control is essential (☞ CATTLE, ■ 5) and should be made weekly during periods of adult tick activity.

(Figure 112, Table 9)

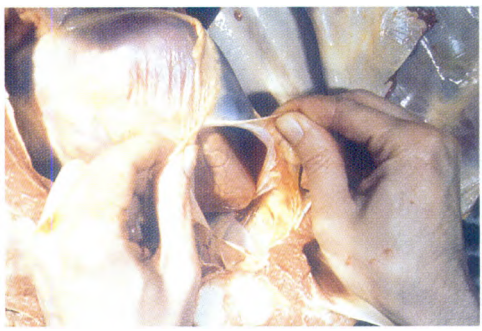

Fig. 112 Icterus and hydropericardium caused by *Babesia bigemina* infection [8]

THEILERIIDAE

Theileriosis is caused by protozoan parasites of the genus *Theileria*. In cattle, there are two highly pathogenic species. *Theileria parva* is the cause of East Coast Fever in East and Central Africa. A variant regarded as either a subspecies, *T. parva lawrencei*, or a type, *T. parva* (*lawrencei* type), is a common benign parasite of the African buffalo (*Syncerus caffer*), which, when transmitted to cattle, causes a condition called corridor disease. *T. parva* (*bovis* type) causes the "January disease" in Zimbabwe. *Theileria annulata* causes tropical theileriosis, which is widespread through the Mediterranean basin, the Middle East and Asia. Other species that infect cattle in Africa are *T. mutans*, *T. taurotragi* and *T. velifera*. They are usually benign, although *T. mutans* may be pathogenic under some circumstances. *T. orientalis* is a generally benign species found in the Mediterranean basin, the Middle

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East, Asia and Australia which may cause disease in imported cattle.
(Figures 113, 114, 115)

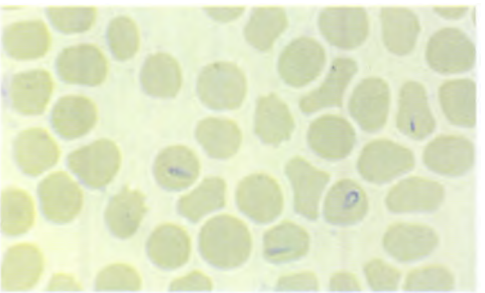

Fig. 113 *Theileria orientalis*; Giemsa-stained blood smear; this species occurs outside of Africa and is lowly pathogenic [4]

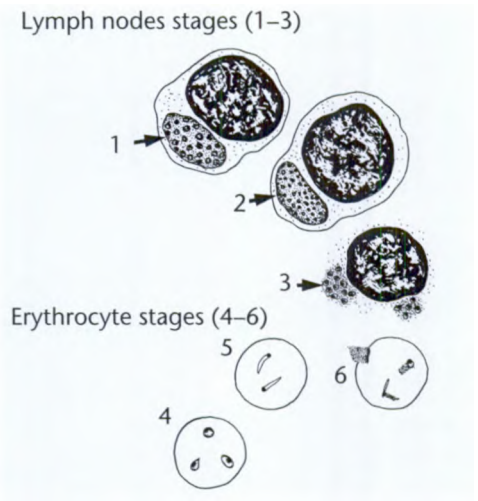

Fig. 114 Morphology of *Theileria* spp. stages in mammals; stages found in the lymph nodes: (1) Macroschizont with medium-sized nuclei (0.4–2 µm; Koch's blue bodies), (2) Microschizont with small nuclei (0.3–0.8 µm) and (3) Merozoites (0.7–1 µm); stages found in the erythrocytes: intraerythrocytic stages may be (4) circular, oval or piriform (0.8 x1.5 µm; 80%) or (5) comma-shaped (0.5 × 2 µm; 20%); (6) some parasites appear to have a velum [16]

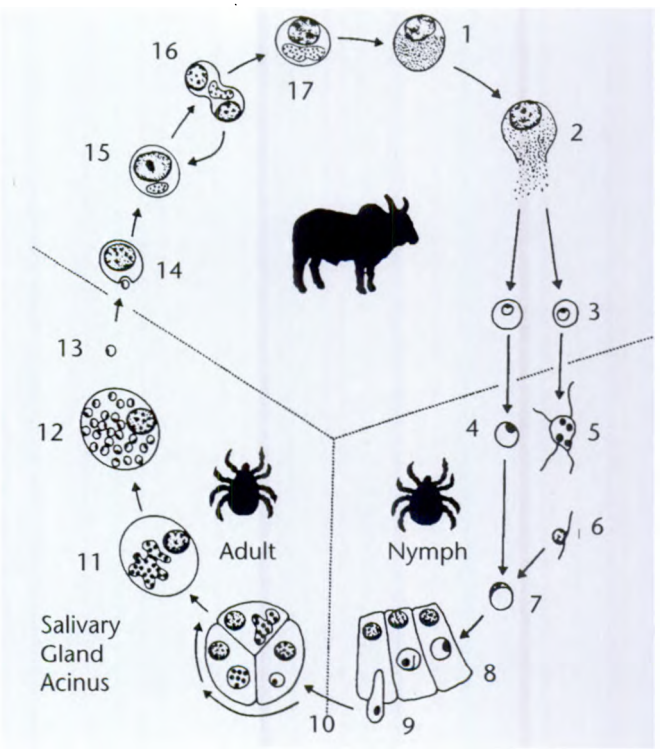

Fig. 115 Life cycle of *Theileria parva* [17]
(1) Merogony, (2) Merozoites (3) Piroplasm in erythrocyte, (4) Macromete, (5) Syngamy, (6) Microgamete, (7) Zygote in gut lumen, (8) Gut epithelial cells, (9) Kinete, (10) Moult, (11) Sporogony, (12) Sporozoites, (13) Sporozoite in saliva, (14) Sporozoite enters lymphocyte, (15) Schizont in lymphoblast, (16) Clonal expansion, (17) Schizont

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Theileria parva (syn. *T. bovis, T. lawrencei*)
East Coast Fever, bovine theileriosis,
Corridor Disease, Rhodesian tick fever

Hosts: Cattle, water buffalo, African buffalo

Vector: *Rhipicephalus appendiculatus* (equatorial East Africa and southern Africa above 1200 m) and *Rhipicephalus duttoni* (Angola plateau). Other vectors are *Rhipicephalus zambeziensis, Hyalomma excavatum, H. dromedarii, H. truncatum* and some other *Rhipicephalus* spp.

Species description: *T. parva* causes a disease called East Coast Fever (ECF), which is responsible for high mortality among susceptible and imported stock. The zebu in endemic areas has a high natural resistance, but animals imported into endemic areas are highly susceptible. Transmission of the parasite in all is on a stage to stage basis (trans-stadial transmission). There is no transovarial transmission in the tick vector as in *Babesia* spp. The organisms occur both in erythrocytes and in lymphocytes. The forms in the red blood cells are mainly rod-shaped, 1.5–2 × 0.5–1 µm, but round, oval, comma- and ring-shaped forms may also occur (Fig. 114, 4–6). Several parasites may occur in individual erythrocytes but there is no multiplication in the red cells. The multiplying forms of the parasite occur in the cytoplasm of lymphocytes and occasionally in the endothelial cells of the lymphatic glands and the spleen. These are schizonts, being circular or irregularly shaped structures about 8 µm in diameter. Two forms of schizonts are recognized. Those which contain large chromatin granules, 0.4–2 µm in diameter, are termed macroschizonts and produce macromerozoites, 2–2.5 µm in diameter (Fig. 114, 1). The other forms contain smaller chromatin granules, 0.3–0.8 µm in diameter, and are referred to as microschizonts and produce micromerozoites (Fig. 114, 2). The latter invade the red blood cells and may represent sexual stages of the parasite. After the intraerythrocytic stages are ingested by ticks, merozoites are liberated and differentiate into sexual stages. There is a sexual development of *T. parva* in the tick vector which results in infective sporozoites. Cattle are infected when vector ticks engorge on an animal and these infective particles (sporozoites) are transmitted. Most recovered animals remain carriers of infection.

Geographic distribution: East, Central and South Africa according to the distribution of the main vector *Rhipicephalus appendiculatus*.

Symptoms: High fever, swelling of the lymph nodes, dyspnoea and death. Fever occurs 7–10 days after the tick bite and continues throughout the course of the disease and may reach > 42°C. Lymph node swelling becomes pronounced and generalized. Anorexia, loss of condition, lacrimation and nasal discharge may occur. Terminally, dyspnoea is common. Just before death, a sharp fall in temperature occurs and pulmonary exsudate pours from the nostrils. Anaemia is not a major diagnostic sign as it is in babesiosis.

Significance: ECF is a serious disease with high mortality in susceptible stock.

Diagnosis: Lymphoblasts in Giemsa-stained lymph node biopsy smears (☞ METHODS, 3.1) during the acute febrile phase contain multinuclear schizonts. Lymph node enlargement and massive pulmonary oedema and hyperaemia are the most striking post-mortem lesions. Haemorrhages are common on the serosal and mucosal surfaces of many organs.

Therapy: Cattle with clinical ECF may be treated with parvaquone (2 × 10–20 mg/kg, im. at 48 h intervals),buparvaquone (2 × 2.5 mg/kg, im. at 48 h intervals) or with the coccidiostat halofuginone (2 × 1.2 mg/kg, po. at 48 h intervals). Chlortetracycline and oxytetracyline are relatively ineffective. None of these drugs will sterilize infections, and recovered animals may be carriers.

Prophylaxis: Immunization of cattle using an infection-and-treatment procedure is practical and relatively effective. The compo-

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nents for this procedure are cryopreserved sporozoite stabilates of the appropriate strains of *Theileria* derived from infected ticks and a single dose of either long-acting oxytetracycline or buparvaquone given simultaneously or of parvaquone given 1 week after infection. Cattle should be immunized 3–4 weeks before being turned out onto infected pasture. The incidence of ECF can be reduced by regular tick control, but in many areas this means biweekly acaricidal treatments. The use of pour-on acaricides is a promising way of tick control in endemic areas (☞ CATTLE, ■ 5.1) (Figures 116, 117, 118, 119, 120, 121, 122, 123)

Fig. 118 Lymph node swelling in a *Theileria parva* infected cattle [4]

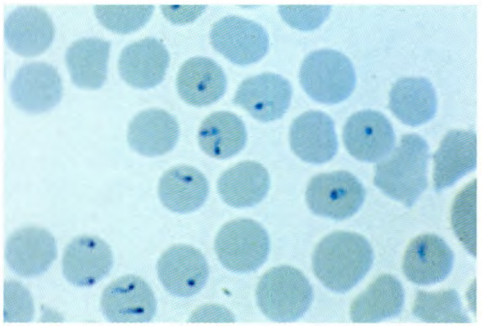

Fig. 116 *Theileria parva*; erythrocyte forms [4]

Fig. 119 Lacrimation and nasal discharges in a *Theileria parva* infected cattle [8]

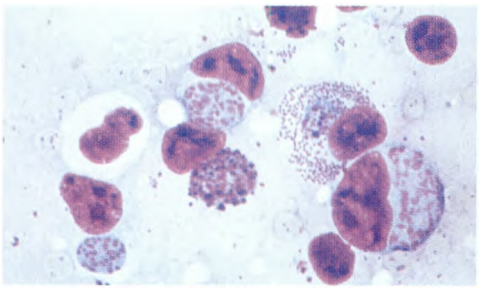

Fig. 117 *Theileria* sp.; macroschizonts ("Koch's blue bodies"and microschizonts in lymph node smears [4]

Fig. 120 *Theileria parva*; petechial haemorrhage on the intestinal serosa [8]

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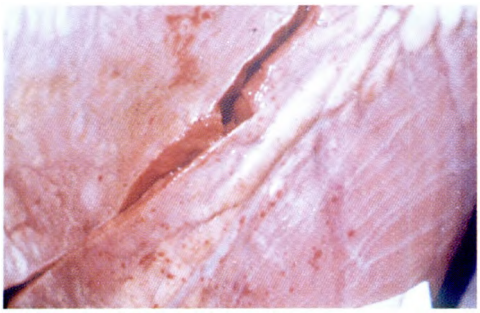

Fig. 121 *Theileria parva*; petechial haemorrhage on the surface (epicard) of the heart [8]

Fig. 123 *Theileria parva*; petechial haemorrhage on the kidneys [15]

Fig. 122 The reported distribution of *Theileria parva* [17]

Theileria annulata Mediterranean Coast Fever, Tropical theileriosis, Egyptian ever

Hosts: Cattle, water buffalo

Vector: *Hyalomma* spp. (*H. detritum, H. truncatum, H. anatolicum* and other *Hyalomma* spp.)

Species description: *T. annulata* produces a highly fatal disease of cattle in North Africa. Mortality rates may be up to 90% in some regions. This species occurs commonly in the lymphocytes and erythrocytes of bovine species. The organisms in the red blood cells are more or less indistinguishable from those of *T. parva*. They occur more commonly as round, oval- or ring-shaped (0.5–1.5 µm) forms. Rod shapes, commas (1.6 µm) and anaplasma-like organisms may also occur. Macroschizonts and microschizonts are found in the lymphocytes of the spleen and lymph nodes. *T. annulata* is transmissible by blood passage, and schizonts are numerous in the circulating blood. The development cycle in the vertebrate host is comparable to that of *T. parva*. The disease may be acute (duration: 3–4 days) or more chronic (duration: 2–3 weeks) in nature. The incubation period is 9–25 days. Animals that recover from *T. annulata* infections are resistant to reinfection. There is no cross-immunity between *T. annulata, T. parva* and *T. mutans*. The multiplication of *Demodex* sp. and *Besnoitia besnoiti* cysts in the subcutis is favoured by the immunosuppression produced by *Theileria* infections.

Geographic distribution: North Africa, Sudan, Mauretania, Ethiopia, southern Europe, southern UIS and Asia.

Symptoms: Fever (40–41.5°C), depression, lacrimation, nasal discharge and swelling of the superficial lymph nodes; emaciation rapidly occurs. A few days after the initial symptoms marked anaemia, jaundice (bilirubinaemia) develop. Haemoglobinuria may occur, but is not always present. In acute cases animals may die within 1–2

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days. In chronic infections a very heterogeneous complex of symptoms may develop over the period of 2 months until the animal recovers. Decreased milk production, digestive disturbances (cessation of rumination, diarrhoea or constipation) and infertility (abortion) may be seen. The severity of the disease does not necessarily correspond to the level of parasitaemia. Similarities with babesiosis may occur and must be differentiated.

Significance: *T. annulata* may cause mortalities of up to 90% in certain areas. It causes one of the most important diseases of cattle in endemic regions.

Diagnosis: This is based on the demonstration of parasites in the red blood cells or in stained smears (☞ METHODS, 3.1) taken from lymph node or spleen biopsies. The differentiation between *T. parva* and *T. annulata* organisms is difficult but the geographic spread of these two diseases is quite different. *T. annulata* often occurs together with *Babesia* spp. and/or *Anaplasma* spp. At necropsy the mucosa membranes of the abomasum and the small intestine show characteristic ulcers (2–12 mm in diameter), surrounded by an inflammation. Multiple petechiae may be found on the epi- and endocardium. The spleen, liver and lymph nodes are markedly enlarged.

Therapy: Tetracyclines have both prophylactic and therapeutic activity against *T. annulata*. Rolitetracycline (4 mg/kg, im.) administered daily during 4 days moderated the clinical signs markedly. Similar results were achieved by a single injection of a long-acting oxytetracycline formulation (20 mg/kg).

Prophylaxis: Immunization of cattle using an infection- and -treatment procedure is very effective and widely used. The application of 2×10^6 parasitised lymphoblasts obtained from subcultured *T. annulata* strains were inoculated into animals prior to exposure. No clinical signs developed and the animals were able to withstand heavy natural challenge. Another possibility is to apply a single injection of a long-acting oxytetracycline formulation (20 mg/kg) prior to exposure. Regular tick control of herds in endemic areas may markedly reduce the disease incidence (☞ CATTLE, ■ 5.1 THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 141).

(Figures 124, 125)

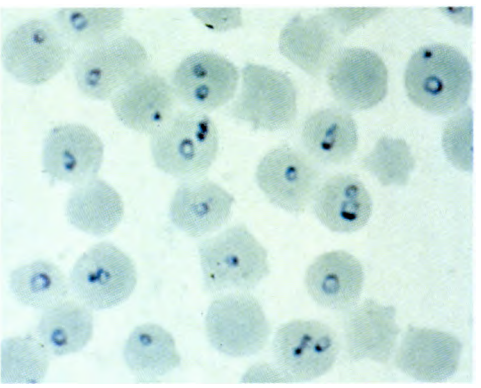

Fig. 124 *Theileria annulata*; erythrocyte forms (experimental infection) [4]

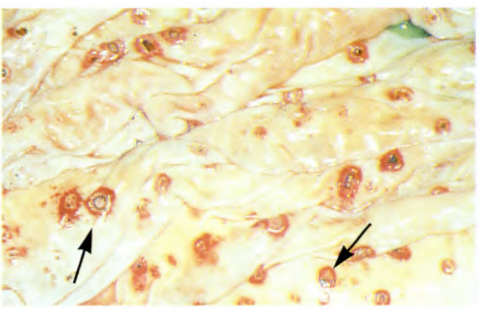

Fig. 125 Abomasal ulcerations caused by *Theileria parva* and *Theileria annulata* [15]

Theileria mutans Benign bovine theileriosis

Hosts: Cattle

Vector: *Amblyomma variegatum* and *Amblyomma hebraeum*

Species description: *T. mutans* causes a mild bovine theileriosis which is usually non-fatal. Morphologically it is not distinguish-

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able from the pathogenic *T. parva* and *T. annulata*. The forms in the erythrocytes are round, oval, piriform, comma-shaped or *Anaplasma*-like. The round forms are 1–2 µm in diameter and the oval forms measure 1.5 × 0.6 µm. Binary fission occurs in the erythrocytes. The incubation period is 10–20 days.

Geographic distribution: Throughout Africa, Madagascar, Réunion, Mauritius, southern Europe, Asia, Australia, UIS, Caribbean

Symptoms: Similar to a mild form of *T. annulata* infection. Anaemia is the major clinical sign. Icterus and lymph node swellings may occur.

Significance: *T. mutans* is usually only slightly- or non-pathogenic for indigenous cattle in endemic areas. An acute form may develop in cattle imported into an endemic area. The mortality is generally less than 1%.

Diagnosis: Same as for other *Theileria* species

Therapy: Unknown; the same drugs as for the pathogenic *Theileria* species may be used.

Prophylaxis: Tick control and immunisation as for the pathogenic *Theileria* species.

(Figures 126, 127, 128)

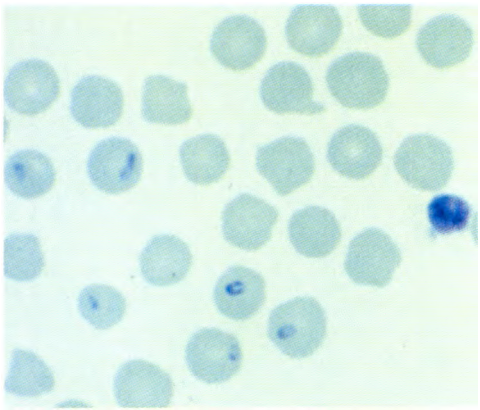

Fig. 126 *Theileria mutans*; erythrocyte forms [4]

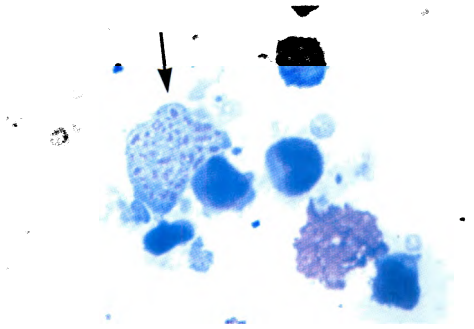

Fig. 127 *Theileria mutans*; macroschizont (rarely encountered) [15]

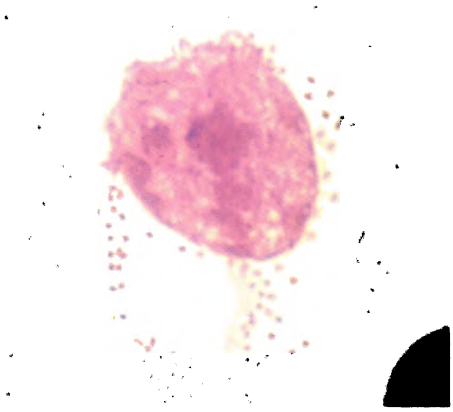

Fig. 128 *Theileria mutans*; microschizonts (rarely encountered) [15]

Theileria velifera

Remarks: This is another mild *Theileria* species of cattle in Africa and in the Caribbean. Its distribution coincides with that of the vector *Amblyomma variegatum* (Africa and Madagascar) and *Amblyomma lepidum* and *Amblyomma hebraeum* elsewhere. The parasites occur as pleomorphic or rod-shaped bodies in the erythrocytes and presumably lymphocytes. Most are 1–2 µm long. The great majority have a rectangular "veil" 1.0–3.5 µm long, extending out from the side. This species is nonpathogenic.

(Figure 129)

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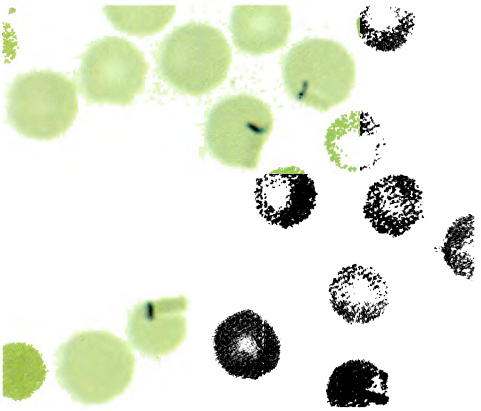

Fig. 129 *Theileria velifera*; erythrocyte forms [4]

Theileria taurotragi

Remarks: This species is found in African antelopes and can occasionally be found in cattle. It can also develop in the vector of *T. parva*. It is of low epidemiological importance.

RICKETTSIALES

ANAPLASMATACEAE

Anaplasma spp.

Anaplasmoses are infectious diseases, virulent, inoculable and not directly transmitted, that affect domestic and wild ungulates. The causative agent is a Rickettsia of the genus *Anaplasma*, usually transmitted by ticks, but which may also be transmitted mechanically by biting Diptera (*Tabanidae, Stomoxys*). The pathological signs are progressive anaemia (acute or slow) ending in cachexia and death. *Anaplasma* are exclusively found within erythrocytes. They are located intracellularly and surrounded by a vacuolar invagination of the host cell. The infection starts with an initial body that grows to become an elementary body. This multiplies by doubling or binary fission to produce new initial bodies. After several binary fissions, initial bodies accumulate in the vacuole to leave the host cell in order to parasitize other erythrocytes. The different *Anaplasma* species are morphologically indistinguishable. The criterion used is the location of the organism in the erythrocyte (marginal or central). (Figure 130)

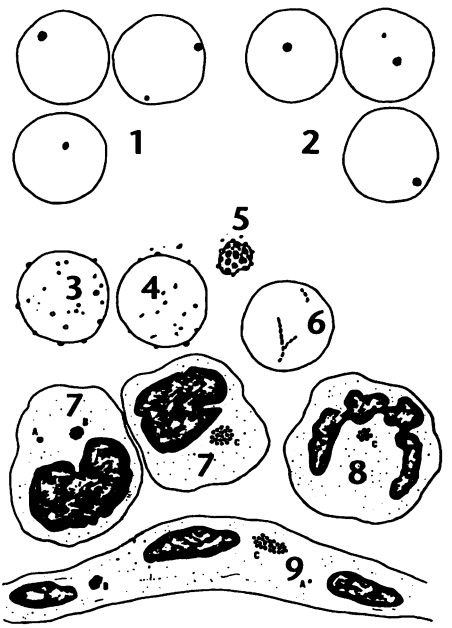

Fig. 130 Morphology (schematic) of various Rickettsiales found in ruminants;
Anaplasmataceae: (1) *Anaplasma marginale* (80–90% parasites marginal or peripheral); (2) *Anaplasma centrale* (80–90% of parasites central); (3) *Eperythrozoon wenyoni*, (on the erythrocyte surface); (4) *Eperythrozoon teganodes* (on the surface and near the erythrocyte); (5) *Eperythrozoon tuomii* (on the thrombocyte surface) and (6) *Haemobartonella bovis*;
Ehrlichiaceae: (7) *Ehrlichia bovis* (in monocytes); (8) *Ehrlichia phagocytophila* (in granulocyte) and (9) *Cowdria ruminantium* (in the vascular endothelium);
development stages: A = elementary body, B = initial body amd C = mass of elementary bodies [16]

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Anaplasma marginale The malignant anaplasmosis of cattle, Gallsickness

Remarks: 80–90% of the organisms are peripheral. *A. marginale* is distributed throughout the tropics corresponding to the distribution area of *Boophilus decoloratus* and *Boophilus microplus* usually considered as the main vectors. Transplacental infection occurs.
(Figures 130, 131)

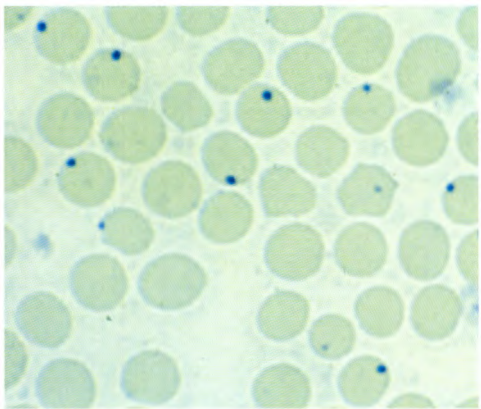

Fig. 131 *Anaplasma marginale*; Giemsa-stained bloodsmear [4]

Anaplasma centrale Mild anaplasmosis of cattle, Gallsickness

Remarks: *A. centrale* is predominantly located in the centre of the erythrocytes. The distribution and vectors are the same as in *A. marginale*. Anaplasmosis due to *A. marginale* is generally mild and often clinically inapparent.
(Figures 130, 132, 133)

Fig. 132 *Anaplasma centrale*; Giemsa-stained bloodsmear [4]

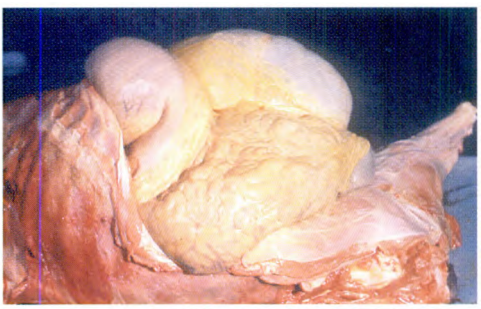

Fig. 133 Icterus associated with anaplasmosis (yellow abdominal fat) [15]

- **General features of bovine anaplasmosis**

Symptoms: Calves undergo mild infections, with little or no mortality. In yearlings the disease is more severe but most animals recover. In adult cattle, the disease is more severe, anaemia is marked and mortality may reach 50%. The disease starts with general depression, indolence, fever (40–41°C). Milk production falls rapidly. Weight loss and progressive anaemia, dehydration and icterus occurs. Affected animals often succumb to hypoxia when moved or handled for treatment. Recovered animals often remain carriers for life.
Significance: *Anaplasma marginale* causes severe losses in endemic areas.

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Diagnosis: In endemic areas, anaplasmosis should be suspected in adult cattle showing chronic anaemia without haemoglobinuria, leading to cachexia. Icterus is an important sign. Anaplasmosis often follows an acute form of babesiosis or theileriosis and appears to be a prolonged convalescence. Demonstration of the organisms in Giemsa-stained blood smears confirms the diagnosis (☞ METHODS, 3.1). Serological tests such as complement fixation and the IFAT and DNA probes are useful tools for diagnostic purposes. Anaemia due to helminth infections should be excluded by coprological examination.

Therapy: Acute anaplasmosis is most effectively treated with tetracyclines (oxytetracycline, 5–10 mg/kg, im. or iv.) or chlortetracycline (1.5 mg/kg, po.), especially if administered early in the course of infection. Long-acting oxytetracycline formulations may give sustained blood levels. Imidocarb dipropionate (1.2–2.4 mg/kg., sc.) may also be used for *Anaplasma* infections. Symptomatic and supportive treatment is important. Transfusion of 4–6 l of normal blood is often indicated and should be repeated in 48 h intervals, until the animal appears stronger. Oral and parenteral rehydration with isotonic saline and glucose solution is vital for dehydrated animals. Handling and disturbances should be avoided, since even mild exertion can produce hypoxia and death. Animals should be kept in shade with free access to fresh water.

Prophylaxis: Regular dipping, spraying of animals during the vector season drastically reduces the incidence of the disease. Several methods exist to immunize animals in endemic areas. Inoculation of blood containing *A. centrale* which gives rise to a mild infection that protects against a subsequent infection with the virulent and very pathogenic *A. marginale*. The use of virulent and attenuated *A. marginale* isolates to induce immunity or a chronic carrier status in calves is a widely accepted technique throughout the tropics where anaplasmosis is endemic. The use of virulent organisms in adult cattle is hazardous, and treatment at the onset of the patent infection with tetracyclines is recommended to temper the course of infection.

Eperythrozoon spp.

Organisms found on the surface of erythrocytes or near thrombocytes, occasionally they are located near erythrocytes. They multiply by binary fission. In general the size of *Eperythrozoon* is 0.4–1 µm. The most important vectors of *Eperythrozoon* spp. are sucking lice, infected through trans-stadial transmission. *Hyalomma anatolicum* possibly transmits *E. wenyoni*. *Eperythrozoon* spp. are generally not very pathogenic in spite of sometimes high infection levels. They may assume increased significance together with other protistan infections (*Babesia* spp., *Theileria* spp., *Anaplasma* spp. or *Trypanosoma* spp.). The only pathogenic *Eperythrozoon* species which produces a disease entity is *E. suis*. The disease is characterised by a slow development of anaemia, comparable to anaplasmosis. Oxytetracyclines (5–10 mg/kg, im. or iv.) are effective against all *Eperythrozoon* spp. Prophylaxis requires lice and tick control.

In cattle the following species are present in tropical Africa: *Eperythrozoon wenyoni* occurs on the erythrocyte surface; *Eperythrozoon teganodes* occurs on the erythrocyte surface or near to it and *Eperythrozoon tuomii* occurs on the thrombocyte surface.

(Figures 130, 134)

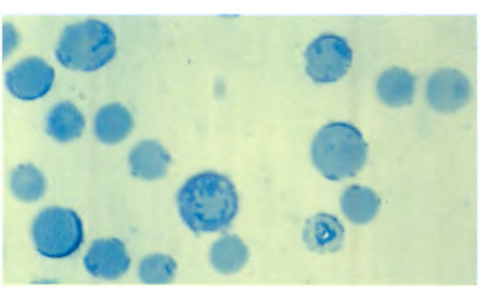

Fig. 134 *Eperythrozoon wenyoni*; Giemsa-stained bloodsmear [13]

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Haemobartonella spp.

These are organisms found on the surface of erythrocytes which form branched chains and multiply by binary fission. The organisms are coccoid (0.1–1 µm) or bacilliform (1–5 × 0.7–1.5 µm). They cause a defined disease entity in dogs and cats and rats. *Haemobartonella* spp. are often detected as resurgent parasites during concomitant infections or following splenectomy. Tetracyclines are the drugs of choice for treating *Haemobartonella* infections, but therapy is rarely indicated (☞ also above ANPLASMATACAE, Fig. 130).

Haemobartonella bovis

Remarks: It occurs on the erythrocyte surface. These coccoid or bacilliform organisms occur on the erythrocyte surface of cattle in Mediterranean regions. Vectors are sucking lice and ticks (species unknown). *Haemobartonella bovis* is relatively non-pathogenic. In rare cases the parasite may be associated with slow anaemia and symptoms similar to anaplasmosis. *Haemobartonella* spp. and *Eperythrozoon* spp. are difficult to distinguish reliably by light microscopy (☞ above ANPLASMATACAE, Fig. 130).

EHRLICHIACEAE

Ehrlichia bovis Tropical bovine ehrlichiosis, "Nofel" or "Nopel"

Hosts: Cattle

Vector: Ticks of the genera *Amblyomma*, *Hyalomma* and *Rhipicephalus*

Species description: *Ehrlichia bovis* occurs in mononuclear cells of cattle. The syndrome corresponds to the disease called "Nofel" (Fulani/Peule in West Africa; ear = nopi in peul) or "Nopel" (Bororos in Central Africa), because of the ears being kept low at the beginning of an acute infection. The clinical signs vary greatly ranging from inapparent forms to serious fatal forms, dominated by nervous signs, ataxic gait, stiff legs, walking in circles, phases of excitement followed by somnolence, epileptic crisis with falls, rolling eyes and salivation. Death may occur in an epileptic attack. The disease is always serious in imported cattle. The nervous signs are similar to those of cowdriosis.

Geographic distribution: North and Central Africa, Middle East, Ceylon

Symptoms: Anorexia, fever, incoordination and enlargement of lymph nodes

Significance: *Ehrlichia bovis* is of low significance for indigenous cattle. However, it is a serious problem for imported or newly introduced cattle in many tropical areas.

Diagnosis: Microscopic examination of peripheral blood smears demonstrates *E. bovis* in monocytes or macrophages. For differential diagnosis infections with *Cowdria ruminantium* have to be excluded. *C. ruminantium* is located exclusively in the endothelial cells (brain and other tissues) and not in the peripheral blood leucocytes, whereas *E. bovis* is found in monocytes and macrophages.

Therapy: Oxytetracyclines (5 mg/kg, im., for 4 days) or streptomycin-penicillin (3 g and 3 mio. units per day, im.); other tetracyclines may be used in emergencies.

Prophylaxis: This consists of regular tick control. There are no immunization procedures described.

(Figures 130, 135)

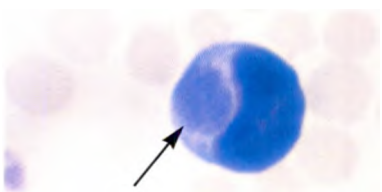

Fig. 135 *Ehrlichia bovis*; mass of elementary bodies in a monocyte [15]

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Ehrlichia phagocytophila
European ehrlichiosis

Remarks: *E. phagocytophila* occurs in the neutrophilic and eosinophilic granulocytes of cattle, sheep and other domestic and wild ruminants in Europe. Goats are not very susceptible. *Ixodes ricinus* transmits the disease by trans-stadial infection. The disease is characterized by fever which occurs after an incubation period of 4–11 days. The infection causes a state of immunosuppression and is therefore a predisposing factor to a variety of secondary infections. The animals usually recover after this febrile phase. However, the most characteristic problems are abortion in pregnant ewes and cattle, and progressive weight loss following infection may occur. At necropsy hypertrophy of the spleen and lymph nodes may be seen. The diagnosis is made by demonstrating the organisms in stained blood smears (☞ METHODS, 3.1). Serological tests such as IFAT (☞ METHODS, 5.1) may be used for epidemiological studies.

Therapy: Long acting oxytetracyclines (5 mg/kg, im.) have a certain curative effect.

Prophylaxis: ☞ above *Ehrlichia bovis*

(Figures 130, 136)

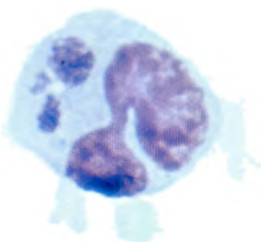

Fig. 136 *Ehrlichia phagocytophila*; mass of elementary bodies in a granulocyte [4]

Ehrlichia ondiri

Remarks: This parasite occurs in granulocytes of cattle in high-altitude grassland regions of East Africa. Members of the genus *Haemaphysalis* and other genera are suspected to be vectors of the disease. The incubation period is 1–2 weeks. The disease starts with a fever attack which is accompanied by a petechial exanthema of the mucosae of the mouth, nose, vulva, eyes (poached egg eye), digestive tract, urinary bladder, pericardium and endocardium. Extended haemorrhage in the gastrointestinal tract and the pulmonary oedema cause death.

Diagnosis is made by microscopic examination of blood smears. Organisms of *E. ondiri* are found in granulocytes.

Therapy and Prophylaxis: ☞ above *Ehrlichia bovis*

Cowdria ruminantium Heartwater, Tyewde

Hosts: Cattle, sheep, goat and other ruminants

Vector: Several species of the tick genus *Amblyomma* transmit the disease: *Amblyomma variegatum* in open savannas from equatorial Africa to Madagascar; *Amblyomma gemma* in East Africa (Massai and Somalian steppes); *Amblyomma pomposum* has limited geographic distribution and occurs in the Angolan plateau, Zambia, southwestern Tanzania; *Amblyomma lepidum* in East Africa (Massai steppes, Sahelian steppes of the Nile in Sudan) and *Amblyomma tholloni* in the Zambezi region. *C. ruminantium* is also present on the Caribbean islands.

Species description: Cowdriosis is specific to ruminants of the family Bovidae and is most severe in sheep and goats but also causes great losses in cattle. Imported ruminants are much more susceptible to cowdriosis than indigenous breeds. They may suffer heavy losses. But also indigenous ruminants are affected especially when they are in poor general condition (endoparasitism, malnutrition, chronic infections, etc.) or with weakened defence (lactation, gestation). This applies especially to the early rainy season in the Sahel-Sudanese region when animals are weakened by the long food shortage of the dry season and when there is a drastic

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proliferation of the *Amblyomma* ticks. Cowdriosis can then become a great problem. The incubation period is 8–15 days. Endothelial cells are the preferred sites of the proliferation of this pathogen, especially of the nervous system, kidney, spleen, lymph nodes, salivary glands and heart muscle. Young calves (< 6 weeks), lambs and kids (< 1 week) are fairly resistant and may recover spontaneously.

Geographic distribution: The distribution of heartwater corresponds to that of all its vectors.

Symptoms: The symptoms are almost identical in both cattle and small ruminants, except that the nervous symptoms are rarer in cattle and not as distinct (e.g. posture of pushing against a wall, ataxia, haggard eye, aggressiveness). Digestive signs are more constant in cattle than in sheep and goats. Diarrhoea may occasionally be seen. Several forms of cowdriosis can occur, ranging from a peracute form (death within 2–3 hours; tyewde of the Fulani) to inapparent infections. There are no pathognomonic signs. In the peracute form an animal may abruptly drop to the ground, struggle, start to pedal, indicating serious nervous disorders. Death follows rapidly. The acute form can last for up to 5 days and is accompanied by initial hyperthermia, breathlessness and dyspnoea. Nervous signs may occur, often at the end of the disease, suggesting tetanus or poisoning. Fluttering of the eyelids and acute gastroenteritis may also occur. The subacute form is rare (12–15 days), developing sometimes pulmonary complications. Attenuated forms are frequent. Animals recover after a short febrile phase. Sometimes, hyperthermia is the only clinical sign.

Significance: Cowdriosis is a serious problem in areas infested by the ticks *Amblyomma* spp.

Diagnosis: In acute forms diagnosis can be based on clinical signs. Demonstration of colonies of the organisms in the cytoplasm of capillary endothelial cells is necessary for definitive diagnosis. This is done with Giemsa-stained "squash" smears of cerebral gray matter. An indirect IFAT (☞ METHODS, 5.1) test may be used to detect antibodies of animals that have recovered from infection. In sheep and goats these antibodies persist for several years, and their presence correlates well with their resistance to reinfections. Seropositive cattle are resistant to challenge, although antibodies disappear within 12 months after infection.

At necropsy hydropericardium is the predominant sign. The exudate is clear, usually amber or yellow. It is a consistent sign of heartwater. It may be as much as 700 ml in small ruminants. Hydrothorax, ascites and splenomegaly are often found. The gall bladder is usually distended. Extensive congestion of the viscera and petechiae on the serous membranes and mucosae of the digestive tract may be seen. Microscopically the deformation of endothelial cells by rickettsial masses is the most striking finding. Smears are made either with material scraped from the internal wall of a major artery, or with a fragment of cerebral cortex crushed between two slides. May-Grünwald/Giemsa staining usually reveals masses of elementary bodies, appearing as blue granules in the cytoplasm of endothelial cells. The initial bodies are dark blue.

Therapy: Oxytetracycline (5–10 mg/kg, im. or iv.) usually effects a cure if administered early. Later in the febrile phase, when clinical signs appear, a higher dose level (10–20 mg/kg) is required.

Prophylaxis: There is no vaccine yet. Several immunization schedules have been applied successfully ("controlled immunization": infection with a laboratory-maintained strain followed by antibiotic treatment) but they require a very careful monitoring of the infected animals and laboratory maintenance of parasite strains.

These methods are expensive and should therefore be reserved for valuable animals (e.g. breeding stock, drought animals, etc.). Tick control is a substantial part of heartwater prophylaxis.

Remarks: Merozoites of *Toxoplasma gondii*

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and *Sarcocystis* spp. may occur in the blood stream during the acute phase.
(Figures 130, 137, 138, 139, 140)

Fig. 137 Nervous symptoms associated with *Cowdria ruminantium* [15]

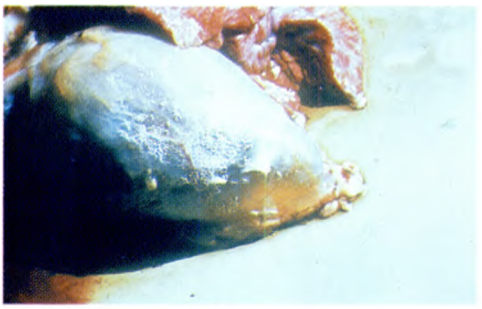

Fig. 138 Hydropericardium (heart water) caused by *Cowdria ruminantium* [15]

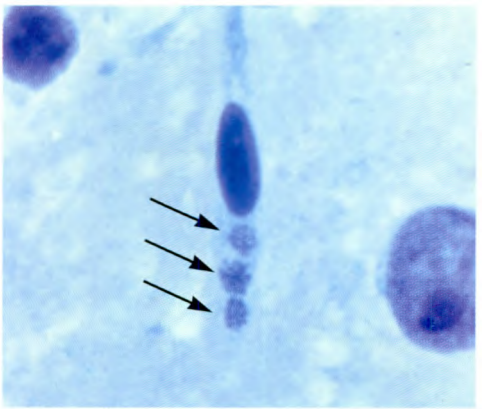

Fig. 139 *Cowdria ruminantium* organisms in the capillary endothelial cells [4]

Fig. 140 The known distribution of the known vectors of heartwater in the African region [13]

HELMINTHS

- **Trematoda found in the blood and circulatory system**

Schistosoma bovis Blood fluke, Bilharziosis

Location: Mesenteric veins
Hosts: Cattle, sheep, goat, equine and dromedary
Species description: *S. bovis* occurs in the portal and mesenteric veins of cattle, sheep, goats, equines, camels and rarely in man. The males of *S. bovis* are 9–22 mm long and 1–2 mm wide. The females are 12–28 mm long. The eggs are spindle-shaped and passed in the faeces. The size of the eggs is 132–247 µm × 38–60 µm.
Geographic distribution: Central, East and West Africa
(Figures 141, 142)

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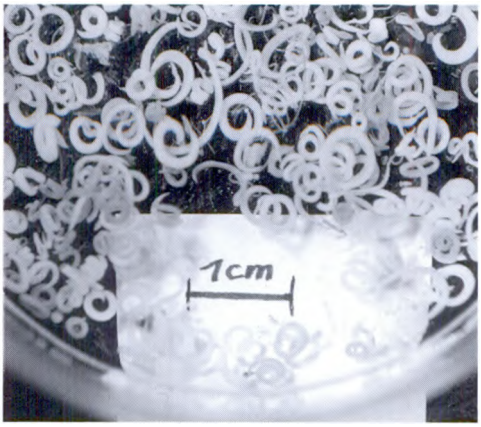

Fig. 141 *Schistosoma bovis*

Fig. 142 *Schistosoma bovis*; male (thick) and female (slender)

Schistosoma mattheei

Location: Portal and mesenteric veins and less frequently cystic and lung veins

Hosts: Cattle, sheep, goat, wild ruminants, equine and man

Species description: Females measure 18–25 mm and males 9–14 mm. The eggs measure 170–280 × 70–85 µm and have a terminal spine. The prepatent period is 7 weeks. The life cycle is similar to *Schistosoma bovis*. Intermediate hosts are *Bulinus africanus*, *Bulinus globosus* and other *Bulinus* spp.

Geographic distribution: Southern Africa, Mozambique, Zambia, Malawi, Zimbabwe, Tanzania; Central Africa (Tchad)

Schistosoma intercalatum

Remarks: This species occurs mainly in man and primates in Central Africa. Sheep and goats and other domestic and wild ruminants may accidentally acquire infection. *S. intercalatum* is found in the portal and mesenteric veins. The eggs measure 140–240 µm × 50–85 µm.

Schistosoma curassoni

Location: Portal and mesenteric veins

Hosts: Cattle, sheep and goat

Species description: Intermediate hosts are snails of the genus *Bulinus*. Eggs are similar to those of *S. mattheei*.

Geographic distribution: West Africa (Senegal, Mauretania, Mali)

- **General features of *Schistosoma* spp. infections**

Life cycle: Aquatic snails (*Bulinus* spp.) act as intermediate hosts in the life cycle of *Schistosoma* spp. In the case of *S. bovis*, *S. mattheei* and *S. intercalatum* snails of the genus *Bulinus* spp. act as intermediate hosts. The eggs hatch after contact with water. The miracidia infect aquatic snails and cercariae emerge 38–126 days after infection from the snail and actively penetrate the skin or the rumen of the definite host. Schistosomosis (bilharziosis) occurs near permanent fresh water bodies (pools, backwaters, ponds, lakes and marshy pastures). To become infected, the final host needs the contact with water. This might occure during drinking in these areas, passing through water or grazing in swamps.

Symptoms: These are rarely seen even in heavy infections. Symptoms are mainly caused by the eggs which are deposited by the adult flukes in the wall of the intestines. Intermittent diarrhoea which may contain specks of blood, anaemia and progressive weakness may occur. Both diarrhoea and constipation can be seen and blood and mucus are sometimes found in the faeces together

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with the eggs. A chronic hepatic syndrome has been described with neurological signs like ataxia, hypermetria, disorientation, leaning against walls, coprophagy, etc. Sheep are more susceptible than cattle and can die in large numbers while cattle survive, unless they have very heavy burdens.

Significance: Although specific clinical signs are often overlooked, blood flukes may constitute a serious problem in endemic areas.

Diagnosis: This is made by faecal examination. Spindle-shaped eggs are found in the sediment. In chronic cases, it may not be possible to find eggs in the faeces and the diagnosis must be confirmed by a post-mortem examination. At necropsy adult flukes can be found in the mesenteric veins or in the portal system.

Therapy: The therapy of schistosomosis is not easy. Highly efficient anthelmintics may have fatal consequences because dead worms may cause massive thrombus formation in the liver. Therapy should aim to kill the worms over a long period of time rather than risk the massive damage caused by the simultaneous death of all worms. In general treatment of schistosomosis is uneconomical since a large number of repeated doses at intervals of 2–3 days is required. Praziquantel at 25 mg/kg body weight is effective, although two treatments, 3–5 weeks apart are required. Other drugs which have some schistosomicidal properties are stibophen (7.5mg/kg, im. 6 ×), lucathone hydrochloride (50 mg/kg for sheep and 30 mg/kg for cattle; both every other day until three treatments have been given), hycanthone (6 mg/kg, im.) and trichlorfon (10–12 mg/kg, im. 11 × at intervals of 3–5 days).

Prophylaxis: In areas where schistosomosis is of great importance, grazing in swamps, rice fields, ponds (habitat of the intermediate host) should be avoided. Water from bore holes should be used for drinking purposes. Infection can be reduced by fencing off contaminated bodies of water and providing clean drinking water. These measures not only reduce the incidence of schistosomosis, but they also control other trematodes such as *Fasciola* spp. and paramphistomes which frequently occur in the same localities as schistosomes. If the drinking water is contaminated with intermediate snail hosts the use of molluscicides may be indicated. An ecologically justifiable way will probably be the use of molluscicidal plants which can be cultured in water bodies containing the snail-intermediate host.

(Figures 31, 35, 36, 143, 144)

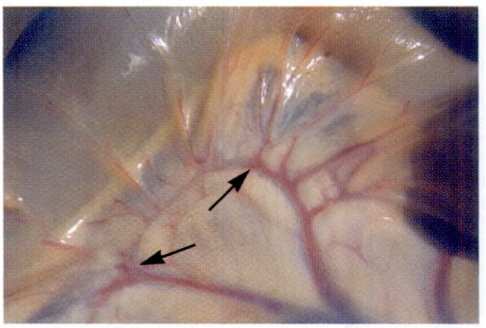

Fig. 143 *Schistosoma bovis* in the mesenteric veins

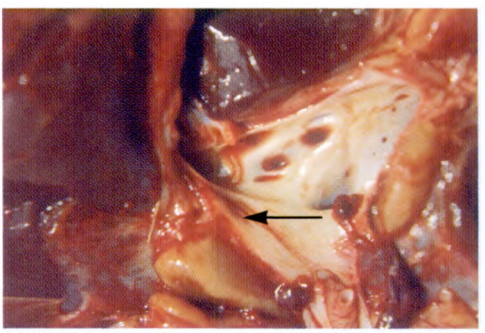

Fig. 144 *Schistosoma bovis* in the portal vein of the liver

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- Nematoda larvae (microfilariae) found in the blood and adult nematodes living in the circulatory system

Onchocerca armillata Aortic filariosis

Location: Adult worms are found within tunica media of the aorta. Microfilariae are found in the blood stream (transiently) and in the skin of the dorsal parts of the body (definitively).

Hosts: Cattle, rarely small ruminants and camel

Species description: Adults are found in the aorta. Males are about 7 cm long and females up to 70 cm. The microfilariae are unsheathed, measure 346–382 µm × 5.5–6.8 µm and are mainly found in and under the skin on the dorsal parts of the animal (hump, wither, neck, rarely dewlap and lower parts). In chronic infections the aortic wall is thickened and the intima shows tortuous tunnels and there are numerous nodules containing yellow caseous fluid and coiled worms. Infection is transmitted by bites of insects of the families Simuliidae and Ceratopogonidae. The infection is very common (up to 90%) in some areas.

Geographic distribution: Throughout Africa

Symptoms: Clinically mostly inapparent; calcification of the aortic wall and aneurysm may cause cardiovascular symptoms.

Significance: Weakly pathogenic but prevalent in some regions (80–90% of all animals examined)

Diagnosis: Microfilariae appear in the blood stream and in the skin. Typical nodular lesions in the aortic wall may be found at necropsy.

Therapy: This is rarely indicated. Diethylcarbamazine is microfilaricid (5–8 mg/kg daily for 21 days). Ivermectin (200 µg/kg) was found to be highly effective against the microfilariae of many *Onchocerca* spp.

(Figures 145, 146, 159)

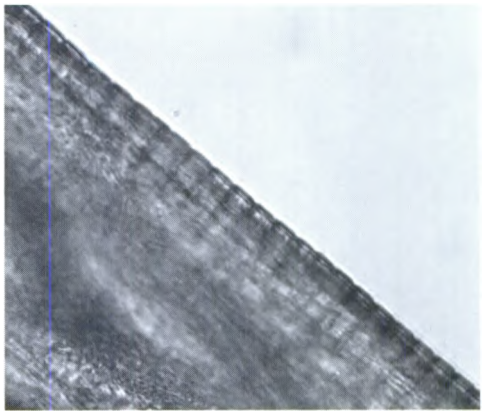

Fig. 145 *Onchocerca armillata*; typical striated cuticula

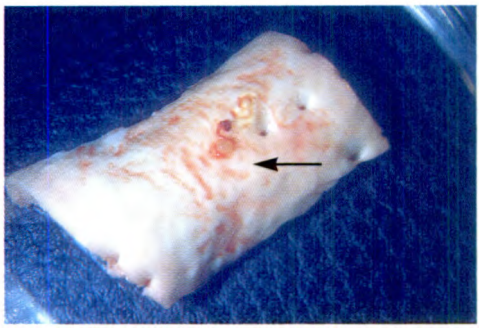

Fig. 146 *Onchocerca armillata*; aortic nodules containing the coiled adult worms (arrow)

Onchocerca spp., *O. gutturosa, O. dukei* and *O. ochengi*

Remarks: These species are found in the nuchal ligament or in/under the skin on several parts of the body (☞ CATTLE, ■ 4.1.2 and 5.1). Microfilariae may occasionally be found in the circulating blood during their migration to their predilection site.

Elaeophora poeli Large aortic filaria

Location: Thoracic portion of the aorta

Hosts: Cattle, water buffalo

Species description: Males are 45–70 mm long and females 40–300 mm. The microfilari-

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ae are 340–360 µm long and 7.0–7.5 µm wide. The male occurs in nodules in the wall of the aorta, while the female is fixed in the nodules with its anterior extremity and the rest of its body hangs free in the lumen of the vessel. The females are 350 µm wide in the intranodular part of the body and 1.5–1.8 mm wide in the free parts of the body. The life cycle is unknown. Affected vessels are swollen and less elastic. The intima is uneven and rised by nodules which are 8–13 mm in diameter.

Geographic distribution: Central and East Africa, South East Asia, Philippines

Symptoms: There is no evidence that the parasite causes clinical signs.

Significance: Unknown

Diagnosis: Nodules in the aorta, unsheathed microfilariae may occur in the blood stream.

Therapy and Prophylaxis: Not indicated

(Figure 147)

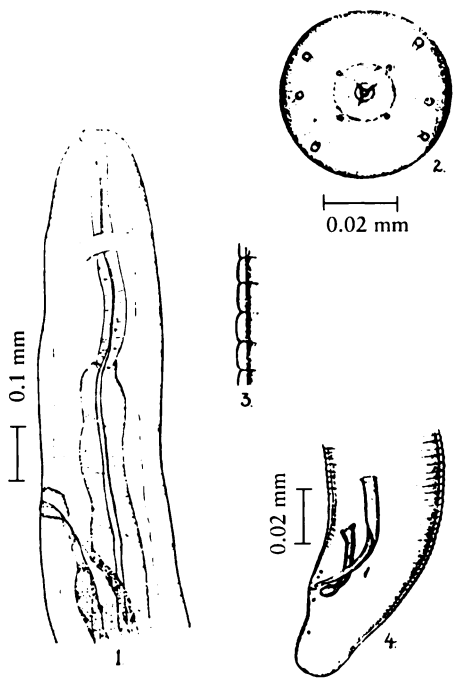

Fig. 147 *Elaeophora poeli*; anterior end of a female (1); anterior end (2), cuticular structure (3) and tail end (4) of a male [18]

Setaria labiatopapillosa

Location: Adult worms occur in the abdominal cavity (☞ CATTLE, ■ 4.4).

Microfilariae are sheathed, 140–230 µm long and appear in the peripheral blood.

(Figure 148)

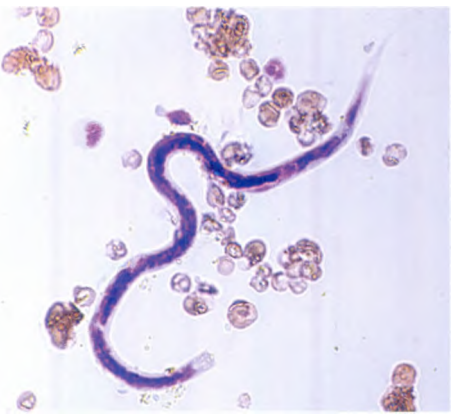

Fig. 148 *Setaria labiatopapillosa*; sheathed microfilariae are 140–230 µm long; Giemsa-stained blood-smear

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PROTOZOA

Tritrichomonas foetus (syn. *Trichomonas foetus*) Bovine trichomonad abortion

Location: In the bull the parsites are found in the preputial cavity. In cows trichomonads are found in the vagina and uterus.

Hosts: Cattle

Species description: This is a venereal protozoal disease of cattle characterized by early foetal death and infertility, associated with greatly extended calving intervals. It is reported from many tropical countries. The organism is ordinarily 10–15 × 5–10 µm, but there is considerable pleomorphism. At the anterior end there are three flagella about the same size as the parasite. An undulating membrane runs the full length of the body and is bordered by a filament which continues beyond the membrane as a posterior flagellum. Multiplication is by longitudinal binary fission. Under natural conditions the infection is transmitted during coitus from the bull to the cow or vice versa. It may also be transmitted mechanically, e.g. by gynaecological examination of cows with contaminated tools. The organisms are found only in the genital tract of the cow and bull. Most bulls remain permanently infected after a primary infection. The bull is usually the source of initial and continuous infection within a herd. Infected cows usually recover spontaneously after mean durations of 20 weeks (primary exposure) and 10 weeks (secondary exposure). Cows can remain infected throughout pregnancy and discharge trichomonads from the genital tract following calving.

Geographic distribution: World-wide

Symptoms: Purulent vaginitis, metritis, placentitis and abortion are commonly found in infected cows and can cause enormous losses. Prolonged sterility and extended calving intervals are predominant features (*cave:* differential diagnosis: *Brucella abortus*!). There is a persistent uterine discharge containing large numbers of the parasites. Bulls are usually carrier of the parasite without clinical symptoms.

Significance: Venereal disease of cattle characterised by abortion and infertility in cows. *T. foetus* can cause considerable losses in infected herds. In many countries the incidence has greatly been reduced by the artificial insemination using a bull known to be free of the disease.

Diagnosis: History of abortions in a herd and demonstration of the organism in the vaginal or uterine discharges or in the foetus (mainly stomach). In bulls the organisms are in the prepuce, frequently in small numbers. Microscopical examination of the preputial smegma for trichomonads is the most common method to confirm a herd diagnosis. Preputial fluid may be collected with a pipette (e.g. AI pipette) which is introduced into the prepuce, and smegma is collected with a combination of scraping and aspiring via an attached rubber bulb or syringe. The same procedure may be used to collect vaginal fluid. It is important to avoid contamination with faecal material, as such material contains masses of protozoa similar in appearance to *T. foetus*. The organisms may not be numerous and careful, systematic examination is necessary. Diagnosis is based on the size and shape of the organisms as well as the characteristic aimless, jerky motion on a warmed slide. Only living organisms are useful for diagnostic purposes. Confirmation depends on finding the organisms in at least one animal in a suspected herd. The organisms may be cultured in special media (e.g. Diamond's Trichomonad Medium). The diagnosis can be confirmed by the Polymerase Chain Reaction (PCR) which is the most sensitive and specific diagnostic method nowadays. Serological examinations on a herd level may be used for epidemiological purposes but are of little value for individual animals.

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Therapy: This is extremely difficult and does not eliminate the parasites. Compounds which have been reported to be effective against trichomonads are dimetridazole, ipronidazole and metronidazole. Thorough rinsing out of affected genital organs with acridine and iodine preparations may reduce the number of parasites but it does not clear the infection. The only effective and sustainable way to control bovine trichomonosis is strict prevention.

Prophylaxis: Control measures are based on the assumption that transmission occurs only during coitus and that infected cows that undergo 3 months of sexual rest after calving with normal uterine involution are free of infection. Animals with pyometra or genital abnormalities should be culled. Artificial insemination (AI) with semen free of trichomoads is the most effective way of controlling the disease. However, if AI is not possible, the exposed animals should be treated repeatedly for recognizable uterine disease and allowed 3 months of sexual rest. For breeding, a young, not infected bull should be used. Bulls and cows should be examined for reinfection. Control may be supported by eliminating all bulls > 3 years old and using younger bulls for mating. This is based on the relative lack of susceptibility of young bulls to trichomonad infection. Slaughter, rather than treatment, of bulls is generally recommended.

(Figures 149, 150)

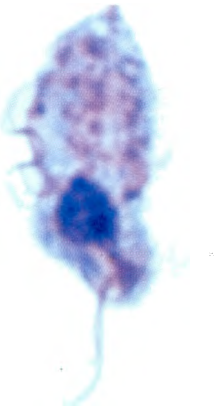

Fig. 149 *Tritrichomonas foetus* (12–18 × 6–10 µm); Giemsa-stained bloodsmear [4]

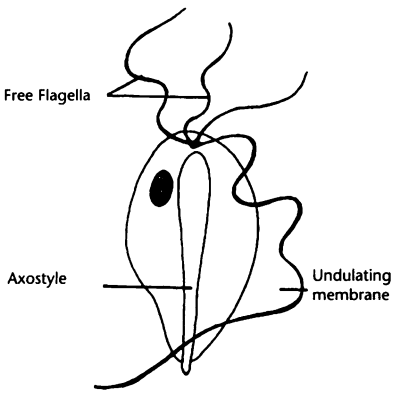

Fig. 150 Morphology of *Tritrichomonas foetus* (schematic) [19]

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4.1 Locomotory system

4.1.1 Muscles

4.1.2 Tendons

4.2 Liver

4.3 Respiratory system

4.4 Abdominal cavity

4.5 Pancreas

4.6 Central nervous system

4.1 Locomotory system

4.1.1 Muscles

PROTOZOA

Sarcocystis bovicanis (syn. *S. cruzi*), ***Sarcocystis bovifelis*** (syn. *S. hirsuta*) **and** ***Sarcocystis bovihominis*** (syn. *S. hominis*)
Bovine Sarcosporidiosis

Location: Cysts, containing the infective stage, called bradyzoites, occur in the striated musculature of cattle.

Hosts: Cattle are intermediate hosts of three *Sarcocystis* species of which the dog (*S. bovicanis*), the cat (*S. bovifelis*) or man (*S. bovihominis*) are final hosts.

Species description: Cysts of several *Sarcocystis* species are found in the muscles of cattle. Cattle always act as intermediate hosts. *S. bovicanis* is the most pathogenic species for cattle. Infection is acquired orally by ingestion of sporocysts from the faeces of infected hosts. Tissue cysts in cattle are infectious for at least 1 year. The life cycle of *Sarcocystis* spp. is as follows: infective sporocysts, excreted by the final host (cat, dog, man) are ingested, sporozoites are released in the intestine and invade many tissues via blood stream. Schizogony occurs in the endothelial cells of blood vessels in most organs, preceding the development of typical cysts in the striated musculature. Cysts may occasionally also be found in the brain. Cysts vary in size, depending on the species, from a few mil-

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Table 10 *Sarcocystis* species found in muscles of cattle

Species	Synonym	Definite host	Pathogenicity IH*	DH**
S. bovicanis	*S. cruzi*	Dog, coyote, wolf	+++	0
S. bovifelis	*S. hirsuta*	Cat	0	0
S. bovihominis	*S. hominis*	Man, rhesus monkey, baboon, chimpanzee	0	+

*IH = Intermediate Host; **DH = Definitive Host; 0 = non-pathogenic; + = mildly pathogenic; +++ = severely pathogenic

limetres to several centimetres in length. The peripheral region of a cyst contains globular forms (metrocytes), which produce two daughter cells by endodyogeny and these after further replication give rise to banana-shaped bradyzoites. Infection of the final host is by ingestion of muscle cysts, containing bradyzoites. *S. bovifelis* is nonpathogenic for cattle and produces microscopic cysts of 12 × 1 µm. *S. bovihominis* has man and some primates as definitive hosts and the sarcocysts are compartmented, with a wall of about 6 µm tick that appears radially striated by light microscopy. The pathogenicity of *S. bovihominis* for cattle is still in debate. The following description refers to this species. Following ingestion of sporulated oocysts or sporocysts by cattle (excreted by dogs, wolves, coyotes, raccoons, foxes, hyenas) several generations of schizonts occur in the endothelial cells of blood vessels and metrocytes are found in the striated musculature about 1 month after infection. Fully developed muscle cysts, containing the infective bradyzoites occur 2–3 months after infection.

Geographic distribution: World-wide

Symptoms: Anorexia, intermittent fever attacks, anaemia, weight loss were observed after experimental infection of calves. Deaths occurred about 30 days after a heavy infection with 10^5–10^6 sporocysts. Reduced milk yield, loss of condition, dyspnoea and abortion were observed in adult cattle.

Significance: *Sarcocystis* infection occurs throughout the world. Economic losses during a fresh infection may be higher than expected. Once muscle cysts are formed the infection is inapparent.

Diagnosis: This is very difficult in the acute phase (1 month post infection). During this period schizonts may be demonstrated in vascular endothelial cells by means of stained-scrapings taken from the blood vessels or by histopathological examination. Detection of meronts in Giemsa-stained blood smears (☞ METHODS, 3.1) or biopsy for detection of schizonts. At necropsy artificial digestion of affected muscles might reveal the muscle cysts. Serological tests (IFAT, ELISA; ☞ METHODS, 5.1 and 5.2) are indicated after the acute phase. The PCR (☞ METHODS, 6.3.2) may be used as a highly sensitive method to detect *Sarcocystis* infections.

Therapy: This is extremely difficult in cattle and rarely indicated. Amprolium (100 mg/kg, po., daily during 30 days) was effective to avoid clinical signs in cattle (endothelial schizogony) during experimental infections. Amprolium and halofuginone (0.66 mg/kg, po., 2 × on consecutive days) may be used in sheep and goats to avoid clinical disease after infection.

Prophylaxis: The contamination of cattle food with faeces of dogs, cats and man as well as the feeding of raw cattle meat to dogs should be avoided.

(Figures 151, 152, 153, Table 10)

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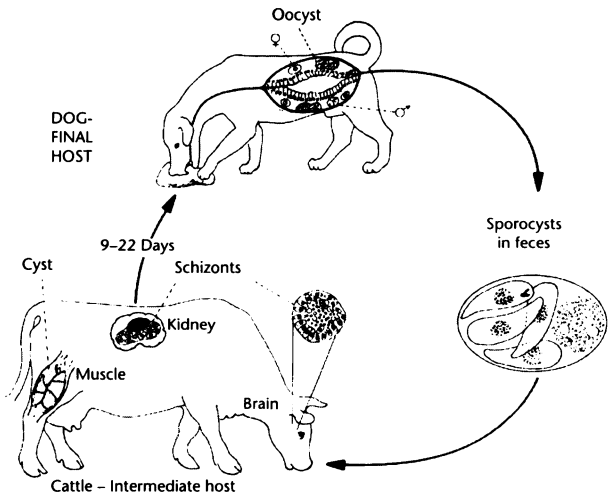

Fig. 151 Life cycle of *Sarcocystis bovicanis* [3]

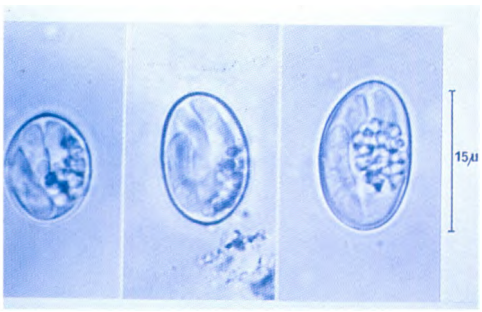

Fig. 152 Sporozoites of *Sarcocystis suicanis* (left), *Sarcocystis ovicanis* (middle) and *Sarcocystis bovicanis* (right) [4]

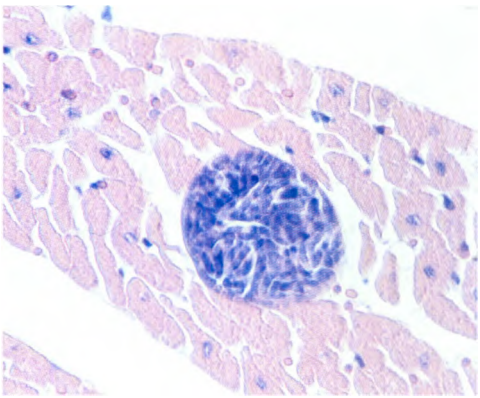

Fig. 153 *Sarcocystis bovicanis* (syn. *S. cruzi*); tissue cyst (70 µm in diameter) [10]

Toxoplasma gondii

Remarks: Tissue cysts of *T. gondii* may also occur in ruminants as they do in pigs (☞ SWINE, 4.1). A diagnosis based on clinical signs is not possible. Cysts may be seen in histopathological examinations. High antibody titers by the Sabin-Feldman-test, IFAT or ELISA (☞ METHODS, 5.1 and 5.2) may indicate an infection. The PCR technique (☞ METHODS, 6.3.2) may be used for the sensitive detection of *T. gondii* infections in all domestic animals species and will thus provide an important epidemiological tool. Tissue cysts of *T. gondii* in cattle may constitute an infection risk of minor importance for man.

Neospora sp.

Remarks: Cysts of *Neospora* sp. are found in the muscles of cattle. The entire life cycle is still unknown. Until 1988 the parasite was diagnosed as *T. gondii*. *N. caninum* causes neuromuscular disorders in dogs and is the main cause of abortion in cattle in the

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USA. Tachyzoites and tissue cysts are the only stages so far known. For diagnosis and treatment ☞ CATTLE, ■ 4.6.

HELMINTHS

- Cestoda larvae found in the muscles

Cysticercus bovis Larvae of the human tapeworm *Taenia saginata* cysticercosis, "beef measles"

Location: Cysticerci (larval stage) are found in striated and non-striated muscles of cattle, anywhere in the body. Adult tapeworms are found in the small intestine of man.

Intermediate hosts: Cattle

Species description: The final host of this parasite is man. Cattle serve as intermediate hosts. In cattle the parasite appears as small, fluid-filled cysts (10 mm × 4.5 mm) which give a spotty or measle-like appearance to the beef muscle. These cysts contain 1 protoscolex and occur predominantly in the heart muscle, diaphragm, tongue and masseters. In infected man proglottids are passed in the faeces. Infection of cattle occurs by ingestion of proglottids or eggs. The larvae (oncospheres) hatch in the intestine and penetrate the intestinal wall and are carried via blood stream to muscles where they become bladder worms 8–20 weeks after ingestion. Some of the cysticerci may become calcified and are no longer infective. Man is infected by eating uncooked measly meat of cattle containing viable cysticerci. Adult tapeworm develop in man in 3–5 weeks.

Geographic distribution: World-wide

Symptoms: Cysticercosis in cattle is usually inapparent, except if vital structures in organs are affected (e.g. heart, diaphragm). Infections with *Taenia saginata* in man may be associated with loss of appetite and diarrhoea but they are usually inapparent.

Significance: Carcasses containing beef measles are condemned at meat inspection. Consumption of infected, inadequately cooked meat may infect man. Therefore consequent inspection of cattle carcasses needs to be done. This is time-consuming and expensive.

Diagnosis: Demonstration of cysticerci in muscles of cattle at meat inspection. Serological tests may be used to detect infections antemortem but some are relatively unspecific.

Therapy: Not indicated

Prophylaxis: Animals should not be exposed to human faeces. Water used for livestock purposes (e.g. drinking, irrigation) should be free of faecal contamination. The disease is spread because pastures are irrigated with human sewage.

(Figures 154, 155, 156, 157)

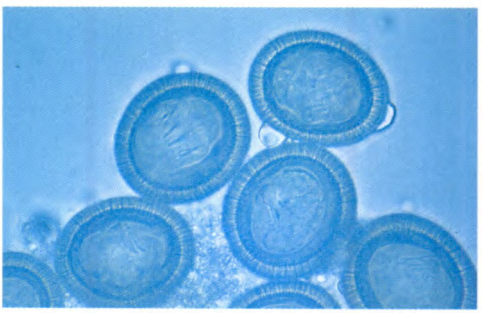

Fig. 154 *Taenia* sp. eggs (34–39 × 31–38 µm) containing oncospheres [4]

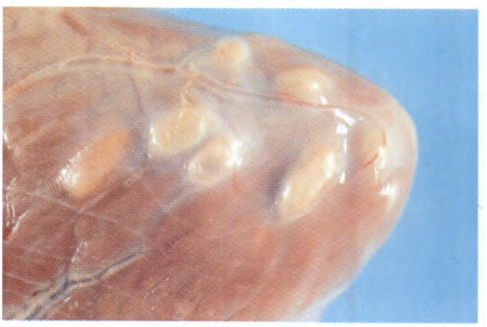

Fig. 155 Cysts of *Cysticercus bovis* (10 × 4–5 mm) in the heart of a cattle [4]

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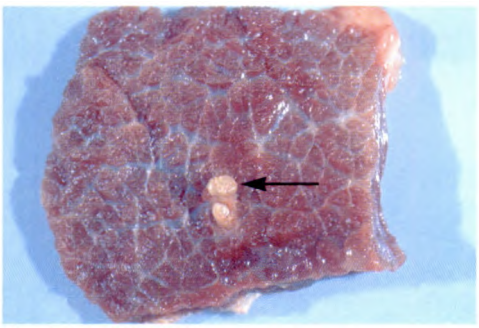

Fig. 156 Calcified tissue cyst of *Cysticercus bovis* in the masseter muscle of a cattle

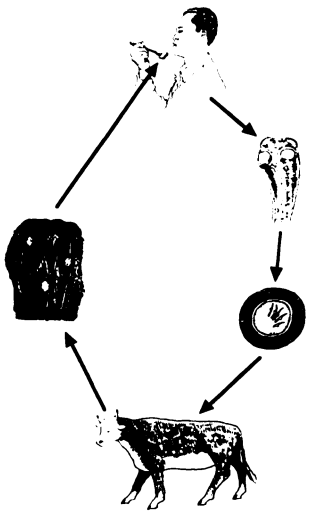

Fig. 157 Life cycle of *Taenia saginata/Cysticercus bovis*

Cysticercus dromedarii (syn. *C. cameli*)

Remarks: The larva (*Cysticercus dromedarii*) of a hyena tapeworm (*Taenia hyaenae*) is frequently found in the muscles of the dromedary, cattle and goats (☞ DROMEDARIES, ■ 4.1.1). It is rarely found in sheep. *C. dromedarii* cysts are twice as large as *C. bovis*, measuring 12–18 mm in length. They are pearly white and the lateral invaginated protoscolex has a double row of hooks. Although they are not dangerous to man, their presence is repugnant, and parasitized meat should be condemned and not fed to dogs or other caniidae.

- **Nematoda larvae and adult nematodes found in the muscles**

Onchocerca dukei Muscular and subcutaneous onchocercosis of cattle

Remarks: Adult worms of this species are found in the subcutaneous, perimuscular and muscular tissue of cattle in tropical Africa (☞ CATTLE, 5.1). It is very frequent in West Africa where sometimes up to 100% of the cattle are infected. *O. dukei* forms intramuscular and subcutaneous nodules. These nodules are fibrous reactions of the host, yellow or grayish-white in colour. They are oval (3–6 mm in diameter) with a raised contour. These nodules are hard to touch, and when sliced one or several worms appear, surrounded by pus. This is the major difference to *Cysticercus bovis*. The intramuscular nodules can be completely embedded in the muscles or only partly covered. The entire musculature may be involved, especially the flank, intercostal and limb muscles. Subcutaneous nodules may be found almost everywhere (☞ CATTLE, 5.1).
(Figures 158, 159)

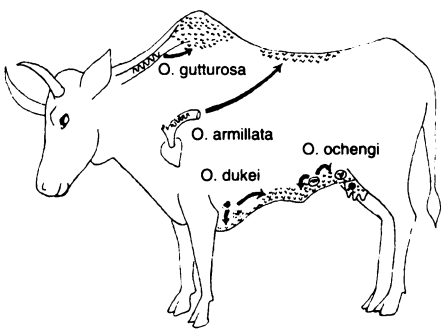

Fig. 158 Sites of occurrence of some *Onchocerca* species found in cattle in Africa [20]

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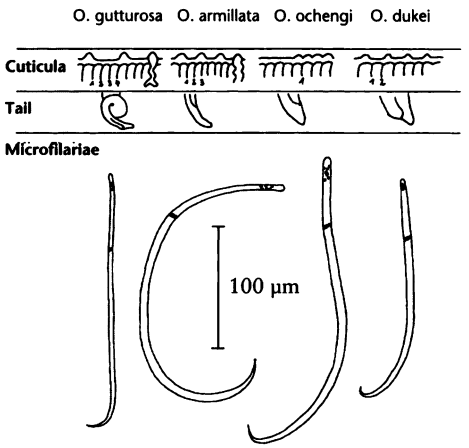

	O. gutturosa	O. armillata	O. ochengi	O. dukei
Lenght	263/249	382/346	298/277	240/217
Diameter	4.4/3.4	6.8/5.5	7.9/5.9	6.7/4.8
n	26/13	15/13	22/13	22/8

Fig. 159 Key for the identification of *Onchocerca* species of cattle in Africa [20]

ARTHROPODS

- Insecta larvae found in the muscles

Hypoderma bovis and *Hypoderma lineatum*
Larvae of the warble flies

Remarks: Larvae of the warble flies may sometimes occur in the muscles during their migratory period. *Hypoderma lineatum* migrates through the wall of the oesophagus (☞ CATTLE, 5.1).

4.1.2 Tendons

HELMINTHS

- Nematoda larvae and adult nematodes found in the tendons

Onchocerca gutturosa
Ligamentary onchocercosis

Location: Adult worms of this species occur in the ligamentum nuchae and also in the ligaments of the hip, elbow, stifle, knee joints and the external side of the scapular cartilage. Microfilariae are found in the skin of the hump and back, sometimes ears and neck of cattle.

Hosts: Cattle

Species description: Males are 2–3 cm long and females are up to 60 cm long or more. The microfialriae are unsheathed and 249–263 µm long and 3.4–4.4 µm wide. The intermediate hosts are members of the family *Simuliidae*.

Geographic distribution: Probably worldwide, extremely common throughout Africa.

Symptoms: Usually inapparent infections. Microfilariae may cause a slightly itching dermatitis, resembling a mild mange.

Significance: Allergic skin reactions against the microfialriae and massive nodule formation may reduce the quality of the skin.

Diagnosis: This is based on the identification of microfilariae in skin biopsies or adult worms at slaughter.

Therapy and Prophylaxis: This is usually not indicated. If allergic dermatitis due to microfilariae are suspected treatment with ivermectin (200 µg/kg, sc.) may be indicated.

Onchocerca lienalis

Remarks: Adults are found in the ligamentum abomaso-lienale. Microfilarieae occur in the skin. The infection is asymptomatic.

4.2 Liver

HELMINTHS

- Trematoda found in the liver

Fasciola gigantica Giant liver fluke

Location: Bile ducts

Hosts: Cattle, sheep, goat, dromedary, horse, pig and many other animal species

Species description: *F. gigantica* is the common liver fluke of livestock in Africa and South

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Asian countries. *F. gigantica* resembles *F. hepatica*. Its size is larger, being 25–75 mm in length and up to 12 mm in breadth. The size of the eggs is 156–197 µm × 90–104 µm. The most frequently involved intermediate hosts are *Lymnaea auricularia* and *L. natalensis*. These are aquatic snails living in fairly large permanent bodies of water. The snail vectors can survive an amphibious existence but can only aestivate for very short periods. Metacercariae (the cyst stage of cercariae formed after the tail is cast off) encyst on plants under water such as rice plants, etc. Metacercariae may survive for up to 4 months on stored plants and thus infection may be transmitted by feeding rice straw. *F. gigantica* adults reach the bile ducts 12 weeks after infection. The immature stages migrate through the liver parenchyma. Infections with *F. gigantica* can occur extremely focal, revealing high prevalences in some regions and low prevalences in others. Infection is acquired by eating herbage which is contaminated with metacercariae. The prepatent period is 9–12 weeks.

Geographic distribution: Throughout Africa and Asia

Symptoms: In sheep an acute and a chronic form can occur whereas in cattle only the chronic form seems to play a role. The pathogenesis is essentially the same as in *F. hepatica*.

Significance: *F. gigantica* causes mainly a chronic fasciolosis in cattle and an acute, often fatal, syndrome in sheep.

Diagnosis: Detection of typical operculated eggs with the sedimentation method. *Cave: F. gigantica* eggs are similar to the eggs of some rumen fluke species! *F. gigantica* eggs only appear 15 weeks after infection! Adult flukes are found in the bile ducts at necropsy.

Therapy: ☞ *F. hepatica*

Prophylaxis: ☞ *F. hepatica* in many parts of Africa it is not possible to avoid grazing around lakes, swamps especially if these places are used for watering. The use of bore holes for watering cattle is an effective means to reduce infections with *F. gigantica*.

(Figure 160)

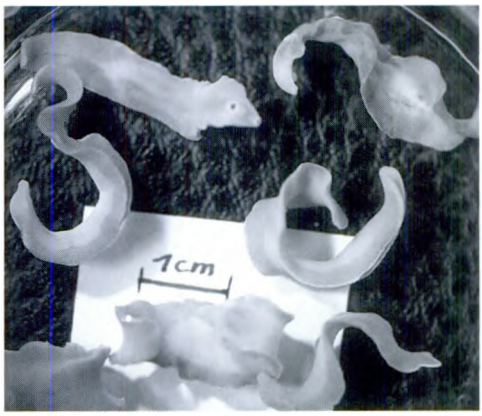

Fig. 160 *Fasciola gigantica*; the giant liver fluke

Fasciola hepatica Common liver fluke

Location: Bile ducts of the liver, sometimes gall bladder

Hosts: Cattle, sheep, goat, horse, donkey, pig and many other animal species

Species description: *F. hepatica* is occurs world-wide – except in Africa and South Asia where it is replaced by *F. gigantica* – and affects especially cattle and sheep. Its size is 30 by 15 mm. The eggs measure 130–150 × 65–90 µm. *F. hepatica* eggs hatch within 10–12 days after deposition and the miracidium, the first larval stage, penetrates actively into the intermediate host which is generally an amphibious snail of the genus *Lymnaea*. Cercariae leave the snail 4–7 weeks after infection and settle on plants just below water level. After casting off the tail they form a cyst about 0.2 mm in diameter (metacercariae). The metacercariae are infective. Ingested metacercariae penetrate the small intestinal wall and reach the liver via abdominal cavity. The duration of the migration within the liver parenchyma is 6–8 weeks after which the young flukes reach maturity in the bile ducts. Infection occurs orally by ingestion of metacercariae on herbage. The incubation period is 2–4 weeks. First symptoms may be seen 5–7 days after infection in acute fasciolosis.

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Geographic distribution: Temperate areas world-wide and high altitude regions in East and South Africa.

Symptoms: *F. hepatica* causes a wide range of clinical symptoms, depending on the number of metacercariae ingested but none of the symptoms is pathognomonic. Chronic fasciolosis is the most common form in cattle, sheep and other hosts. The symptoms of chronic fasciolosis are generally associated with hepatic fibrosis and hyperplastic cholangitis. Anaemia, oedema (bottle jaw), digestive disturbances (constipation, diarrhoea) and cachexia develop gradually.

Acute fasciolosis is less common than the chronic disease and occurs mainly in sheep. It is basically a hepatitis caused by the simultaneous migration of large numbers of immature flukes. Sudden death may occur in acute fasciolosis.

Significance: *F. hepatica* is the most important trematode of domestic ruminants and the most common cause of liver fluke disease in temperate areas.

Diagnosis: The oval, operculated, golden-brown eggs (130–150 × 65–90 µm) appear in the faeces 10 weeks after infection. The sedimentation is the most reliable method for the detection of *Fasciola* spp. eggs (☞ METHODS, 1.5). Eggs are expelled intermittently, depending on the evacuation of the gall bladder. *Fasciola* spp. eggs are very similar to eggs of the rumen flukes (family Paramphistomidae). Rumen flukes are very prevalent in ruminants in most parts of Africa whereas fasciolosis is rather focal. The differentiation between *Fasciola* spp. eggs and eggs of Paramphistomidae is not easy. The detection of the adult flukes in the liver at necropsy or slaughter is the most reliable method to confirm fasciolosis. Prevalence studies should be based on abattoir surveys rather than on coproscopic investigations.

Therapy: A number of compounds is available to treat fasciolosis in cattle. These are oxyclozanide (10 mg/kg, po.), rafoxanide (7.5 mg/kg, po.), nitroxynil (10 mg/kg, sc.), albendazole (10 mg/kg, po.), closantel (5 mg/kg, po.), triclabendazole (12 mg/kg, po.), netobimin (20 mg/kg, po.) and clorsulon (7 mg/kg, po.). Some of these diamphenethide (100 mg/kg, po.), nitroxynil (15 mg/kg, sc.), closantel (10 mg/kg, po.), triclabendazole (12 mg/kg, po.) and clorsulon (7 mg/kg, po.) are active against immature flukes. The treatment interval for strategic control should be chosen according to the local epidemiological situation.

Prophylaxis: The snail intermediate host may be controlled by drainage of land, fencing off water pools to exclude animals from the snail habitats. The use of plant molluscicides offers new prospects for future snail control.

(Figures 30, 31, 32, 161, 162, 163, 164, 165)

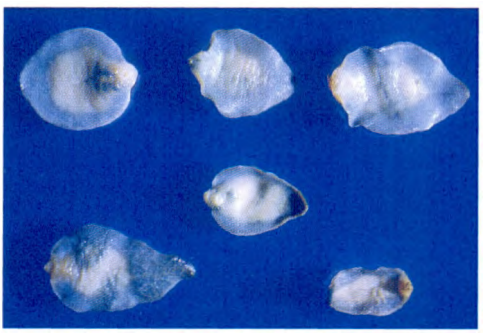

Fig. 161 *Fasciola hepatica*; the common liver fluke (30 × 15 mm)

Fig. 162 *Lymnaea truncatula*; the intermediate host of *Fasciola hepatica* [10]

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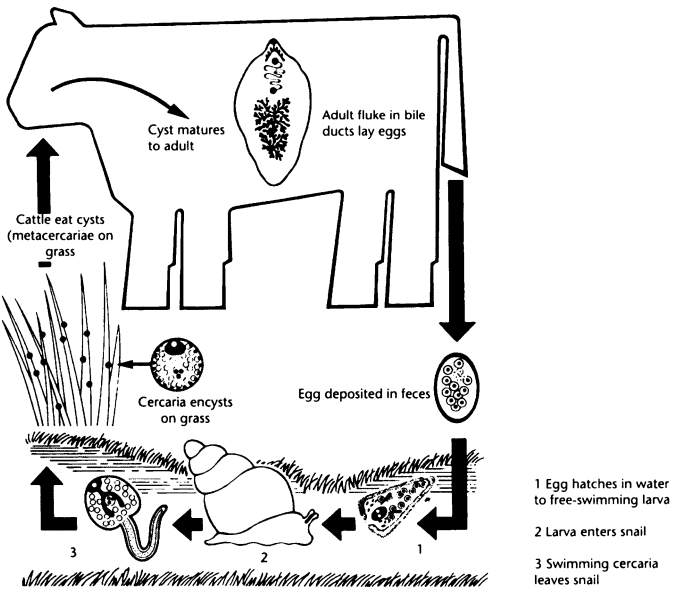
Fig. 163 Life cycle of *Fasciola hepatica* [6]



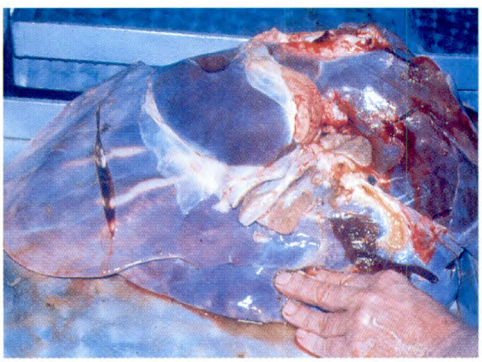


Fig. 164 *Fasciola hepatica* infection; cholangitis and calcification of the bile ducts [4]

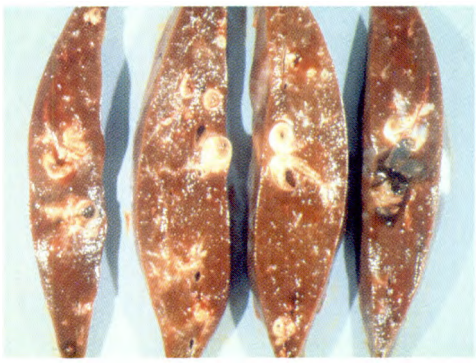


Fig. 165 *Fasciola hepatica* infection causing cholangitis and calcification of the bile ducts

Dicrocoelium hospes African lancet fluke

Location: Bile ducts and gall bladder
Hosts: Sheep, goat, cattle and other ruminants
Species description: *D. hospes* is 6–10 mm long and 0.8–1 mm wide. The eggs measure 36–45 µm × 25 µm. The life cycle involves two intermediate hosts. The first is a land snail (*Limicolaria* spp., *Achatina* spp. and probably others) and the second an ant (*Dorylus* spp., *Cematogaster* spp. and other genera). *D. hospes* is closely related to *D. dendriticum*. Infection is acquired by ingestion of infected ants (containing metacercariae) via herbage. Migration of immature flukes from the intestine via ductus choledochus into the biliary system. The prepatent period is up to 97 days.
Geographic distribution: North, Central and West Africa
Symptoms: Generally clinically inapparent; heavy infections cause diarrhoea, cholangitis and weight loss.
Significance: In some regions a high percent-

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age of animals is infected. Losses occur due to poor growth and condemnation of infected livers at slaughter.

Diagnosis: Adult *D. hospes* in the bile at necropsy; detection of parasite eggs in the sediment of faeces.

Therapy: ☞ below *D. dendriticum*

Prophylaxis: ☞ below *D. dendriticum*

Dicrocoelium dendriticum (syn. *D. lanceolatum)* Common lancet fluke

Location: Bile ducts or gall bladder

Hosts: *D. dendriticum* occurs mainly in sheep, goat, cattle but also in pig, dog, donkey, rabbit and rarely in man.

Species description: The fluke is 6–10 mm long and 1.5–2.5 mm wide. The eggs measure 36–45 × 20–30 µm. The life cycle involves two intermediate hosts, a land snail as the first (*Zebrina* spp., *Cionella* spp. and 29 other species are described) and ants of the genus *Formica* and *Lasius* as the second intermediate hosts. Ants infected with metacercariae attach to herbage overnight and are available to grazing animals in the early morning. Infection occurs by ingestion of infected ants (containing metacercariae) via herbage. Migration of immature flukes from the intestine via ductus choledochus into the biliary system. The prepatent period is 47–54 days. Cirrhosis of the liver and thickening of the bile ducts may occur in heavy infections.

Geographic distribution: Europe, Asia, the Americas, North Africa, occasionally in imported ruminants in other parts of Africa; autochthonous infections seem not to occur in Africa.

Symptoms: Often without clinical symptoms. Heavy infections may produce weight loss, anaemia and hypoproteinaemia.

Significance: Lancet flukes may cause extensive liver damage, causing the host to "do poorly". Condemnation of livers at slaughter may cause severe economic losses among cattle herds.

Diagnosis: Demonstration of typical eggs in the sediment of faeces. The eggs are small (40 × 25 µm), thick walled, yellowish-brown and contain a miracidium. Adult parasites may be found in the bile ducts at necropsy.

Therapy: Albendazole (20 mg/kg, po.), fenbendazole (50 mg/kg, po.), netobimin (20 mg /kg, po.) thiophanate (50 mg/kg, po.), thiabendazole (200 mg/kg, po.) and praziquantel (50 mg/kg, po.) are effective against the lancet fluke.

Prophylaxis: Elimination of the intermediate hosts is not feasible.

(Figures 32, 33, 166, 353)

Fig. 166 *Dicrocoelium dendriticum* found in the bile ducts

***Schistosoma* spp.**

Remarks: Schistosomes may often be found at necropsy in the portal veins of the liver (☞ CATTLE, ■ 2).

- **Cestoda found in the liver**

Echinococcus granulosus (syn. *E. unilocularis*) Hydatid tapeworm, hydatidosis

Location: Larvae (hydatid cysts) of *E. granulosus* are found in the liver and the lungs of cattle, sheep, goats, swine, horse, dromedary and other domestic animals and man (= intermediate hosts). Adult tapeworms are found in the small intestine of the dog and other related carnivores (= final hosts).

Hosts: Canids are the final hosts of *E. granulosus*. Intermediate hosts are ungulates and man.

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Species description: *E. granulosus* is a small tapeworm with 3–4 proglottids and a total length of 2–6 mm. It occurs in the small intestine of the dog and other canids (hyaena, coyote, fox, etc.). Adult tapeworms in the dogs pass proglottids or eggs in the faces. When ingested by cattle or other intermediate hosts, these eggs hatch in the intestine. The oncospheres (hexacanths) penetrate the gut wall, enter the portal vein and reach the liver, which is the first capillary filter for the larvae and subsequently the lungs, which are the second capillary filter. The remaining oncospheres may reach via the arteries other organs. In all these organs hydatid cysts (bladder worms) develop. These are large cysts filled with fluid and tapeworm heads (protoscolices). The hydatid cysts develop slowly over several months. They are commonly 5–10 cm in diameter and contain a liquid. The hydatid cyst is composed of a thick outer, concentrically laminated membrane. About 5 months after infection brood capsules, each containing several protoscolices develop from the inner part of the capsule (germinal membrane). The brood capsules may become detached and float free in the cyst fluid, being termed "hydatid sand". At this time the cyst is infective for the definitive host. The life cycle is completed when a dog ingests protoscolices. Hydatid cysts are usually found in the lungs of sheep and cattle, but primarily in the livers of horses. In pigs, hydatid cysts are found in both the liver and the lungs. The hydatid cysts may be multilocular (sheep, cattle, pigs) or unilocular (horse). In man hydatid cysts may be found in many organs. The significance of the hydatid cyst depends on the severity of the infection and the organ in which it is situated. In domestic animals clinical signs are not commonly seen despite heavy infections. Human hydatidosis is often associated with clinical signs and the function of the affected organ is often impaired. This is especially true if the heart or the brain are involved. Adult tapeworms are harmless to the dogs and seldom cause clinical symptoms.

Geographic distribution: World-wide

Symptoms: Hydatid cysts do not usually cause clinical symptoms unless the cysts are numerous or become very large.

Significance: Hydatidosis in cattle is not usually a problem, but infected organs are condemned at slaughter. In man hydatidosis is a serious problem. Infected dogs are a reservoir for both infection of domestic animals and man.

Diagnosis: Usually the diagnosis of hydatidosis is made at slaughter or necropsy. Infected dogs pass eggs in the faeces which cannot be differentiated from those of *Taenia* spp. Immundiagnostic tests (ELISA, Immunoblotting; ☞ METHODS, 5.2 and 5.3) are widely used in medical parasitology and may assume significance in epidemiological studies. *E. granulosus* eggs, excreted by dogs are very similar to those of *Taenia* spp. The differentiation may be carried out by means of monoclonal antibodies.

Therapy: Treatment of hydatidosis in cattle and other intermediate hosts is rarely indicated. Control of hydatidosis is based on prophylaxis.

Prophylaxis: Dogs should not be fed with uncooked meat in general and infected organs in particular. In endemic areas where human hydatidosis is of great importance special attention should be paid to the safe destruction of affected organs, the reduction of stray dogs and the anthelmintic treatment of domestic dogs. Praziquantel (5 mg/kg, po. or 5.7 mg/kg, sc. or im.), bunamidine hydrochloride (25–50 mg/kg, po.) and the combination of febantel/praziquantel/pyrantel are sufficiently effective against adult *E. granulosus* tapeworms in dogs.

(Figures 167, 168, 169, 170)

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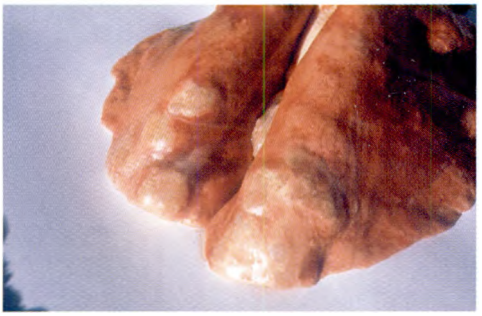

Fig. 167 *Echinococcus granulosus*; hydatid cyst in the lungs of a cattle [4]

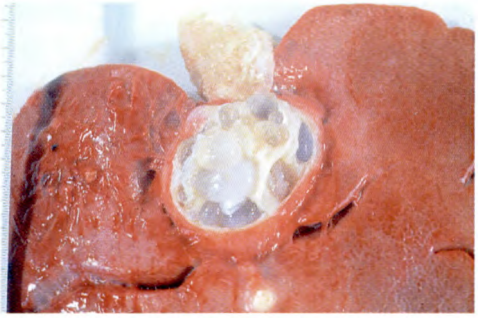

Fig. 169 *Echinococcus granulosus*; hydatid cyst [42] opened [4]

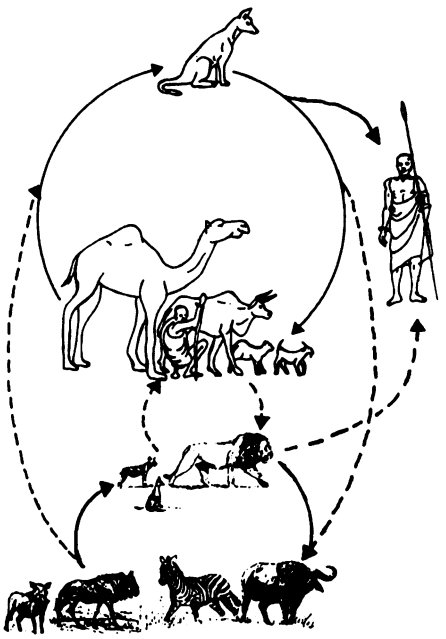

Fig. 168 Life cycle of *Echinococcus granulosus* in sub-Saharan Africa [21]

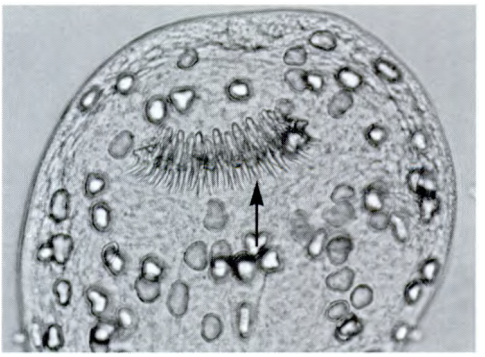

Fig. 170 *Echinococcus granulosus*; protoscolex (100 × 160 in diameter) with typical hook collar

Stilesia hepatica

Remarks: This parasite occurs in the bile ducts and small intestine of sheep and goats (☞ SHEEP AND GOATS, ■ 4.2). In endemic areas wild ruminants and rarely cattle may also be affected. This parasite is very prevalent in tropical and southern Africa. *S. hepatica* is non-pathogenic but extremely prevalent (in 90–100% of sheep) in many parts of Africa. It is non-pathogenic and does not require treatment.

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Cysticercus tenuicollis

Remarks: Migrating post-oncospheral stages may occasionally be found in the liver parenchyma and thin-necked cysts may be found on the serosa of the liver (☞ CATTLE, ■ 4.4 and SHEEP AND GOATS, ■ 4.4).

POROCEPHALIDA

Linguatula serrata "tongue worm"

Remarks: This is a cosmopolitan parasite and it occurs in the nasal and respiratory passages of the dog and other canids, snakes, very rarely in man, horse, goat and sheep. The parasite is tongue-shaped and the cuticle is transversely striated. Males are 1.8–2 cm, females are 3–13 cm long. The eggs measure about 90–70 µm. The eggs are ingested by a herbivorous intermediate host (horse, sheep, goat, cattle, rodents, rabbit, etc.) and hatch in the alimentary canal and the larvae migrate to the mesenteric lymph nodes, in which they develop to the infective nymphal stage. The larva is up to 500 µm long and undergoes about six to 9 moults. It usually lies in a cyst surrounded by a viscid fluid. These cysts may be found at necropsy in the intestinal lymph glands, the liver or in other organs. Dogs become infective by eating the infective viscera, especially of sheep and cattle. Infected dogs show sneezing and discharge from the nostrils. The infection in cattle is clinically inapparent.
(Figures 171, 172)

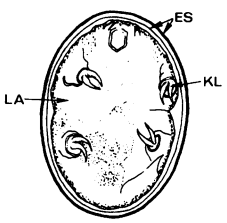

Fig. 171 *Linguatula serrata*; embryonated egg (90 × 70 µm); [47]; ES = egg shell; KL = claw; LA = larva [7]

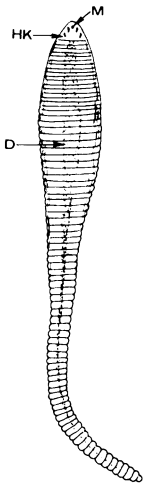

Fig. 172 *Linguatula serrata*; adult female (up to 13 cm long); M = mouth opening; HK = hooks; D = intestine [7]

4.3 Respiratory system

HELMINTHS

- Cestoda found in the lungs and trachea

Echinococcus granulosus (syn. *E. unilocularis)* Hydatid tapeworm, hydatidosis

Remarks: Larvae (hydatid cysts) of *E. granulosus* are often found in the lungs and liver of cattle and other domestic animals (☞ CATTLE, ■ 4.2).

- Nematoda found in the lungs and trachea

Dictyocaulus viviparus Cattle lungworm, parasitic bronchitis

Location: Trachea, bronchi and bronchioles
Hosts: Cattle, camel and some deer species
Species description: Males are 4–5 cm long and the females 6–8 cm. The eggs measure 82–88 × 33–38 µm. The adult worms live

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in the airways and produce eggs which contain larvae. These eggs are moved with respiratory secretions towards the larynx. They are then coughed up and swallowed. Hatching occurs in the intestine and first-stage larvae are passed in the faeces. On the pasture the larvae moult twice to reach the third stage which is infective for cattle. When swallowed the infective larvae penetrate the gut wall and move to the local lymph nodes, where they moult to become fourth-stage larvae. Then they migrate via the thoracic duct to the jugular vein, after that to the right side of the heart and then to the lungs where they are arrested in the capillaries and break through to the air passages. Development to maturity in the bronchi of the host takes about 4 weeks. Larval stages remain inhibited for several months. Irritation of the respiratory mucosa by larvae and adult worms cause increased respiratory secretions and lung congestion with blockage of air passages. Aspiration of eggs and larvae into the alveoli leads to consolidation of lobules. Severe epithelialization and peribronchial fibrosis usually occur a few weeks after the infection has started. These chronic lung alterations may be the cause of the unthriftiness, commonly observed in animals after a heavy infection. Immunity is built up as quickly as 10 days after an initial infection but it wanes in the absence of reinfection.

Geographic distribution: World-wide

Symptoms: Parasitic bronchitis is characterized by severe coughing, rapid breathing, dyspnoea and rapid loss of condition. Severe cases lead to emphysema and pneumonia. Death may occur in heavy infections.

Significance: Parasitic bronchitis due to *D. viviparus* may cause great losses in endemic areas. Even animals after a moderate initial infection show retarded growth for prolonged periods of time. In Africa *D. viviparus* has been frequently found in imported animals. Autochthonous infections occur sporadically.

Diagnosis: This is based on the clinical signs, rapid and heavy breathing, coughing, nasal discharges and the demonstration of larvae in the faeces by the Baermann method (☞ METHODS, 1.7 and CATTLE, 1). However, coughing may occur in the prepatent period before larvae are found in the faeces. Usually parasitic bronchitis is a herd problem, seen especially in young calves which have recently been exposed to an infected pasture.

Therapy: Levamisole (5 mg/kg, sc. or 7.5 mg/kg, po.), the benzimidazoles (fenbendazole, 7.5 mg/kg, po.; oxfendazole, 4.5–5 mg/kg, po.; albendazole, 7.5 mg/kg, po.; febantel, 7.5 mg/kg, po.) netobimin (12.5 mg/kg, po.) and ivermectin (200 µg/kg, sc.) are highly effective against all stages of *D. viviparus*. These drugs are also effective against lungworms in sheep, horses and pigs. In calves, aggravated coughing a few minutes after treatment (especially with levamisole, sc.) is characteristic for the infection and often regarded as a confirmation of the diagnosis. Heavily affected calves should be moved inside and supportive treatment should be administered.

Prophylaxis: Pastures with a recent history of lungworm infection are highly dangerous for young calves. Lungworm infections may be controlled in several ways. Calves may be vaccinated with X-radiated *Dictyocaulus* larvae (Dictol®), in countries where the vaccine is available, twice (4 weeks apart) prior to exposure. The animals should be housed during the vaccination period and for another 2 weeks after the second dose to allow time to build up an adequate level of immunity before release on pasture. Another approach is the strategic control of infections. In first-season cattle, a first treatment is applied 2–3 weeks after turn-out and, depending on the infection risk and the duration of the drug, repeated treatments are required during the period of high challenge (benzimidazoles monthly, ivermectin 3 and 10 weeks after turn-out). A sustained release device (anthelmintic with a prolonged activity)

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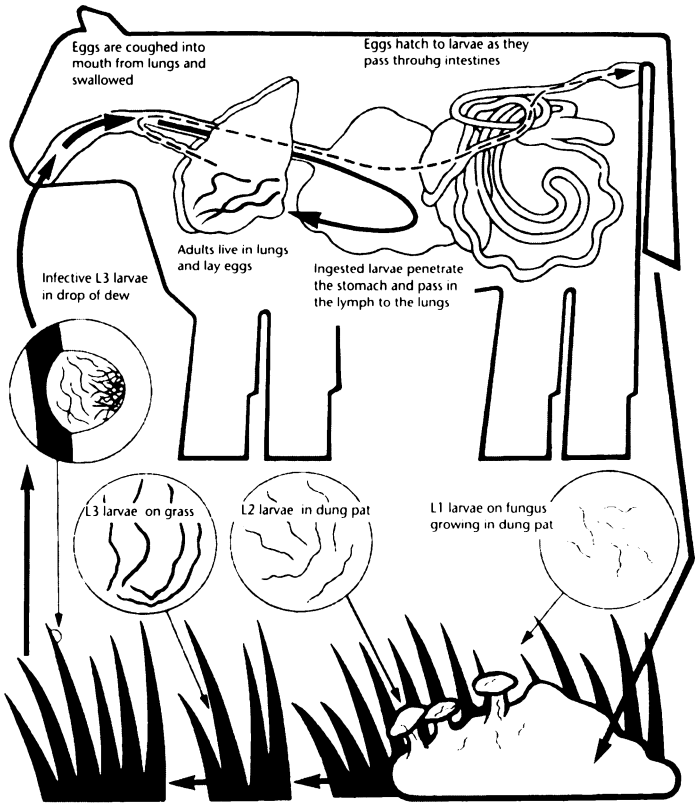

Fig. 173 Life cycle of *Dictyocaulus viviparus*; the third-stage larvae penetrate the intestinal mucosa and not the mucosa of the stomach [6]

may also be applied about 2–3 weeks after turn-out or prior to the period with the highest infection risk.
(Figures 83, 173, 174)

Fig. 174 *Dictyocaulus viviparus*; adult worms in trachea

Mammomonogamus laryngeus

Location: Larynx
Hosts: Cattle, water buffalo, occasionally sheep, goat and rarely man
Species description: *M. laryngeus* is not very pathogenic for cattle. The worms are attached to the mucosa of the larynx. The males and females are found in permanent copulation. The life cycle is unknown.
Geographic distribution: Caribbean Islands, South America, Asia, Central Africa (Cameroon)
Symptoms: Affected animals may cough and sometimes lose condition. Calves may develop bronchitis. Pneumonia due to respiratory secretions which are aspirated, causing pulmonary reactions have been observed.

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Significance: Heavy infections in endemic areas may cause losses due to massive irritation of the larynx by the parasites. Bronchitis and pneumonia may occur.

Diagnosis: This is made by finding the eggs in the faeces or adult worms at necropsy.

Therapy: Unknown. Nitroxynil (10mg/kg, sc.), which is used against *Syngamus* spp. of poultry may also be used against *Mammomonogamus laryngeus*. Modern benzimidazoles and ivermectin (200 µg/kg. sc.) may be used in heavily infected cattle.

Prophylaxis: Unknown

(Figures 175, 176, 177)

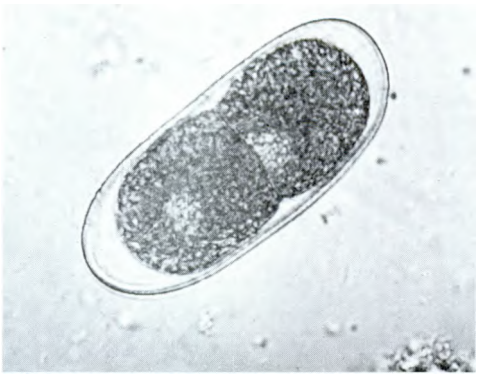

Fig. 176 Egg of *Mammomonogamus laryngeus* (323 × 50 µm) [8]

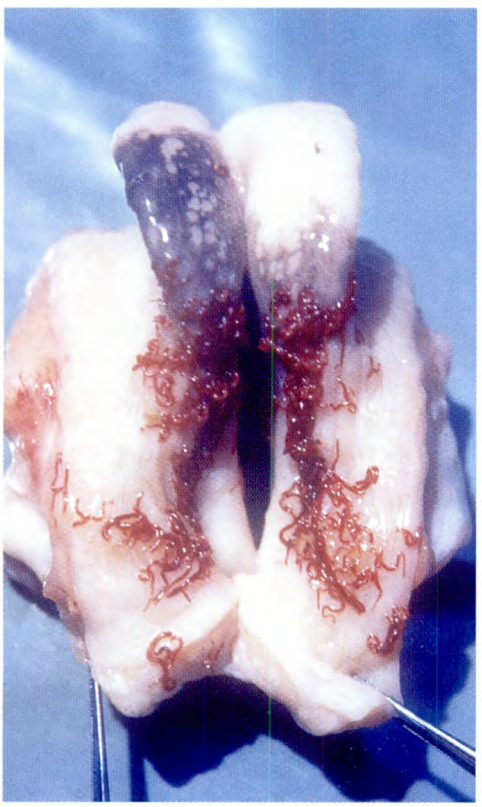

Fig. 175 *Mammomonogamus laryngeus* (males are 3–3.5 mm long, females are 8.5–10 mm long) [8]

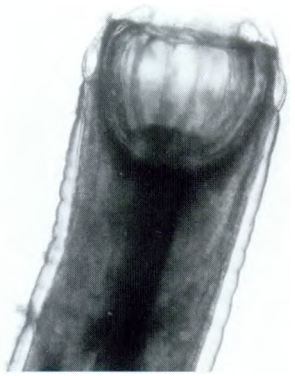

Fig. 177 *Mammonogamus laryngeus*; head end [8]

Mammomonogamus nasicola

Location: Nasal cavities

Hosts: Cattle, sheep and goat

Species description: The males and females of this parasite are permanently joined in copulation. Males are 4–6 mm long and females 11–23 mm. Eggs are 54 × 98 µm and have only a few blastomeres. Life cycle and pathogenesis are unknown. An optional reservoir host (e.g. earthworm or snail) is being suspected. After oral ingestion of infective larvae the parasite reaches the respiratory tract through the blood stream. The parasites suck blood and are bright red in fresh samples.

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Geographic distribution: Central and South America, West Indies, Central Africa.
Symptoms: Heavy infections cause irritation of the nasal mucosa, sneezing and nasal discharges. *M. nasicola* may be differentiated from *Schistosoma* infection of nasal passages.
Significance: ☞ above *M. laryngeus*
Diagnosis: ☞ above *M. laryngeus*
Therapy: Unknown
Prophylaxis: Unknown
(Figure 178)

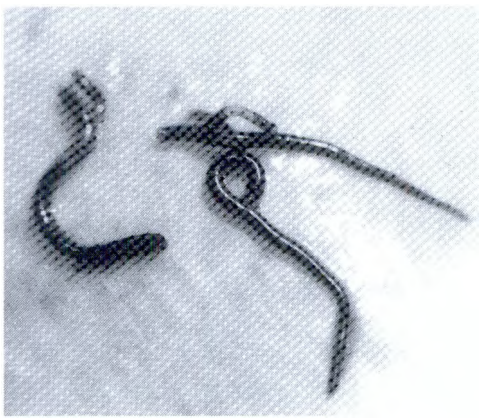

Fig. 178 *Mammomonogamus nasicola* [18]

4.4 Abdominal cavity

HELMINTHS

- Cestoda found in the abdominal cavity

Cysticercus tenuicollis Larval stage of the canine tapeworm *Taenia hydatigena*

Remarks: The thin-necked larvae (*Cysticercus tenuicollis*) of *Taenia hydatigena* are often found attached to the omentum, the intestinal mesentery and to the serosal surface of abdominal organs, especially the liver of cattle, sheep and other herbivores. Cysticerci are often found at meat inspection without any previous clinical signs. The feeding of condemned material to dogs should be avoided (☞ SHEEP AND GOATS, ■ 4.4).
(Figures 179, 180)

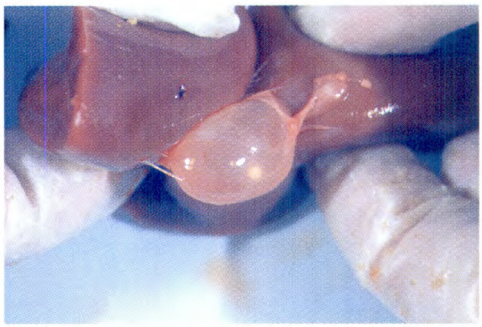

Fig. 179 *Cysticercus tenuicollis*; thin-necked cyst attached to the liver of a sheep. The protoscolex can be seen through the cyst wall

Fig. 180 *Cysticercus tenuicollis*; thin-necked stage found in cysts

- Nematoda found in the abdominal cavity

Setaria labiatopapillosa Bovine abdominal filariosis

Location: Peritoneal cavity
Hosts: Cattle, deer, giraffe and other ruminants
Species description: Males are up to 4–6 cm long and females 6–12 cm. There is a distinct peribuccal ring with dorsal and ven-

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tral prominences. The tail of the female terminates in a marked button which is divided into a number of papillae. The microfilariae are sheathed, measure 240–260 µm and appear in the peripheral blood (☞ CATTLE, ■ 2). Several species of mosquitoes act as vectors (*Aedes* spp., *Culex* spp. and *Anopheles gambiae* and other *Anopheles* spp.). Infective larvae are produced in the vector in 12–16 days. Adult worms in the peritoneal cavity are non-pathogenic. The major pathogenic effect occurs when immature stages migrate erratically in the central nervous system of abnormal hosts (e.g. *Setaria digitata* or *S. labiatopapillosa* microfilariae introduced by vectors in horses, sheep and goats). The affected animals suffer from an acute, focal encephalomyelomalacia. The lesions are usually single tracks left by migrating larvae and may be found in any part of the CNS.

Geographic distribution: World-wide

Symptoms: Adult worms in the peritoneal cavity do not cause clinical signs. Erratic larvae in the CNS may cause nervous signs, depending on the location of the lesions. Muscular weakness, ataxia, paralysis and death may occur.

Significance: Adult worms in the peritoneal cavity are non-pathogenic. Erratic larvae in the CNS of an abnormal host may cause nervous symptoms and even death.

Diagnosis: This is made by demonstrating the microfilariae in stained blood smears or with the dark ground/buffy coat method (☞ METHODS, 3.1 and 3.4) or by finding the adult worms in the abdominal cavity. The antemortem diagnosis of immature worm-associated CNS disturbances under field conditions is impossible. Histopathological examination of stained brain sections may result in an aetiological diagnosis.

Therapy: Chemotherapy is difficult because adult worms are very resistant to most drugs. Ivermectin (200–500 µg/kg, sc.) and diethylcarbamazine (25–100 mg/kg/day during several days, sc.) are effective against the microfilariae.

Prophylaxis: Unknown
(Figures 148, 181, 182)

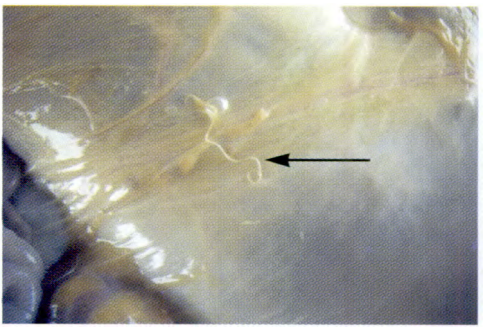

Fig. 181 *Setaria labiatopapillosa*; adult worm on the intestinal serosa; males are 4–6 cm long and and females are 6–12 cm long

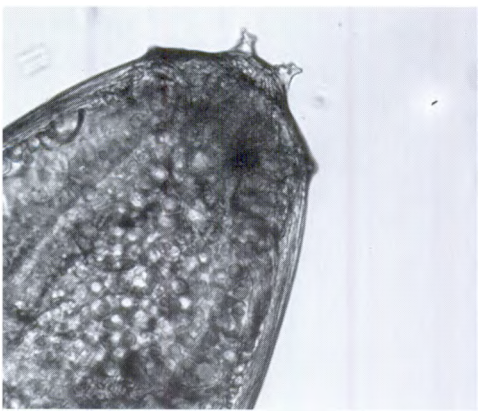

Fig. 182 *Setaria labiatopapillosa*; anterior end

4.5 Pancreas

HELMINTHS

- Trematoda found in the pancreas

Eurytrema pancreaticum Pancreatic fluke

Remarks: This parasite occurs in the pancreatic ducts, bile ducts, occasionally duodenum of sheep, goats (☞ SHEEP AND GOATS, ■ 4.5) and occasionally cattle in Asia, Brazil,

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Madagascar and Réunion. There are no obvious clinical signs unless heavy infections are present. However, recent reports suggest that in areas with a high prevalence and intensity of infection, *Eurytrema pancreaticum* may be responsible for chronic wasting and mortality, especially in sheep.
(Figures 34, 183)

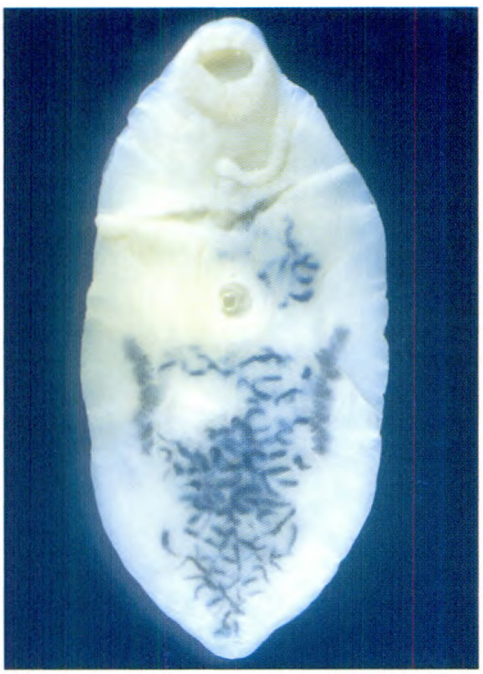

Fig. 183 *Eurytrema pancreaticum*; (8–16 mm long and 5–8. 5 mm wide) [8]

4.6 Central nervous system

PROTOZOA

Toxoplasma gondii

Remarks: Tissue cysts of this parasite may occasionally be found in the brain of sheep and cattle. These cysts contain merozoites (6–8 µm long) and are infective for the final host (cat) (☞ also SWINE, ■ 4.1 and 4.6).

Neospora sp.

Remarks: *Neospora caninum* is a recently recognized new parasite species. It resembles *T. gondii* and causes neuromuscular disorders (polyradiculoneuritis, myositis, paralysis) in dogs and abortion in cattle. *Neospora* sp. is suspected to be responsible for up to 20% of cattle abortion in some parts of the USA. The entire life cycle is unknown. No final host has been found so far in which the sexual development occurs. The only stages of this parasite are tachyzoites and tissue cysts. These are thick-walled (1–4 µm) as compared to *T. gondii* which produces thin-walled (< 0.5 µm) cysts. *Neospora*-like organisms were found in the central nervous system of dogs, cats, lambs, calves and foals. Transplacental transmission has been demonstrated in all these cases. The diagnosis is based on the demonstration of parasites in stained tissue sections or isolation from freshly aborted foetuses. Positive IFAT titres (☞ METHODS, 5.1) in neonatal calves prior to colostrum application is a strong indication of a prenatal infection. The main location of tissue cysts in cattle is the central nervous system. Sulfonamids combined with trimethoprime, pyrimethamine (1mg/kg, daily po. for 4 weeks) and clindamyzine (13.5 mg/kg, 3 × daily po. during 10 days) are used in dogs for treatment. No specific treatment is yet available for ruminants.
(Figures 184, 185)

Fig. 184 A weak-born calf with neuromuscular disorders due to a congenital *Neospora caninum* infection [22]

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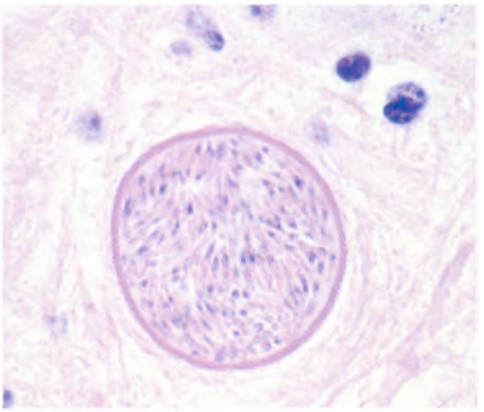

Fig. 185 *Neospora caninum*; thick walled cyst (1–4 µm thick; 70–120 µm in diameter) in the brain of a calf [22]

HELMINTHS

- **Cestoda cysts found in the central nervous system**

Coenurus cerebralis Larval stage of the canine tapeworm *Taenia multiceps* (syn. *Multiceps multiceps*) "gid", "stuggers", "sturdy"

Remarks: The intermediate stage of the canine tapeworm *Taenia multiceps*, a coenurus, develops in the brain and spinal cord mainly of sheep and goats and less commonly in cattle (☞ SHEEP AND GOATS, ■ 4.6).

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5 Stages on the body surface

5.1. Skin and coat

5.2 Eyes

5.1. Skin and coat

PROTOZOA

Besnoitia besnoiti (syn. *Sarcocystis besnoiti*)
Elephant skin disease

Location: *Besnoitia* cysts are found in the dermis, subcutaneous tissue, fasciae, laryngeal, nasal and other mucosae.

Hosts: Domestic and wild cats act as definitive hosts. Intermediate hosts are cattle, goat, wildebeest, impala and kudu. Sheep, rabbit and some rodents may be experimental intermediate hosts.

Species description: This is a protozoan disease of the skin, subcutis, blood vessels and mucous membranes. These *Toxoplasma*-like organisms multiply in endothelia, histiocytic and other cells and produce characteristic large, thick-walled cysts filled with bradyzoites. The *Besnoitia* cysts may reach up to 600 µm in diameter. They are usually spherical and packed with crescentic trophozoites (bradyzoites) each 2–7 µm in length. Parenteral application of blood, taken from an infected animal may transmit the disease to other animals. Unsporulated oocysts (15 × 13 µm) are shed in the faeces of the final host (Felidae). The mode of transmission is not completely known. In particular the transmission from infected cattle to cats remains to be substantiated. Biting flies (e.g. *Glosssina palpalis* and other biting flies) and ticks may transmit *B. besnoiti* mechanically to cats. Needle inoculation of tissues that contain cysts can transmit some *Besnoitia* spp. to other hosts. It has also been suggested that contaminated water troughs may be an important source of infection. The mortality is usually below 10% although animals may lose condition. Pregnant animals may abort. Bulls may become sterile and hides are of little value for leather-making purposes. Animals of all ages, from 6 months upwards may be infected.

Geographic distribution: Southern Europe, Africa, Asia, South America

Symptoms: The initial stage of the disease starts with fever after an incubation period of 6–10 days. Cyst formation in the skin starts 1–4 weeks after the start of the temperature rise. Inappetence, photophobia, enlargement of the lymph nodes and oedematous swellings on the limbs and the lower parts of the body, orchitis and rhinitis may accompany the febrile stage. Animals are reluctant to move, respiration is rapid and diarrhoea and abortion may occur during this phase. In mild infections recovery may occur in this stage. In the second stage of the disease, the skin becomes hard, thick and wrinkled and develops cracks that allow secondary bacterial infections and myiasis to develop. Movement is painful. A serosanguineous fluid oozes from the cracks. The skin is scurfy and folded and the whole appearance is one of extensive mange. There is hair loss over the swollen skin. Severe infections cause emaciation and death may occur. Cysts may appear in the scleral conjunctiva and nasal mucosa. Although mortality is low, conva-

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lescence is slow in severe cases. Severely affected bulls may remain permanently sterile. Previously infected animals may be carriers for life. The hairless condition may remain for several months.

Significance: Besnoitiosis may be a severe problem in endemic areas. Although the mortality is low, affected animals may be emaciated and sick for months.

Diagnosis: This is based on the clinical signs with the typical skin lesions following a febrile phase. Microscopical examination of affected skin by means of stained biopsies may reveal the typical cysts, containing the bradyzoites. Spherical, encapsulated pseudocysts are pathognomonic. The best method of diagnosis is the examination of the scleral conjunctiva. The cysts can be seen in the scleral conjunctiva with the naked eye, revealing many chronic cases without signs.

Therapy: There is no satisfactory treatment. Affected animals should be separated and treated symptomatically (antibiotics, insect-repellants, etc.). Sulfonilamide application prevented cyst development in experimental studies. Antimony (1%, 0.6 ml/kg, po.) cured besnoitiosis in a goat.

Prophylaxis: This is difficult until the exact mode of transmission is known. In some countries, a tissue-cultured vaccine is available and quite effective. Separation of cattle from cats, and of domestic cattle from wild ruminants, and the elimination or isolation of infected animals should help to prevent transmission of this parasite.

(Figures 186, 187, 188)

Fig. 186 Thickened skin ("elephant hide disease") following a *Besnoitia besnoiti* infection [4]

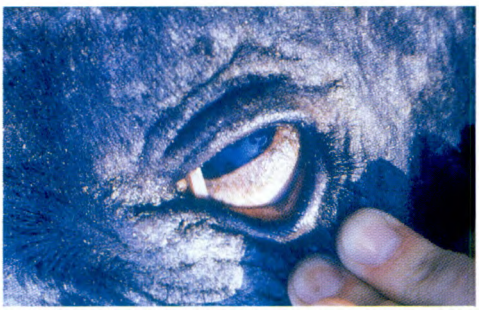

Fig. 187 *Besnoitia besnoiti*; cysts can be seen in the scleral conjunctiva with the naked eye [4]

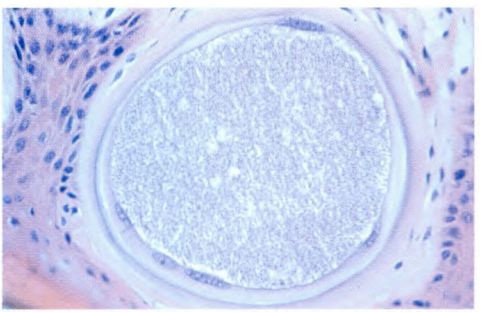

Fig. 188 *Besnoitia besnoiti*; thick-walled cyst found in the subcutaneous tissue, the cysts may be up to 600 µm in diameter [4]

HELMINTHS

- Nematoda found in the skin

Parafilaria bovicola "Verminous nodules", "verminous haemorrhagic dermatitis"

Location: Adults are found in subcutaneous nodules on the upper side of the body. Microfilariae are found in the bloody excretions of the skin nodules.

Hosts: Cattle

Species description: Males are 2–3 cm and females are 4–5 cm in length. The eggs measure 45 × 30 µm and contain microfi-

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lariae. Development occurs in *Musca* spp. which become infected when they feed on the skin lesions. When feeding on lacrimal secretions or wounds they transmit the microfilariae to the next definitive host. The verminous nodules are likely to break and ooze blood, causing a haemorrhagic dermatitis.

Geographic distribution: World-wide, especially many parts of Africa

Symptoms: Subcutaneous nodules on the upper side of the body; these nodules are painful and contain the worms. Nodules are likely to break when the gravid female starts with the excretion of eggs. These haemorrhages and nodules are typical signs of the infection with *P. bovicola*.

Significance: Nodules are painful and irritating, and the carcasses at slaughter are downgraded.

Diagnosis: Verminous nodules, haemorrhage, microfilariae in the exudate

Therapy: Ivermectin (0.2 mg/kg, sc.) is fully effective against adults. Nitroxynil (20 mg/kg, sc.) is also effective but must be repeated 3 days later. High doses of levamisole and fenbendazole (50 mg/kg, po.) daily for 5 days were effective.

Prophylaxis: Fly (and tick) control may reduce the entrance points of infective larvae.

(Figure 189)

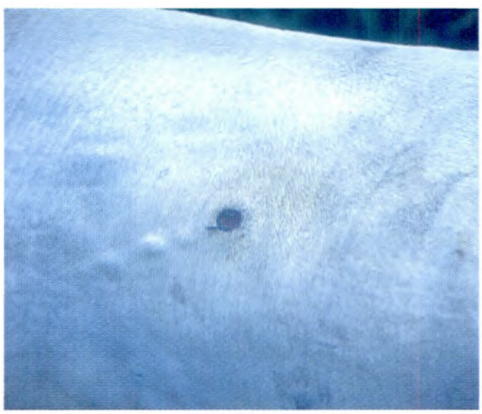

Fig. 189 *Parafilaria bovicola* causing "bleeding spots" and nodules on the skin of cattle; "bleeding spots" are markedly seasonally

Onchocerca dukei Muscular onchocercosis

Location: Adult worms of this species are found in the subcutaneous, perimuscular and muscular tissue of cattle in Africa.

Hosts: Cattle

Species description: The nodules are found in the ventral thorax region, abdomen, diaphragm and thighs. These nodules may be confused with the cysts of *Cysticercus bovis* (*Taenia saginata*). Microfilariae are 240–217 µm long and 4.8–6.7 µm wide and their predilection site is the subcutis of the lower thorax and the umbilical region. The parasite is transmitted by species of the genus *Simulium*.

Geographic distribution: Tropical Africa and also in sub-Saharan West Africa

Symptoms: Infections are usually clinically inapparent.

Significance: Losses occur mainly by condemnation of carcasses when a lot of subcutaneous and intramuscular nodules are seen during meat inspection. Such meat is repugnant.

Diagnosis: This is often made at meat inspection. Nodules are found particularly in the thorax, abdomen and diaphragm and may be confused with the cysts of *Cysticercus bovis* (☞ CATTLE, ■ 4.1). Skin biopsies soaked for 12 hours in physiological saline may be investigated after staining with Giemsa (☞ METHODS, 3.1). Typical microfilariae may be seen.

Therapy: This is not usually indicated.

Prophylaxis: This is generally not feasible in endemic areas (☞ Cattle, ■ 4.1.1).

Onchocerca gibsoni

Location: The adult worms are found in subcutaneous nodules on the brisket, shoulders and external surfaces of the hind-limbs. Microfilariae are found intradermally around the nodules with a predilection to the brisket region.

Hosts: Cattle

Species description: Adult worms cause nodular swellings in the skin but infected ani-

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mals are not clinically ill. Infected carcasses must be trimmed to remove the nodules. The adults lie in groups or "worm nests" in the subcutaneous tissue and a nodule is formed around them. The worm nests may be up to 5 cm in diameter. There is a fibrous capsule around the nodules which becomes thicker as the nodule ages. This capsule may eventually become calcified. The males are 30–53 mm and the females 140–190 mm long (sometimes up to 500 mm). The tail of the male is curved and it bears lateral alae and 6–9 papillae on either side. The spicules are unequal in size and measure 0.14–0.22 and 0.047–0.094 mm, respectively. The microfilariae are not sheathed and 240–280 µm long. The intermediate hosts are members of the genus *Culicoides* (e.g. *Culicoides pungens* and probably others).

Geographic distribution: Asia, Australia, southern Africa

Symptoms: The infections do not usually cause clinical signs. Nodules may be found at the predilection sites.

Significance: Infected carcasses may be trimmed or even condemned at slaughter.

Diagnosis: This is based on finding the adult worms in subcutaneous nodules or microfilariae in the skin of the brisket. Microfilariae are not evenly distributed in the skin, but occur in irregularly distributed "nests". A number of small skin biopsies (2 mm in diameter) may be placed in a drop of physiological saline solution for 1–2 hours. Microfilariae then migrate into the solution and can be examined.

Therapy: ☞ below *Onchocerca ochengi*

Prophylaxis: Unknown; insect repellents may reduce the attacks by the vectors.

Onchocerca ochengi (syn. *Onchocerca dermata)* "Intradermal onchocercosis of cattle"

Location: This species is found in subcutaneous and intradermal nodules on the ventral regions and flanks, primarily the udder and scrotum.

Hosts: Cattle

Species description: In endemic areas up to 50% of the cattle are infected. The intermediate host has not been identified yet but a biting insect is suspected. Small nodules occur deep in the hide. Microfilariae occur intradermally around the nodules. The microfilariae of *O. dukei* and *O. ochengi* are concentrated in the skin of the posterior and anterior belly.

Geographic distribution: East and West Africa

Symptoms: Intradermal nodules may be formed mainly in the abdominal region, on the udder and scrotum and sometimes in other parts of the body. Microfilariae can cause allergic reactions and local hyperkeratosis.

Significance: The value of leather, which is often perforated by these nodules, is markedly decreased by this parasite.

Diagnosis: Intradermal nodules can be detected by palpation. At slaughter they are visible on the internal surface of the hide.

Therapy: This is generally not indicated. However, the microfilariae may be killed by diethylcarbamazine (5–8 mg/kg, po. during 3 weeks), ivermectin (200 µg/kg, sc.), but also levamisole at increased dosage rates.

Prophylaxis: Unknown; insect repellents may reduce the attacks by the vectors.

(Figures 159, 190)

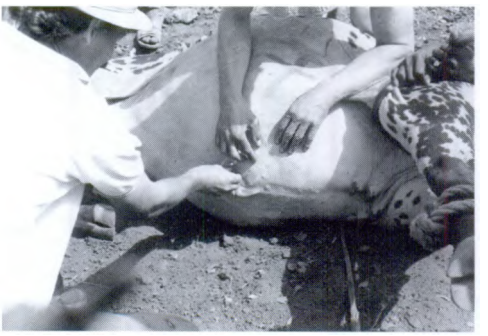

Fig. 190 *Onchocerca ochengi*; skin nodules [23]

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Dipetalonema dermicola and *Dipetalonema ruandae*

Remarks: These filariid worms are rarely found in cattle. The former occurs in the skin and the latter in the connective tissue of the oesophagus. They are non-pathogenic and the life cycle is unknown.

Setaria labiatopapillosa Bovine abdominal filariosis

Remarks: Microfilariae of this filariid worm which occurs in the abdominal cavity of cattle (☞ CATTLE, ■ 4.4) may occasionally be found in the skin. The microfilariae are sheathed, measure 240–260 µm and appear usually in the peripheral blood. Several species of mosquitoes act as vectors (*Aedes* spp., *Culex* spp. and *Anopheles gambiae* and other *Anopheles* spp.).

Stephanofilaria spp. (*S. stilesi*, *S. assamensis* and other *Stephanofilaria* spp.)

Remarks: *Stephanofilaria* spp. are small filarial parasites responsible for circumscribed dermatitis. *S. stilesi* causes a dermatitis along the ventral midline (between the brisket and the navel) of cattle. Adult worms are 3–6 mm long and usually found in the dermis, just beneath the epidermal layer. Microfilariae are 50 µm long and are enclosed in a spherical, semi-rigid vitelline membrane. The intermediate host for *S. stilesi* is the female horn fly *Haematobia irritans* and a *Musca* sp. for *S. assamensis*. The infective larvae are introduced into the skin as the horn fly feeds. For diagnosis, deep skin scrapings are macerated in isotonic saline and examined microscopically for adults and microfilariae must be differentiated from the microfilariae of *Onchocerca* spp. and *Setaria* spp. which are much larger (200–250 µm). No effective treatment is known yet for *S. stilesi*, but topical application of organophosphates (trichlorfon, 6–10%, daily for 7 days) have proved effective against other species of *Stephanofilaria*. Ivermectin (200 µg/kg, sc.) is effective against adult worms of *S. assamensis*.

(Figure 191)

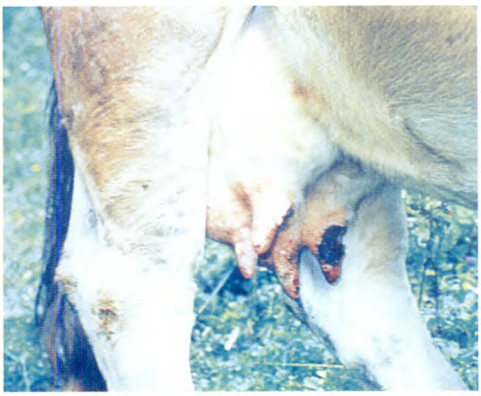

Fig. 191 *Stephanofilaria* sp., circumscribed dermatitis on the udder [15]

ARTHROPODS

Arthropods are divided into two main groups:

Arachnida
- *Ticks*
- *Mites*

Insecta
- *Lice*
- *Fleas*
- *Dipterida*

- **Arachnida found in/on the skin**

– Ticks

(Figure 192)

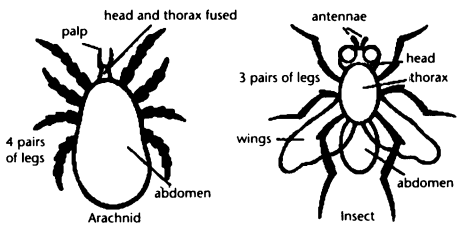

Fig. 192 Principle differences in the morphology of arachnids and insects [6]

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Ticks may impair cattle health in 3 different ways:

- **Direct noxious effects**

Attachment to the host causes irritation of the skin with subsequent ulceration and secondary infections. The wounds are attracted by screwworms and other flies and myiasis may develop. Heavy infestations are associated with anaemia, since adult female ticks imbibe up to 10 ml of blood. Furthermore the presence of large numbers causes annoyance and animals become anxious and restless which may be a cause of loss of weight and condition. The life cycle of ticks may involve one, two or three hosts (Figures 193, 194, 195, 196).

In cattle the following ticks have a direct noxious effect:

Fig. 193 Skin wounds: noxious effects of tick infestation

IXODIDAE ("hard ticks")

Amblyomma spp. *(A. variegatum, A. hebraeum)*
Boophilus spp. *(B. decoloratus, B. microplus)*
Dermacentor spp.
Haemaphysalis spp.
Hyalomma spp.
Ixodes spp.
Rhipicephalus spp. *(R. appendiculatus, R. sanguineus, R. evertsi, R. parvus, R. pulchellus, R. sinus)*

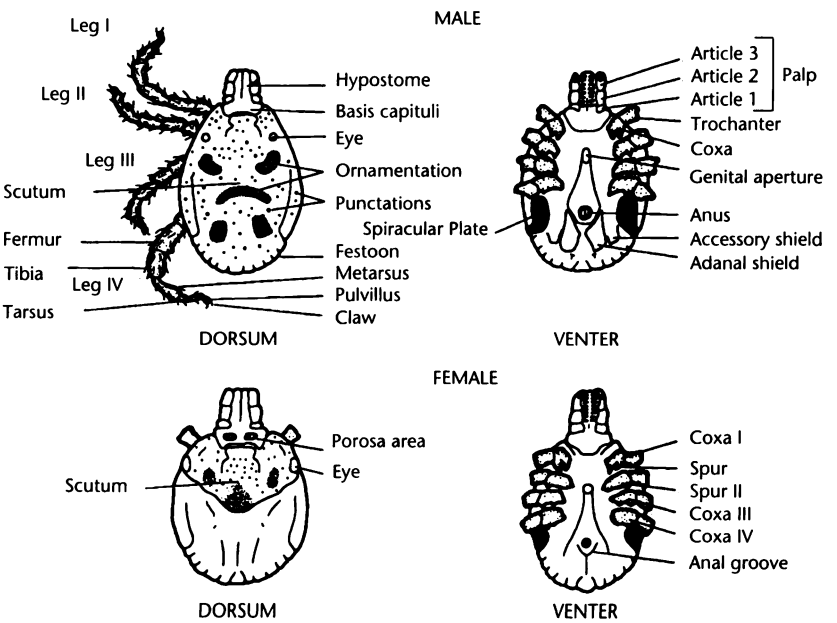

Fig. 194 Characteristics of hard ticks (Ixodidae) [24]

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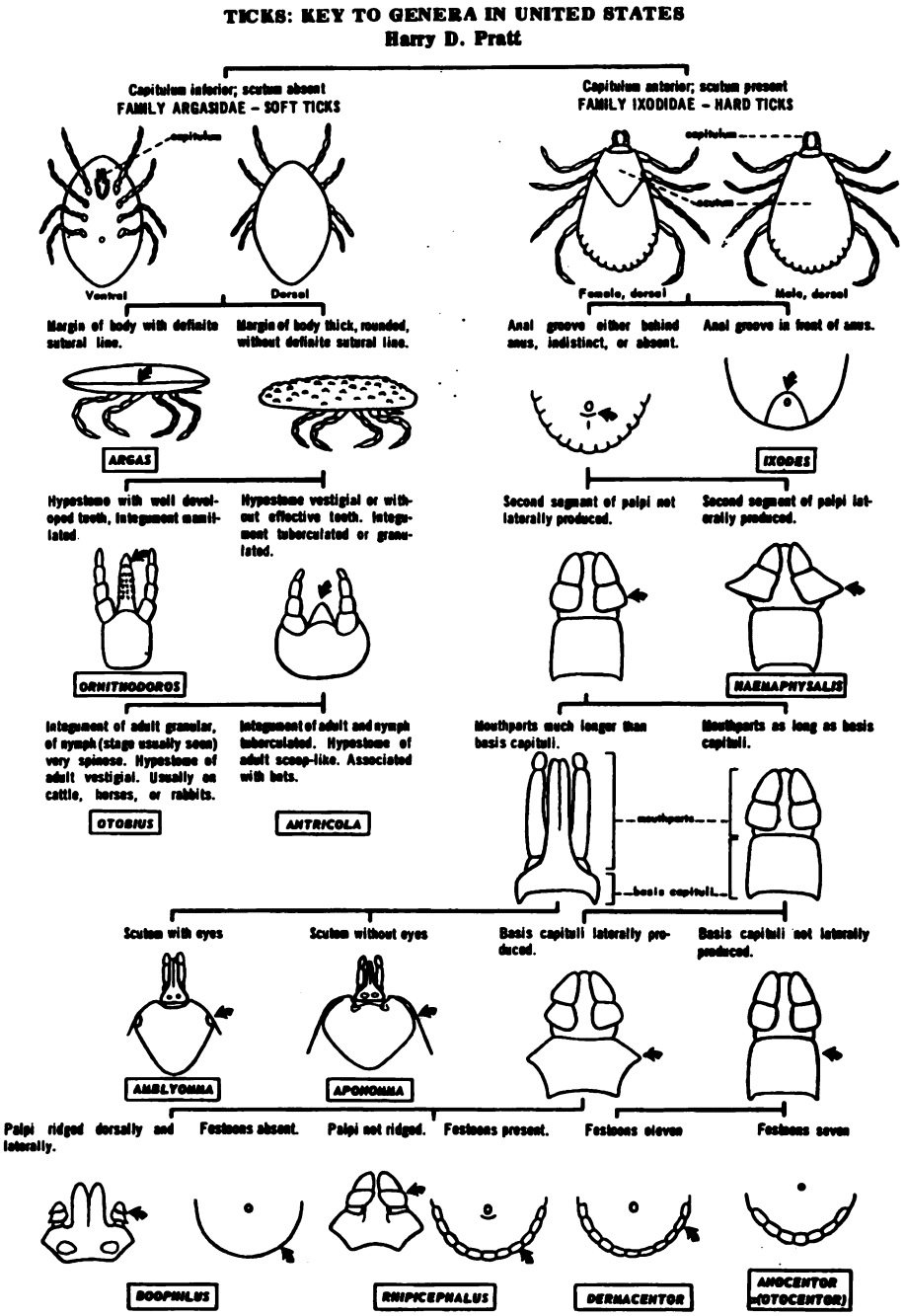

Fig. 195 Key for the identification of tick genera [25]

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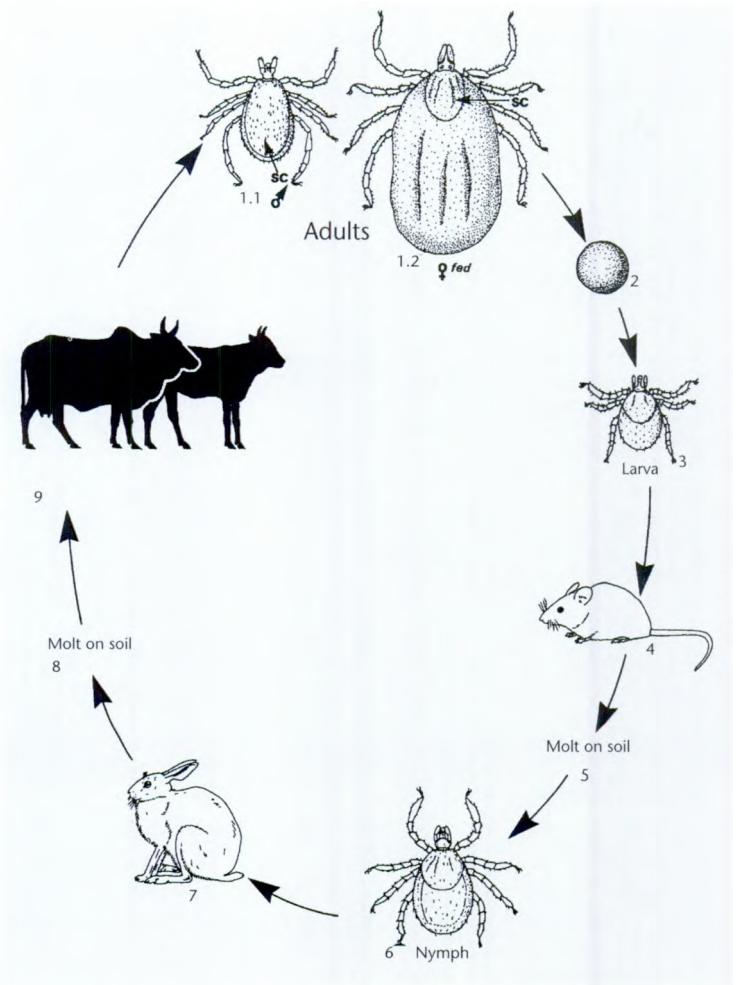

Fig. 196
Life cycle of a three-host tick (*Ixodes ricinus*)
1.1 and 1.2: Eyeless adults on their hosts; fed females (1.2) reach a length of up to 1.5 cm. Their scutum (SC) then appears small. Copulation occurs while the females feed; they then drop down to the soil;
2: Over a period of 1 month females lay about 2000 spherical to ovoid eggs, which become attached to each other and thus appear as clusters on the soil;
3 and 4: Larvae hatch from the eggs after 3–36 weeks (depending on the temperature) and creep onto the tops of grass, from where they attach to passing hosts (mostly small mammals, but also humans);
5 and 6: Fed larvae drop to the soil, and moult within 5–7 weeks (sometimes up to 5 months) to become eight-legged nymphs;
7 and 8: The nymphs attack larger mammals, suck blood for 2–7 days and drop to the soil, where they moult within 2–8 months and become sexually mature adults (1.1 and 1.2);
9: Mostly in spring the adults attack larger mammals including a variety of domestic animals and man, where especially females suck for 5–14 days. The whole development is temperature-dependent and in Europe needs about 3 years [26]

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ARGASIDAE ("soft ticks")
– ***Otobius megnini*** Spinose ear tick
(Figures 197, 198)

- **Transmission of diseases**

One of the most damaging effects of ticks is their ability to transmit diseases to their hosts. Some of these are serious with fatal consequences. Transovarian transmission from one tick generation to another via the eggs is possible and contributes to spreading the disease.

The following pathogens are transmitted by ticks:

Babesia bovis Redwater, tropical bovine babesiosis

Remarks: This is transmitted by *Boophilus microplus*, the pantropical blue tick (☞ CATTLE, ■ 2).

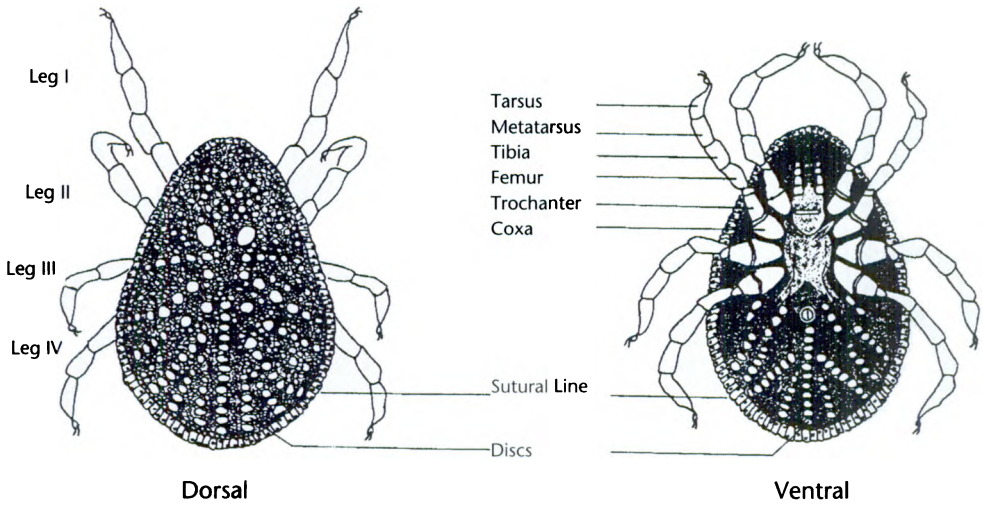

Fig. 197 Characteristics of soft ticks (Argasidae, *Argas* sp.) [26]

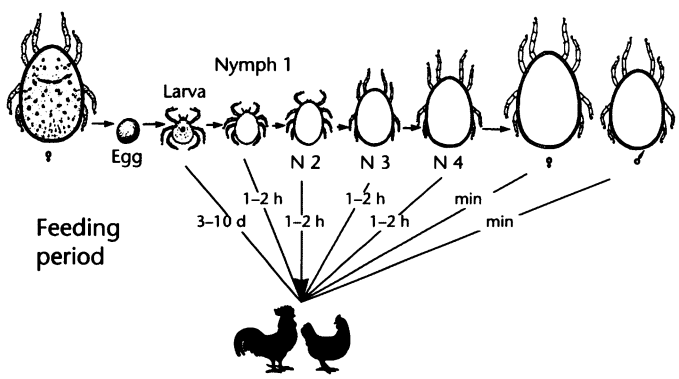

Fig. 198 Example of a typical argasid life cycle; development stages of *Argas* sp., which need about 3–36 months to mature (depending on the temperature); except for larvae, which suck blood for 3–10 days, all stages feed several times but only for a few minutes each time [24]

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Babesia divergens
European bovine babesiosis

Remarks: *B. divergensis* transmitted by *Ixodes ricinus* (☞ CATTLE, ■ 2).

Babesia bigemina (syn. *Piroplasma bigeminum*) Redwater, tropical bovine piroplasmosis, tick fever, "Texas fever"

Remarks: This parasite is transmitted by *Boophilus microplus; B. decoloratus* (the common blue tick), *B. calcaratus, Rhipicephalus* spp. (*Rh. evertsi, Rh. bursa, Rh. appendiculatus*) and *Haemaphysalis* spp. (*H. punctata*) (☞ CATTLE, ■ 2).

Babesia major (syn. *Piroplasma major*)
European bovine piroplasmosis

Remarks: *B. majoris* transmitted by *Haemaphysalis punctata* (☞ CATTLE, ■ 2).

Cowdria ruminantium
Heartwater, Tyewde

Hosts: Cattle, sheep, goat and other ruminants

Remarks: This parasite is transmitted by several species of the tick genus *Amblyomma*, particularly by *Amblyomma variegatum* (the tropical bont tick) and *A. hebraeum* (the South African bont tick). Other species which may be involved are *A. gemma, A. pomposum, A. lepidum* and *A. tholloni* (☞ CATTLE, ■ 2).

Theileria parva (syn. *T. bovis, T. lawrencei*)
East Coast Fever, bovine theileriosis, Corridor disease, Rhodesian tick fever

Remarks: This parasite is transmitted by *Rhipicephalus appendiculatus* (the brown ear tick) and *Rhipicephalus evertsi* (the red-legged tick) and *Rhipicephalus duttoni.* Other vectors are *Hyalomma excavatum, H. dromedarii, H. truncatum* and some other *Rhipicephalus* spp. (☞ CATTLE, ■ 2).

Theileria annulata Mediterranean Coast Fever, Tropical theileriosis, Egyptian Fever

Remarks: This parasite is transmitted by *Hyalomma detritum, H. truncatum* and *H. anatolicum* (☞ CATTLE, ■ 2).

Theileria mutans Benign bovine theileriosis, mild gallsickness

Remarks: This parasite is transmitted by *Amblyomma variegatum, A. hebraeum* and possibly also by *Rhipicephalus appendiculatus* and *R. evertsi* (☞ CATTLE, ■ 2).

Anaplasma marginale Malignant anaplasmosis of cattle, gallsickness

Remarks: This parasite is transmitted by *Boophilus decoloratus* and *B. microplus* (☞ CATTLE, ■ 2).

Anaplasma centrale Mild anaplasmosis of cattle, gallsickness

Remarks: This parasite is transmitted by *Boophilus decoloratus* (☞ CATTLE, ■ 2).

Eperythrozoon wenyoni

Remarks: This parasite is transmitted by *Hyalomma anatolicum.* The pathogen occurs on the erythrocyte surface (☞ CATTLE, ■ 2).

Ehrlichia bovis Tropical bovine ehrlichiosis, "Nofel" or "Nopel"

Remarks: This parasite is transmitted by tick species of the genera *Amblyomma, Hyalomma* and *Rhipicephalus* (☞ CATTLE, ■ 2).

Ehrlichia phagocytophila
European ehrlichiosis

Remarks: This parasite is transmitted by *Ixodes ricinus* (☞ CATTLE, ■ 2).

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Ehrlichia ondiri

Remarks: This parasite occurs in granulocytes of cattle in high altitude grassland areas of East Africa. The exact vector is unknown but ticks of the genus *Haemaphysalis* are suspected (☞ CATTLE, ■ 2).

Borrelia theileri Tick Spirochaetosis

Remarks: This is transmitted by *Boophilus decoloratus* and *Rhipicephalus evertsi*. Tick spirochaetosis occurs in cattle, sheep, goats and horses.

- **Tick toxicosis**

Tick paralysis

43 tick species belonging to 10 genera are known to induce tick paralysis in man and a variety of mammals. These ticks are capable of releasing a toxin into the host which causes a condition associated with progressive, ascending, afebrile, symmetrical paralysis, with hind legs being affected first followed by the forelegs.

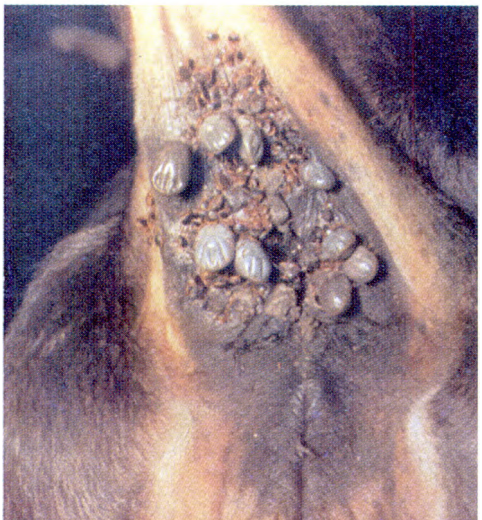

Fig. 199 *Rhipicephalus evertsi*; the red-legged tick, an important cause of tick paralysis [10]

Animals may die. Paralysis is relieved if ticks are removed in time. Most domestic animal species appear to be susceptible to tick paralysis.
Lambs and calves and to a lesser extent adult cattle are susceptible to tick paralysis. *Ixodes rubicundus* (the karoo paralysis tick) mainly affects sheep and goats in South Africa and *Rhipicephalus evertsi* causes the "spring lamb paralysis" in lambs but also in calves.
(Figure 199)

Sweating sickness

An acute, febrile tick-borne toxicosis characterised by a profuse, moist eczema and hyperaemia of the skin and visible mucous membranes. Watering of the eyes and nose, salivation and a extremely sensitive skin with a sour odour are other typical signs. Eventually the skin becomes cracked and predisposed to secondary infections (incl. screw-worm infections and myiasis). Often the course is acute and death may occur within a few days. In less acute cases recovery may occur.
It is mainly a disease of young calves but also sheep in eastern, central and southern Africa. The causative agents are certain strains of *Hyalomma truncatum* which produce an epitheliotropic toxin. Calf mortality may reach 70%.

Symptoms: Generalized hyperaemia with subsequent desquamation of the superficial layers of the mucous membranes of the upper respiratory, gastrointestinal and external genital tracts and profuse moist dermatitis. For diagnosis it is essential to determine the presence of the vector. Clinical signs appear 4–11 days after the tick bite.

Therapy: Ticks must be removed quickly. Antibiotics and anti-inflammatory agents are useful to combat secondary infections. Immune serum may be used.

Prophylaxis: Control of tick infestations is the only effective measure. Removal of ticks, symptomatic treatment and good nursing are indicated.

(Figures 200, 201)

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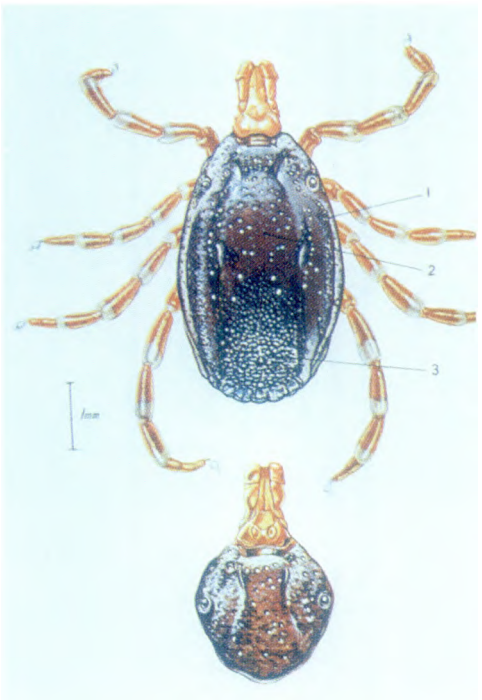

Fig. 200 *Hyalomma truncatum*; an important cause of sweating sickness; (1) marginal groove, (2) scutum and (3) pronounced punctuation [4]

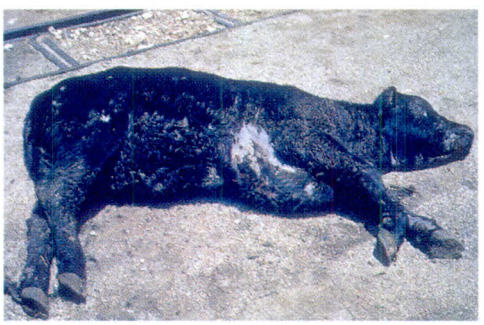

Fig. 201 Sweating sickness, an acute, febrile tick-borne toxicosis characterised by a profuse, moist eczema and hyperaemia of the skin. This condition is often caused by *Hyalomma truncatum* [4]

General toxicosis

Tick toxicosis is a general aggravation of the toxic effect of the parasite's saliva. Certain toxins have a suppressive effect on the animals and occasionally this may reactivate chronic infections. Infections with *Babesia* spp. and *Anaplasma* spp. may occur during massive *Rhipicephalus appendiculatus* infestations. Toxicosis, associated with general disorders, occurs with *Ornithodoros savignyi* (sand tampan) in young calves and lambs, especially when there are many bites. Animals show cutaneous oedema, haemorrhage, rapidly progressing weakness and prostration. Death can occur within 6 hours. Toxicosis may occur in recumbent animals, during rest.
(Figure 202)

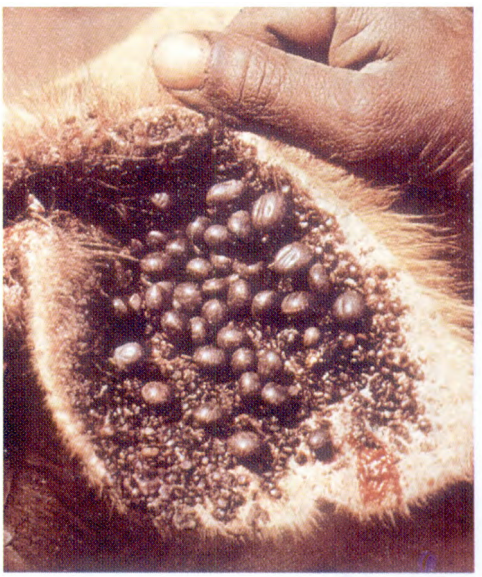

Fig. 202 *Rhipicephalus appendiculatus* (the brown ear tick); an important cause of tick toxicosis [10]

- **Description of ticks affecting cattle**

IXODIDAE ("hard ticks")

***Amblyomma* spp.**

Hosts: All domestic livestock species
Species description: 3-host ticks. *Amblyomma*

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spp. are large ticks and have ornate (patterned) upper body surfaces. Festoons (= rectangular division of the rear body edge) are present. *Amblyomma* ticks have long, prominent mouth parts, easily distinguished from the short mouth parts of *Dermacentor*. *Amblyomma variegatum* (the tropical bont tick) is the vector of heartwater and produces skin wounds with its large mouth parts. These wounds may become secondarily infected and may develop into abscesses. Tick paralysis may also occur due to the bont tick. The bont tick occurs mainly on the perineum, udder and in the axillae and inguinal areas. Some *Amblyomma* species, notably *Amblyomma hebraeum* (the South African bont tick) are often resistant to chemicals now in use.

Geographic distribution: *Amblyomma* spp. occur in central and southern Africa and many other parts of the world.

(Figures 203, 204, 205, 206, 207)

Fig. 203 *Amblyomma variegatum*; female [13]

Fig. 205 *Amblyomma variegatum*; male [13]

Fig. 204 *Amblyomma hebraeum*; female [13]

Fig. 206 *Amblyomma hebraeum*; male with bright festoons [13]

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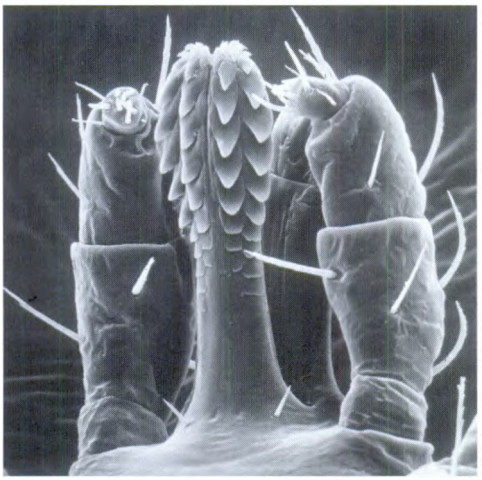

Fig. 207
Amblyomma variegatum; chelicera and palpes

Boophilus spp. Cattle fever tick, tropical cattle tick

Hosts: Horse and cattle

Species description: 1-host ticks. *Boophilus* spp. are plain brown in colour. They are inornate with eyes. Males are small (3–4 mm) and fully engorged females may be up to 12 mm in length. Members of *Boophilus* transmit several diseases and cause skin irritation which leads to licking and scratching and to secondary bacterial infections. Large numbers may cause anaemia. 1-host ticks such as *Boophilus* spp. are often the first to develop resistance to chemicals. Therefore control measures have to be changed frequently. *Boophilus decoloratus* (the blue tick) is one of the most important species of this genus and is found mainly on the neck, thorax, back and udder.

Geographic distribution: Throughout the world in warm climates

(Figure 208)

Fig. 208 *Boophilus* sp.; engorged female (up to 2 cm in diameter) [10]

Dermacentor spp.

Hosts: Many wild and domestic mammals

Species description: Some species are 1-host ticks while others are 3-host ticks. *Dermacentor* ticks have ornate (patterned) upper body surfaces. Festoons are present. The mouth parts are short.

Geographic distribution: Asia, Europe, North and South America and some parts of Africa; *Dermacentor* may damage their hosts by transmitting diseases (e.g. Q fever, tularemia), causing tick worry and anaemia.

(Figure 209)

Fig. 209 *Dermacentor marginatus*; male [4]

Haemaphysalis spp.

Hosts: Dogs, birds and in Africa to a lesser extent cattle

Species description: 3-host ticks which are

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small, inornate without eyes. Festoons are present. They may be found on the skin on all parts of the body. In addition to annoying cattle they may transmit cattle tick fever, anaplasmosis, and Q fever. They may also produce paralysis and anaemia in heavy infestations.

Geographic distribution: World-wide

(Figure 210)

Fig. 210 *Haemaphysalis punctata*; female [4]

Hyalomma spp. The bont-legged tick

Hosts: Horse, domestic ruminants and camel

Species description: Usually 2-host ticks, although three hosts may be used by some species. Inornate (sometimes ornate) eyes are present and festoons may be absent or present. Hypostome and palps are long. *Hyalomma truncatum* causes sweating sickness. Predilection sites are the distal parts of the legs, udder, tip of the tail and perianal region.

Geographic distribution: *Hyalomma* spp. occur in many parts of Africa, depending on the particular species.

(Figure 200)

Ixodes spp. *Paralysis ticks*

Hosts: Cattle, horse and many other animal species

Species description: 3-host tick. *Ixodes* ticks are plain brown in colour and the only eyeless species. The paralysis tick (*Ixodes rubicundus*) has a 2-year life cycle with specific requirements of temperature and humidity. *Ixodes* spp. are found anywhere on the skin of their host. *I. rubicundus* prefers lower parts of legs and abdomen. *Ixodes* spp. are a primary cause of tick paralysis in sheep and goats but they may also transmit diseases.

Geographic distribution: Europe, North America, South Africa

(Figures 211, 212)

Fig. 211 *Ixodes ricinus*; mouth parts [4]

Fig. 212 *Ixodes ricinus*; female (large) and male (small) [4]

Rhipicephalus spp.

Hosts: Cattle, sheep and goat and many other animal species

Species description: 2- or 3-host ticks; festoons are present. *Rhipicephalus appendicula-*

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tus (the brown ear tick) is a 3-host tick, plain brown in colour and occurs in the ears of the host. It is the chief vector of ECF and other diseases. Brown ear ticks are impossible to control by pasture rotation because they can survive up to 2 years on the pasture without feeding and they can feed on a large number of different species of wild and domestic animals.

Rhipicephalus evertsi (the red-legged tick) is a 2-host tick and has red legs. The adults typically occur around the anus and the nymphae are found deep in the ears. The red-legged tick is known to develop resistance to acaricidal drugs very quickly. It transmits ECF and other diseases.

Geographic distribution: Africa south of the equator

(Figures 199, 202, 213)

Fig. 213 *Rhipicephalus appendiculatus*; male (small) and egg excreting female (1 cm in diameter) [8]

ARGASIDAE ("soft ticks")

***Otobius megnini* Spinose ear tick**

Location: Larvae and nymphae feed deep in the external ear canal. Adults are non-parasitic and live on the ground.

Hosts: Horse, donkey, mule, cattle, sheep, goat

Species description: 1-host tick. Free-living adult females lay eggs on the ground which hatch within 3 weeks. Larvae and nymphs live and feed on one host for up to 7 months. Infestations of this species usually build up in kraals and stables, where the host densities are high. *O. megnini* can persist in empty kraals and stables for more than 2 years.

Geographic distribution: Arid and semi-arid areas of South and South West Africa

Symptoms: Infested animals shake their heads. Loss of appetite, debilitation and anaemia may be present. Irritation in the ear associated with secondary infections (incl. myiasis) may dominate the clinical picture. Ulceration (ear canker) may occur and ticks may clog the ears, causing deafness. Waxy and oily material is discharged from the ear. Larvae and nymphs may sometimes be seen in masses. Infested animals hold their head to one side and feeding is impaired. Weight loss may occur.

Significance: Heavy infestations cause otitis externa, great annoyance and blood loss. *O. megnini* may cause severe problems in livestock in many parts of Africa.

Diagnosis: Ticks may be found by swabbing the ear or by direct inspection of heavily infected ears. Larvae and nymphs are found inside the ear. The adult ticks are difficult to find as they breed in hidden cracks of barns, fences and trees.

(Figures 214, 215)

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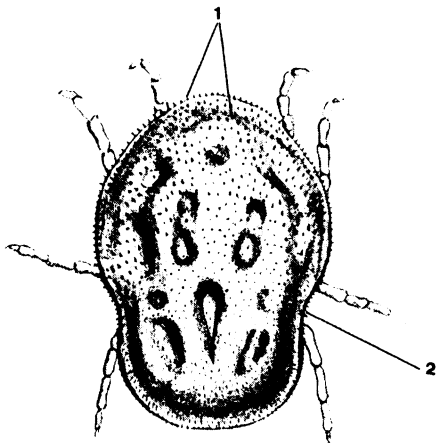

Fig. 214 *Otobius megnini* (the spinose ear tick); nymph (3–8 mm long): (1) integument covered with short, sharp spines and (2) posterior part of the body constricted [27]

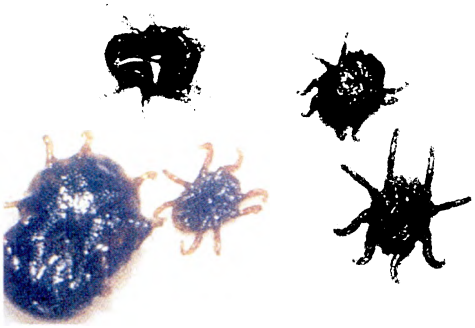

Fig. 215 *Otobius megnini*; nymphs (up to 8 mm long) [6]

Ornithodoros savignyi The sand tampan

Remarks: This tick may be a serious pest of cattle. It may cause death in calves and tick toxicosis in adult cattle (☞ SHEEP AND GOATS, 5.1).

- **Tick control in cattle**
  ☞ THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 141

– Mites

(Figures 216, 217)

Chorioptes bovis Chorioptic mite, foot mange, leg mite

Location: On the legs, base of tail and upper rear surface of the udder

Hosts: Cattle

Species description: The tarsal suckers have unjointed and short pedicels. Typical mite life cycle. Chorioptic mange feed on skin debris and lymph.

Geographic distribution: World-wide

Symptoms: Scabs or scales develop on the skin of the lower parts of the body. There is some exudation and crust formation on the lower body and legs, but this does not spread over a wide area. Cattle may stamp and scratch infected areas.

Significance: The disease is not as serious as sarcoptic or psoroptic mange but it is very prevalent.

Diagnosis: Demonstration of mites in skin scrapings taken from the edge of the lesions (☞ METHODS, 4.1).

Therapy: These mites are superficial and are therefore not very sensitive to Ivermectin (200 µg/kg, sc.) but it may be an aid in controlling the foot mange. Dips and sprays may be used (☞ below *Psoroptes ovis*). Ivermectin applied topically (500 µg/kg)

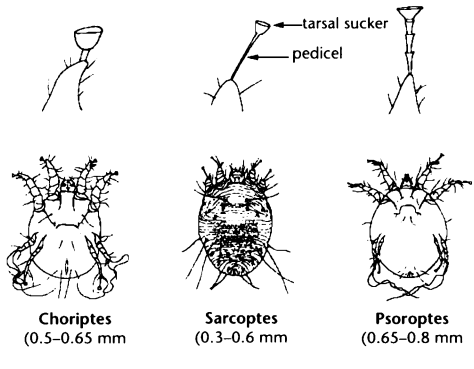

Fig. 216 *Chorioptes*, *Sarcoptes* and *Psoroptes* mites with genus-specific morphology of pedicels and tarsal suckers (schematic)

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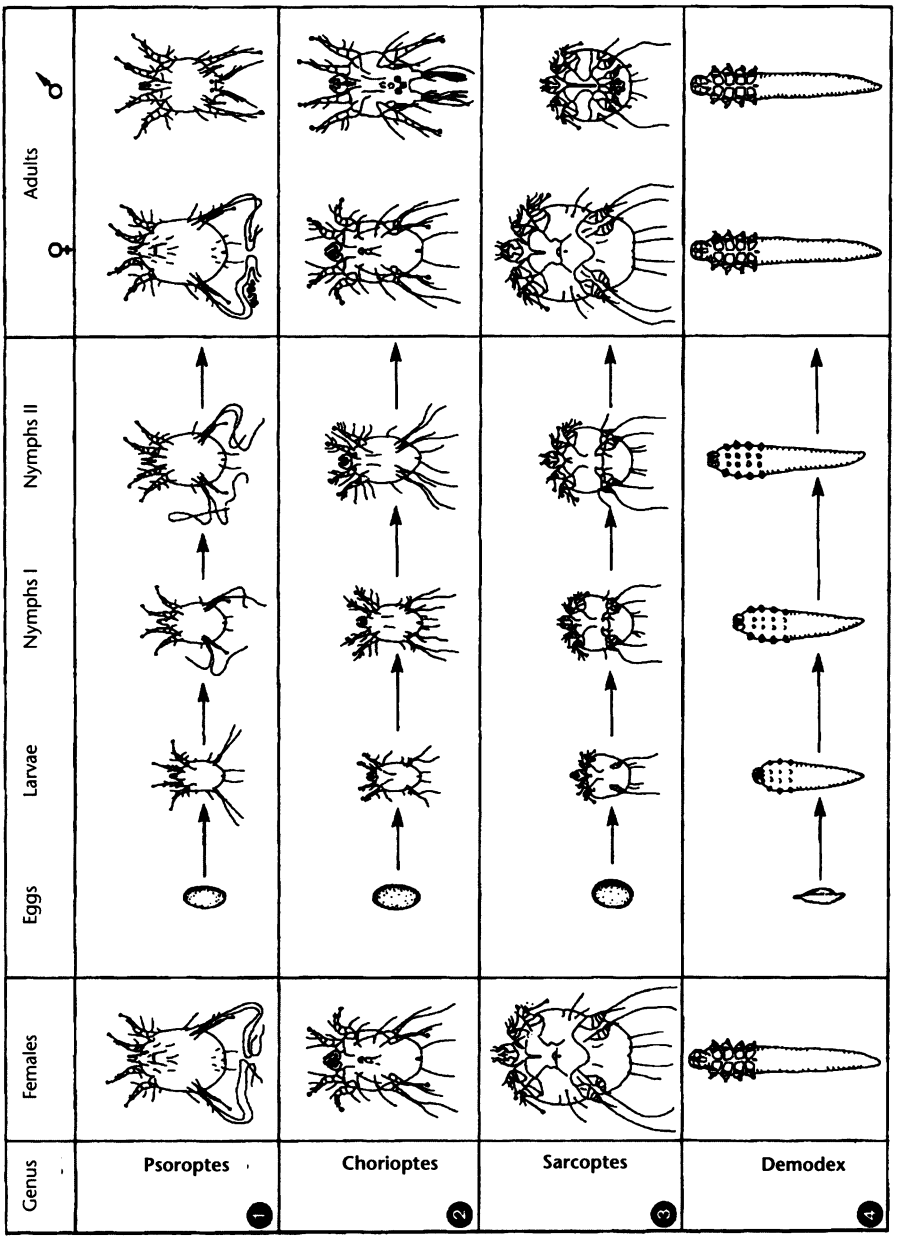

Fig. 217 Developmental stages in the life cycle of important groups of mites. All stages live on/in the skin of their hosts. Larvae have only three pairs of legs. Feeding larvae and nymphs increase in size and moult. In some species there is clear sexual dimorphism; (1) *Psoroptes* spp. feed (as piercing mites) on the lymph fluid and occasionally on the blood of their hosts, (2) *Chorioptes* spp. feed (as chewing mites) on the epidermal products, (3) *Sarcoptes* spp. penetrate the epidermis, forming canals and (4) *Demodex* spp. feed on hair follicles or on sebaceous glands [26]

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is effective against chorioptic mange. Crotoxyphos (0.25%) applied as a spray can also be used against the leg mite.

Prophylaxis: ☞ below *Psoroptes ovis* (Figures 218, 219)

Fig. 218 *Chorioptes bovis*, adult female (max. 650 µm long) with short, unjointed pedicels |4|

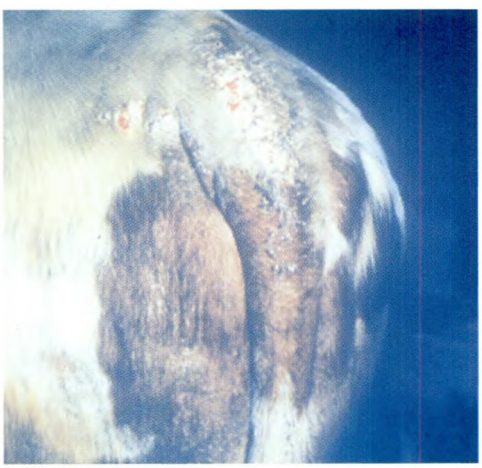

Fig. 219 *Chorioptes bovis* infestation |4|

Sarcoptes bovis Itch mite, mange mite

Location: Neck, the back in front of the tail, inner surface of the thighs and the udder and sometimes the whole body surface

Hosts: Cattle

Species description: This is a minute parasite in outline. The tarsal suckers have unjointed and long pedicels. Females have a number of spines on the upper surface. The life cycle is typical for mites, the entire period of development is spent on the host. (☞ SWINE, ■ 5.1) The mange may occur very seasonally in temperate zones (late winter). *Sarcoptes* mites prefer areas of thin hair but the lesions may spread to other parts. *Sarcoptes* mites puncture the skin to feed on lymph and skin debris.

Geographic distribution: World-wide

Symptoms: Intense itching is caused by irritation. Animals scratch which may result in dermatitis, accompanied by an exudate that coagulates and dries to form crusts. The skin is thickened, wrinkled and hair is lost.

Significance: Irritation to the host causes weight loss and emaciation. Skins of carcasses are of poor quality. Animals in poor condition due to mite attacks are subject to other diseases.

Diagnosis: Demonstration of mites of deep skin scrapings taken from the edge of the lesions (☞ METHODS, 4.1). *Sarcoptes* mites are usually found deep in the skin whereas *Psoroptes* mites are more superficial.

Therapy: This mite is very sensitive to Ivermectin (200 µg/kg, sc.). After one treatment no living mites were recovered. Dips and sprays may also be used to reduce the clinical effects (☞ below *Psoroptes ovis*).

Prophylaxis: ☞ below *Psoroptes ovis* (Figures 220, 221)

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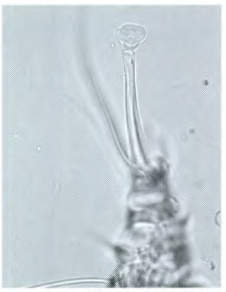

Fig. 220 *Sarcoptes* sp.; mite with unjointed, long pedicels (30–35 µm long) [4]

Fig. 221 *Sarcoptes* sp. infestation of a cattle [4]

Psorpotes ovis (syn. *Psoroptes communis var. bovis)* Psoroptic mite

Location: Any part of the body, especially areas of dense hair such as withers, back and root of tail

Hosts: Cattle, sheep

Species description: The mites are oval in shape and the tarsal suckers have long, jointed pedicels. Typical mite life cycle, taking 9 days. This is a notifiable and quarantinable disease in many parts of the world.

Geographic distribution: World-wide

Symptoms: Affected skin is covered with exudate. This dries to form a scab. Massive loss of hair usually occurs. Lesions may cover the entire body. Deaths in untreated calves are not uncommon. The course may be acute in young calves and chronic nature may also be found. The prognosis is usually favourable following early treatment.

Significance: Psoroptic mange is a widespread and serious disease of cattle. Infested animals lose weight and the skins are of lower quality.

Diagnosis: Demonstration of mites in skin scrapings taken from the edge of the lesions (☞ METHODS, 4.1).

Therapy: Infested cattle should be dipped, not sprayed. The following compounds may be used for dips: toxaphene (0.5–0.6%), coumaphos (0.3%), phosmet (0.2–0.25%), diazinon (0.025%), flumethrine (1 l of a 6% solution in 1500 l of water) and many other compounds. Depending on the product, repeated treatments are required (label instructions should be checked). Ivermectin (200 µg/kg, sc.) is effective against *Psoroptes ovis*. One treatment resulted in clinical healing, but a second treatment is indicated in eradication programs.

Prophylaxis: Newly introduced animals are the main source of infection for a herd. These animals must be checked carefully and possibly treated before being introduced into the new herd. A quarantine may be indicated.

(Figures 222, 223, 224)

Fig. 222 *Psoroptes* sp. female mite (max. 800 µm long) with egg [4]

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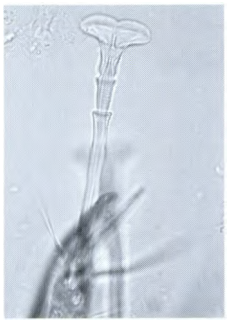

Fig. 223 *Psoroptes* sp.; mite, jointed, long pedicels (68 µm long) [4]

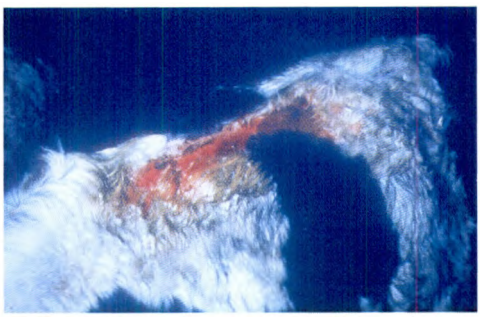

Fig. 224 *Psoroptes* sp. infestation of a cattle [4]

Demodex bovis
Demodectic or follicular mite

Location: Neck, brisket, shoulder, face
Hosts: Cattle
Species description: *Demodex* mites are cigar-shaped, elongated mites, about 0.25 mm long. The thorax bears 4 pairs of stout, short legs and the abdomen is transversely striated. The life cycle is not clearly understood. *Demodex* is transmitted from the cow to the calf while nursing and may cause considerable damage to hides. Lesions are pronounced in young dairy cattle but are rarely visible or palpable on beef cattle. Rarely the lesions may appear over the entire body.
Geographic distribution: World-wide
Symptoms: Small papules and nodules may be seen. They are red and thick and a waxy, white material can be expressed from them. This material contains numerous mites. Nodules may also be filled with pus and abscesses covered with small scales may be seen. The course of bovine demodectic mite infection is usually mild but may extend over many months.
Significance: Damage to skin may affect the production of leather. Demodectic mange is not considered to be a major parasite of cattle but it may open the skin for secondary problems (bacterial and fungal infections, myiasis, etc.).
Diagnosis: Microscopical examination of the cheesy, waxy fluid from the nodules which may contain many mites. Long-standing nodules of the skin are characteristic.
Therapy: There is no satisfactory treatment. Systemic and topical applications of chemical compounds have given some relief.
Prophylaxis: Unknown
(Figures 225, 226)

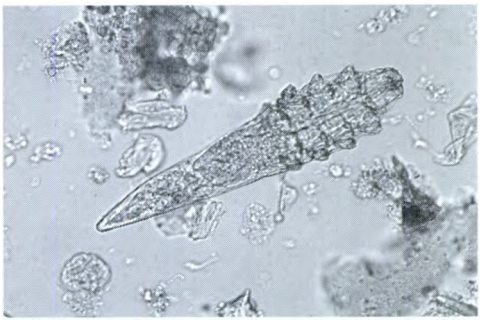

Fig. 225 Cigar-shaped *Demodex bovis* mite, (250–400 × 70 µm)

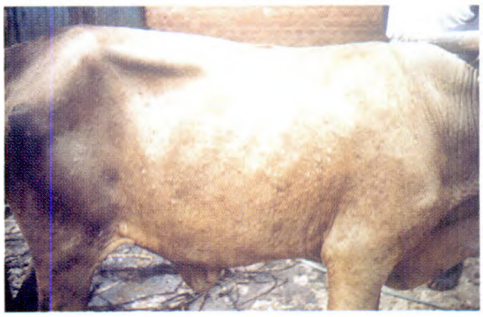

Fig. 226 *Demodex bovis* causing multiple skin nodules [8]

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Psoregates bos Itch mites of cattle, "Australian itch"

Remarks: Itch mites occur in Canada, Australia and South Africa. These mites are minuscule (189 × 189 µm) and difficult to collect. They are spherical and have paired claws and legs which are arranged radially. Alopecia and desquamation occur, but the lesions lack the scab formation associated with mange mite infestations. Lime-sulfur spray or dip applied twice, with a 2-week interval, is indicated to be the acaricide of choice.
(Figure 227)

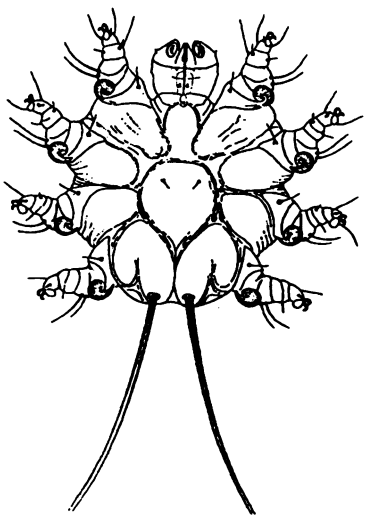

Fig. 227 *Psoregates* sp.; female mite [28]

***Raillietia* spp. (*R. auris* and *R. caprae*)**

Location: Middle and inner ear
Hosts: Cattle, sheep (*R. auris*) and goat (*R. caprae*)
Species description: The mites feed on epidermal cells and wax but not on blood.
Geographic distribution: North America, Australia, East Africa, Europe.
Symptoms: Infestations are usually inapparent but otitis media and interna with nervous signs, including head shaking, head rotation to the affected side, circling, and general incoordination, can be found in a progressive stage of the infestation.
Significance: *Raillietia* spp. infections can cause signs similar to those of other CNS infections and should therefore be excluded.
Diagnosis: It is almost impossible to diagnose *Raillietia* spp. antemortem. Demonstration of the mites in the middle or inner ear at necropsy.
Therapy: Ivermectin may eliminate the mites. Acaricides, applied topically may also be effective.
Prophylaxis: Unknown
(Figures 228, 229)

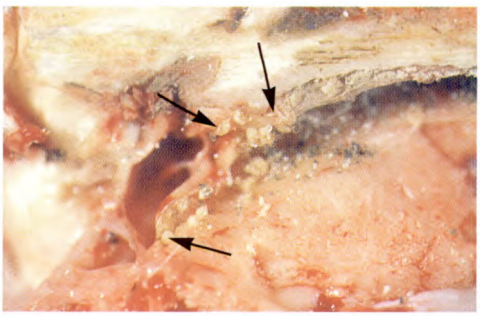

Fig. 228 *Raillietia auris* causing otitis media and interna

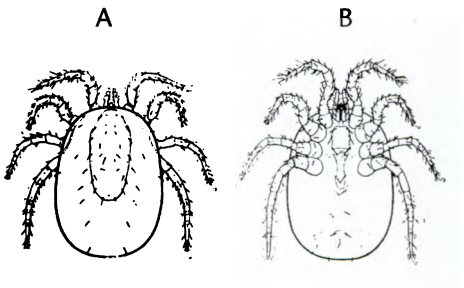

Fig. 229 *Raillietia auris*; dorsal view (A) and ventral view (B) [29]

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- Insecta found on the skin

– Lice

(Figure 230)

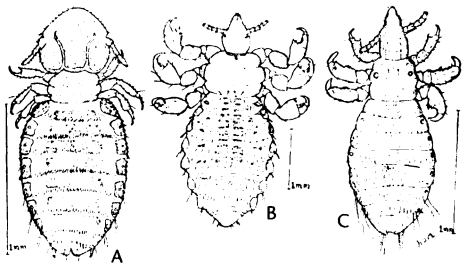

Fig. 230 Cattle lice: *Bovicola bovis* (A), *Haematopinus eurysternus* (B) and *Linognathus vituli* (C) [3]

MALLOPHAGA Chewing lice

Bovicola bovis *(syn. Damalinia bovis)*
Red louse, "chewing louse"

Location: Skin surface, especially the neck, withers and root of tail

Hosts: Cattle

Species description: These lice belong to the Mallophaga and are 1–2 mm long and wingless. They have a broad head with mouth parts adapted for chewing. The entire life is spent on the host. The infestations are heaviest in winter when the coats are dense.

Geographic distribution: World-wide, primarily in cooler areas

Symptoms: Intense itching associated with these lice may be observed. Animals show typical parasite worry (not feeding, not sleeping). Scratching may produce skin wounds or bruises and the coat becomes rough.

Significance: Biting lice are widespread and important parasites. Losses such as reduced growth and secondary skin infections due to intense itching may occur.

Diagnosis: Lice may be seen on skin. The eggs occur as white specks attached to the hairs.

Therapy: ☞ below THERAPY AND PROPHYLAXIS OF ECTOPARASITES

Prophylaxis: ☞ below THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 141

(Figure 231)

Fig. 231 *Bovicola bovis* (1.5–2 mm long); the cattle chewing louse [4]

ANOPLURA Blood sucking lice of cattle

Haematopinus eurysternus
Short-nosed cattle louse

Remarks: This species occurs world-wide. It is 3.4–4.8 mm long. The louse is fairly broad and the head is short.

(Figures 232, 233, 234)

Fig. 232 *Haematopinus eurysternus*; eggs attached to the hair of a cattle

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Fig. 233 *Haematopinus eurysternus* (3.4–4.8 mm long); the short-nosed cattle louse

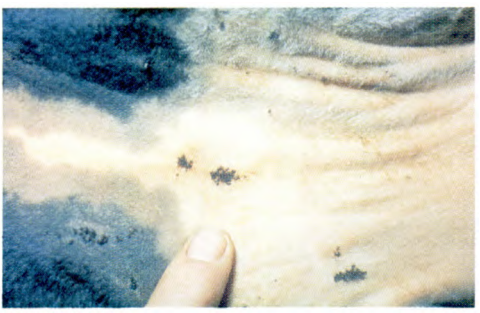

Fig. 234 *Haematopinus* sp.; on the skin of a cattle [15]

Haematopinus quadripertusus
Tail louse of cattle

Remarks: This species occurs on cattle in North America, Queensland, Papua-New Guinea and tropical Africa.

Haematopinus tuberculatus Buffalo louse

Remarks: This is a large species (up to 5.5 mm long) and occurs in Asia and the Pacific area. It also occurs on camels in Australia.

Linognathus vituli
Long-nosed cattle sucking louse

Remarks: This species has a long narrow head and a slender body. It is 2.5 mm in length. (Figure 235)

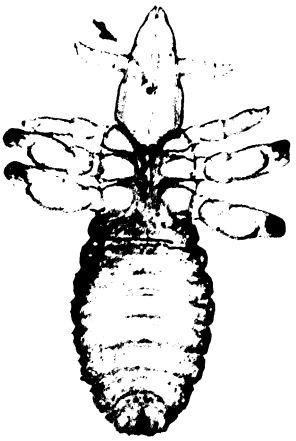

Fig. 235 *Linognathus vituli* (up to 2.5 mm long); the long-nosed cattle sucking louse [4]

Solenopotes capillatus

Remarks: This is the smallest of the sucking lice of cattle and occurs in conspicuous clusters on the neck, head, shoulders, dewlap, back and tail. (Figure 236)

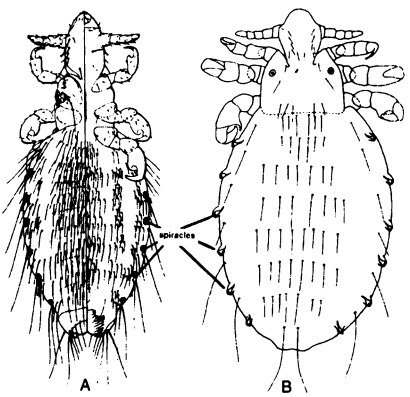

Fig. 236 (A) *Linognathus ovillus* (2.0– 2.5 mm) and (B) *Solenopotes capillatus* (1.3–1.7 mm) [29]

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- **General features of blood sucking lice of cattle**

Location: These lice are often found on protected areas of the skin such as side of the neck, brisket, back, head and between the legs.

Hosts: Cattle

Species description: Wingless insects with long mouthparts adapted for sucking. The life cycle takes 1 month. Heavy infestations may occur in immunosuppressed animals (e.g. calves following trypanosome infections).

Geographic distribution: World-wide. Some species are restricted to certain regions.

Symptoms: Louse worry is characterized by licking, scratching and rubbing. The coat becomes rough and secondary infections may occur. Heavy infestations may cause anaemia. Such animals may be more susceptible to other infectious diseases and environmental stress.

Significance: These are common parasites of cattle which can cause severe problems if present in large numbers.

Diagnosis: Lice and their eggs may be found on the skin.

Therapy: A wide variety of insecticides and application modi may be employed. Dipping is being replaced by delivery systems such as "pour-on" and "low-volume spray" for pyrethroid insecticides. Low tolerance for insecticides in milk limits the insecticides that may be used on dairy cattle and goats. Effective compounds include crotoxyphos, crotoxyphos combined with dichlorvos, coumaphos, fenvalerate, stirofos, coumaphos, dioxathion, malathion, methoxychlor, phosmet and permethrin. Label instructions should be considered. Dipping and spraying provides excellent coverage, and usually two treatments 2 weeks apart will effectively control lice. Ivermectin (200 µg/kg, sc.) is effective against sucking lice.

Prophylaxis: This is difficult wherever direct contact between the animals of the herd is possible.

- **Fleas**

No fleas have been found on the body surface of cattle.

- **Dipterida**

CULICIDAE Mosquitoes

***Aedes* spp., *Anopheles* spp. and *Culex* spp.**

Remarks: They belong like flies to the order Dipterida, with a single pair of wings. The main genera are *Anopheles, Culex* and *Aedes.*

They are slender with small spherical heads and large eyes. Both sexes live on fluids which are derived from organic sources, such as jus of fruits and vegetables. Females are capable to suck blood which appears to be necessary for the laying of eggs. Eggs are laid either on water or on vegetation floating on the surface of water. Therefore mosquitoes are found near stagnant pools. Because of their dependence on water their numbers generally become less during the dry season.

Significance: Mosquitoes can cause considerable distress to livestock but their main importance is their ability to act as intermediate hosts or vectors of viral (Rift Valley Fever, Equine encephalomyelitis, African Horse Sickness, Fowl pox, Blue tongue, Lumpy skin disease), bacterial (avian spirochaetosis), protozoan (Avian and human malaria) and filarial (*Setaria equina, Setaria labiatopapillosa*) and probably other diseases.

(Figures 237, 238, 239)

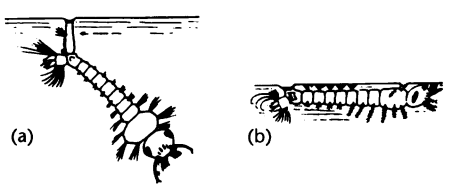

Fig. 237 *Culex* sp. larva (a) and *Anopheles* sp. larva (b) showing their feeding position in water

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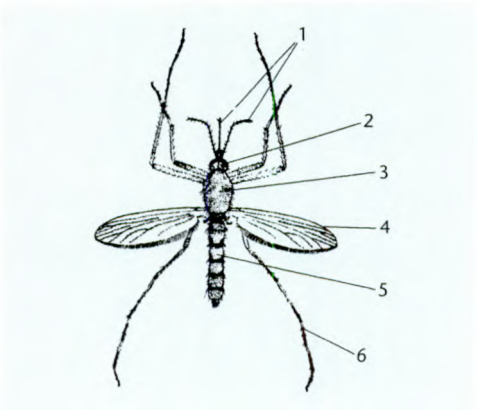

Fig. 238 Culicidae (schematic): adult mosquito with antennae and proboscis long (1), small head with large eyes (2), thorax (3), wings long and narrow (4), abdomen elongate (5) and legs long and slender (6) [27]

Fig. 239 *Culex* sp.; female in feeding position

SIMULIIDAE

Simulium spp. Black flies, midges

Remarks: Small size (1–5 mm), found in swarms near free-running well aerated streams. *Simulids* cause severe irritation to livestock when they occur in large numbers and herds and flock will stampede, often with disastrous results. Man, animals and poultry are liable to attack. Bites are inflicted on all parts of the body, giving rise to vesicles which burst exposing the underlying flesh. Skin wounds caused by simulids heal very slowly. Certain areas of the tropics are rendered uninhabitable by simulids.

Significance: Irritation of livestock; skin wounds with secondary infections and myiasis; transmission of *Onchocerca volvulus* in man

(Figures 240, 241, 242, 243, 244, 245)

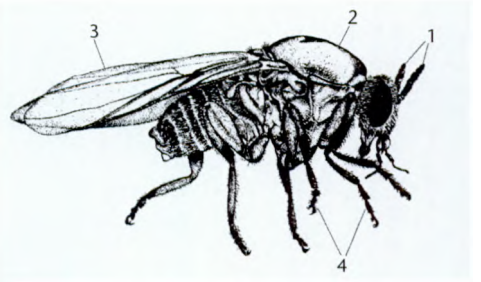

Fig. 240 *Simulium* sp. (blackfly) adult; (1) antennae short and horn-like, (2) humped thorax, (3) wings broad and clear and (4) legs short [27]

Fig. 241 *Simulium* sp., female (5 mm) [4]

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Fig. 242 *Simulium* larvae develop in free-running, well-aerated streams [30]

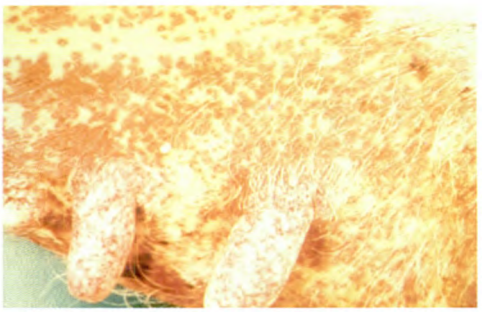

Fig. 244 Erythema and massive skin irritation on the udder due to *Simulium* bites [30]

Fig. 243 *Simulium* sp. larva with typical mouth brushes [30]

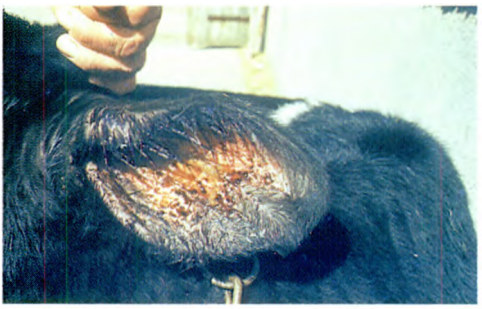

Fig. 245 Moist eczema-like skin alterations on the ear of a cattle following *Simulium* bites [30]

CERATOPOGONIDAE

***Culicoides* spp.** Biting midges, seasonal dermatitis, sweet itch

Remarks: They are very small (1–3 mm long). Adult female midges attack cattle, sheep, poultry, horse, man and other species, causing marked irritation by penetration of the skin with their proboscis. The bites cause intense itching. Only the females suck blood mainly during the twilight periods and at night. *Culicoides* occur often in large swarms a few hundred metres around the breeding sites. These are moist areas such as fresh or brackish water or seepages from decaying vegetable or dung heaps.

Significance: 1) Massive irritation of livestock, itching, allergic skin reactions (sweet-itch, seasonal dermatitis) in horses (☞ HORSES AND DONKEYS, 5.1), and 2) transmission of *Onchocerca gibsoni* and other *Onchocerca* spp. in cattle and horses, African Horse Sickness, Blue tongue of sheep. (Figure 246)

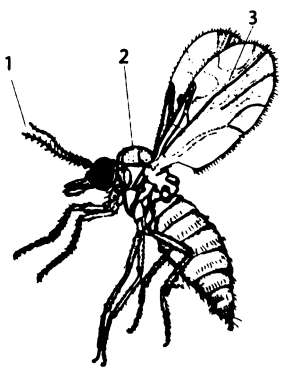

Fig. 246 *Culicoides* sp. (biting midge); adult (1–3mm long); (1) antennae long, (2) humped thorax and (3) mottled wings [20]

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TABANIDAE

***Tabanus* spp. *Haematopota* spp. and *Chrysops* spp.** Horse flies

Remarks: These robust flies have powerful wings and breed on leaves of plants in the vicinity of water. Female tabanids attack mammals to suck blood. They cause deep, painful, irritating bites. They generally bite several times for one blood meal because they are disturbed by the host's defense. A number of diseases (besnoitiosis, anaplasmosis, trypanosomosis and anthrax, hog cholera, equine infectious anaemia) are mechanically transmitted by tabanids. This is of special importance especially when horseflies are numerous among a crowded livestock population.
(Figures 247, 248, 249)

Fig. 247 *Tabanus* sp.; adult (17–25 mm) [13]

Fig. 248 *Haematopota* sp.; adult (8–13 mm)

Fig. 249 *Chrysops* sp.; adult (9–12 mm)

MUSCIDAE

***Musca* spp., *Lyperosia* spp. and *Haematobia* spp.** Muscid flies

Remarks: Muscid flies are annoying livestock especially during the summer or rainy season. They are swarming around farm livestock. The resulting irritation is incessant and much of the energy of the animals is lost to defend against fly attacks. In addition to the nuisance the flies often carry pathogens on their feet and bodies. Some also act as intermediate hosts for other parasites. Two genera need to receive special attention: *Musca* and *Haematobia*.

Musca domestica (the common house fly) and other *Musca* spp. are non-biting muscid. *Musca autumnalis* is attracted to wounds and other moist parts of the body, especially the eyes. It may provoke conjunctivitis and an ulcerative dermatitis. The infectious keratoconjunctivitis ("pinkeye") mainly caused by *Moraxella bovis* may be transmitted by muscid flies, especially *M. autumnalis*. *M. domestica* is known to transfer pathogenic bacteria mechanically from one wound to another. It is also capable of transmitting numerous pathogenic agents of medical and veterinary importance (*Salmonella*, *Shigella*, *Entamoeba histolytica*, cestodes of poultry, *Habronema* spp., *Thelazia* spp., *Parafilaria bovicola* and others). The preferred breeding place is in the faeces of animals or in decaying organic matter.
(Figures 250, 251, 252)

Fig. 250 *Musca domestica*; adult (6–7 mm) [12]

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Fig. 251 *Musca domestica*; larvae [4]

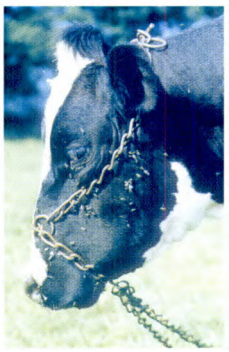

Fig. 252 *Musca autumnalis*; adult flies in situ

Haematobia minuta and other *Haematobia* spp. (the horn flies, buffalo flies) are biting muscids and act as vectors of *Stephanofilaria stilesi* in cattle and *Parafilaria multipapillosa* in horses. The adults of this fly live almost permanently on cattle, buffalo, sheep and other animals. They congregate along the backs where their bites cause severe irritation so that the cattle rub themselves raw. The female only leaves her host briefly to lay her eggs in fresh cattle or buffalo dung. The larval development requires a high relative humidity of nearly 100%. The fly is therefore widely distributed in Central, East and southern Africa. The horn flies are obligatory parasites of cattle and a serious pest wherever they occur. *Haematobia* spp. cause intense worry and irritation to animals, the bite being very painful. Serious blood loss may occur when large numbers attack and loss of condition, reduced performance is a common result. The flies cause sores at the base of the horns, on the poll, ears, neck, withers and tail root.
(Figures 253, 254, 255)

Fig. 253 *Haematobia* sp.; adult flies on the back of a cattle

Fig. 254 *Haematobia irritans*; adult flies (5–6 mm) [10]

Stomoxys calcitrans and *Stomoxys nigra*
Stable fly

Remarks: *S. calcitrans* also belongs to the muscids. It attacks almost all livestock species. *Stomoxys* breeds in manure (preferably horse manure) or in decaying organic matter. They require a very damp situation (heaped-up decaying vegetable matter). It is a biting muscid and occurs world-wide. Both sexes of this fly are bloodsuckers and can become extremely irritating pests of man and domestic animals. On cattle the flies prefer to feed on the legs while on dogs the ears may be so bitten that they become sore and scabby. Their salivary secretions

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cause toxic reactions with an immunosuppressive effect, rendering the host more susceptible to diseases.

Significance: *S. calcitrans* acts as intermediate host of *Habronema microstoma* (☞ HORSES AND DONKEYS, 1), *Trypanosoma evansi, Anthrax, Dermatophilus congolensis*, agents of the lumpy wool in sheep and probably many other pathogens. Its painful bite causes intense worry and irritation to animals. It may produce toxic reactions and immunosuppression. Blood loss may be marked following continuous, heavy attacks.

Control: Sanitation is the most important control measure in stable fly control. Manure, straw and decaying matter should be kept away from the environment of cattle, since this provides the development medium for the flies. If good sanitation procedures are practiced, then chemical control is less likely to be needed. Without sanitation chemical control measures are likely to fail.

(Figures 255, 256, 257)

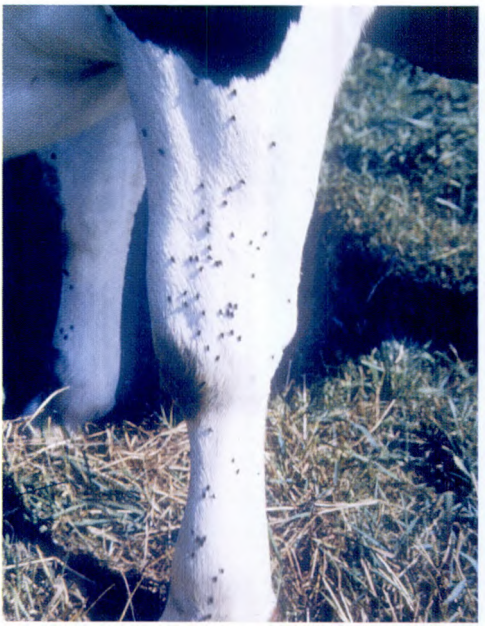

Fig. 257 *Stomoxys calcitrans*; adult flies in situ

Fig. 255 *Stomoxys calcitrans* (the stable fly); adult fly (4–6 mm) with a prominent and rigid proboscis; the maxillary palps are shorter than the proboscis and the thorax has longitudinal stripes [27]

Fig. 256 *Stomoxys calcitrans*; adult fly (4–6 mm) with prominent biting mouthparts (arrow) [10]

GLOSSINIDAE

***Glossina* spp.** Tsetse flies, "flies that are destructive of cattle" from Sechuana

Remarks: Most tsetse flies are active during the daytime and hunt by sight and smell and feed every 2 to 3 days. Both sexes suck blood and are equally capable of transmitting trypanosomes. The female is larviparous, producing one larva at a time and an estimated total of 8–12 larvae during her life. Apart from two species in south-western Arabia, tsetse flies are confined to the African continent, where they infest around 7.5 million square kilometres, mainly tropical regions on either side of the equator. Wherever these bloodsucking flies are, they render the area unsuitable for man and his livestock.

(Figures 258, 259)

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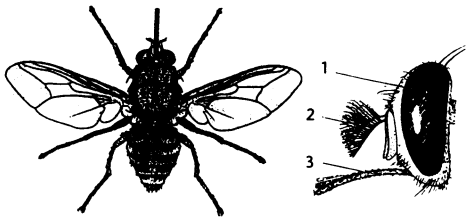

Fig. 258 *Glossina* sp. (tsetse fly); adult flies are robust insects, ranging in size from 6–16 mm and can be distinguished from other flies by their wing venation; the head shows prominent eyes (1), charactersitic antennae (2) and a prominent piercing proboscis [27]

Fig. 261 *Glossina palpalis*; adult in feeding position [4]

Fig. 259 Pupae (6–7 mm long) of tsetse flies (*Glossina* spp.) [13]

Fig. 262 Riverine habitat of *Glossina palpalis* [13]

Some 31 species of tsetse flies are known which can be divided into three groups, each with different habits and requirements: 1. Flies of the *Glossina palpalis* group are riverine species and feed on reptiles and ungulates. 2. Flies of the *Glossina morsitans* group are savannah and dry thorn-bush species which feed mainly on large animals. 3. Members of the *Glossina fusca* group occur in the rainforest and have require-ments between the first 2 groups, preferring dense shade and riverine thickets.

Fig. 260 *Glossina palpalis*; adult [13]

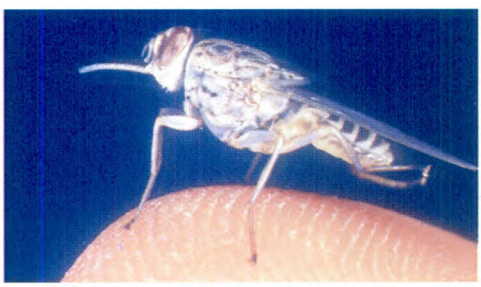

Fig. 263 *Glossina morsitans* with a prominent piercing proboscis [13]

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Fig. 264 *Glossina morsitans*; adult (8–11 mm) [13]

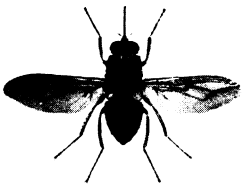

Fig. 265 *Glossina fusca*; adult [13]

Fig. 266 *Glossina tachinoides*; adult [13]

Significance: Tsetse flies are vectors of human and animal trypanosomosis. Trypanosomosis of domestic animals is caused mainly by *T. congolense*, *T. vivax*, *T. brucei* and *T. simiae*. (☞ CATTLE, ■ 2). The most important vectors are *Glossina morsitans*, *G. palpalis*, *G. tachinoides*, *G. fusca*, *G. brevipalpis* and *G. pallidipes*.
(Figures 260, 261, 262, 263, 264, 265, 266, 267)

Fig. 267 Biconical tsetse trap [4]

OESTRIDAE

Hypoderma spp. Warble fly, cattle grubs, heel flies

Location: Adult flies lay eggs on hairs of legs and occasionally of the body. Larvae migrate through several tissues of the host's body and appear in the subcutaneous tissue of the back.

Hosts: Cattle

Species description: Adult warble flies are hairy insects like bumble-bees. They do not feed and only live for a few days. The females lay their eggs on the hind legs of cattle and occasionally horses. The eggs of *Hypoderma bovis* are attached singly to an individual hair and the hatched larvae penetrate the skin and migrate along nerves to the spinal cord and then through the dorsal muscles till they come to lie below the skin on the back. *H. lineatum* eggs are attached in rows of 7–20 eggs per hair. Larvae penetrate the skin, migrate first to the oesophageal region and thence to the back. In spring the larvae leave through the holes, drop to the ground and burrow into soil to pupate.

Geographic distribution: Many countries of the northern hemisphere. Warble flies are reported from northern and southern Africa and are not widespread in tropical areas.

Symptoms: Clinical symptoms are rarely seen during the migratory phase, except if larvae are killed during their passage through the spinal cord. Paralysis may then occur. Nodules with an opening are usually seen in the skin of the back. These cysts contain a larva. When the adult flies attack cattle, stampeding may occur.

Significance: The adult flies severely disturb cattle which become apprehensive and attempt to escape by running away, often aimlessly. Consequently feeding and performance is reduced. In addition, warble flies cause extensive damage to the skin of cattle and the commercial value of the hides is markedly reduced. *H. bovis* larvae may cause paralysis when dying or being killed

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during their migration through the spinal canal. Severe oedema may occur when larvae of *H. lineatum* are killed during their migration through the oesophageal region.

Diagnosis: Larvae in cysts or lumps under the skin of the back indicate warble infestation. Eggs may be found on hair of the animal's legs.

Therapy: Cattle, especially calves, in areas where grub numbers are high, should be treated as soon as possible after the end of the warble fly season. They should not be treated later than 8–12 weeks before the anticipated first appearance of grubs in the backs, since adverse reactions may occur when migratory larvae are killed in the spinal cord. Pour-on treatments of coumaphos, famphur, fenthion, phosmet, trichlorfon or ivermectin may be used and poured evenly along the midline of the back. Sprays containing coumaphos or phosmet also control cattle grubs. The entire surface of the skin should be wet for sufficient absorption. Coumaphos and phosmet may also be used as dips. Ivermectin (200 µg/kg, sc.) is highly effective against cattle grub. Oral application is also effective.

Prophylaxis: Attacks by adult warble flies are difficult to avoid and entail excessive use of insecticides. Early treatment in endemic areas is indicated.

(Figures 268, 269, 270, 271)

Fig. 268 *Hyporderma bovis* (warble fly); adult (15 mm long) [4]

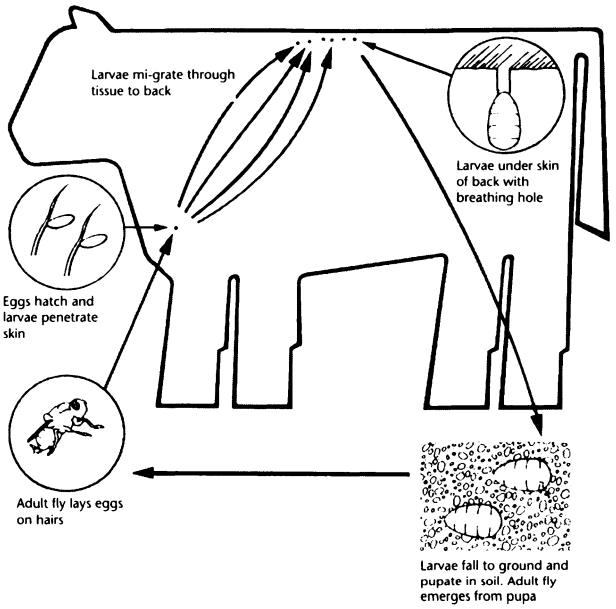

Fig. 269 Life cycle of *Hyporderma* spp. [6]

Fig. 270 *Hyporderma bovis* larvae causing nodules on the back

Fig. 271 *Hypoderma bovis*; section through a skin nodule containing a larva

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CALLIPHORIDAE The blowflies and their allies

Remarks: They are highly important in many domestic animal species and man. The adults are free-living and the larvae are parasitic maggots which develop in the tissue of their host, causing a condition called myiasis. The larvae may be laid into pre-existing wounds. Myiasis is often a secondary skin problem.
(Figure 272)

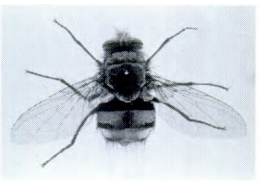

Fig. 272 *Calliphora* sp.; adult (12 mm long) [4]

The family of Calliphoridae can be divided into two groups, the **metallic flies** and the **non-metallic flies.**

The **metallic flies** are green, blue or purplish coloured and belong to the genera *Lucilia, Chrysomyia* and *Callitroga*

***Lucilia cuprina* and other *Lucilia* spp.** "Green-bottle" or "copper-bottle" flies

Remarks: *L. cuprina* is the predominant cause of sheepblow fly strike in South Africa and Australia. It causes myiasis in sheep. It is the most important primary blow fly initiating strikes on living sheep (☞ SHEEP AND GOATS, 5.1).
(Figure 273)

Fig. 273 *Lucilia sericata* (the green-bottle flies); adult (8–9 mm)

Chrysomya bezziana Cattle screwworm, the Old World screwworm, blow fly strike of cattle

Remarks: *C. bezziana* infests cattle but also horses, sheep, dogs and sometimes man. It occurs in tropical and southern Africa and oriental regions. *C. bezziana* causes severe myiasis and toxins produced by the larvae result in retarded healing of wounds. Death may occur in severe cases.
(Figure 274)

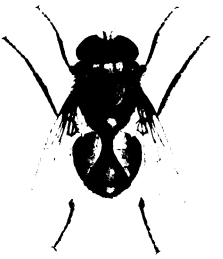

Fig. 274 *Chrysomya bezziana* (the cattle screwworm); adult (8–10 mm) [20]

Callitroga hominivorax (syn. *Cochliomyia hominivorax)* "American screwworm"

Remarks: *C. hominivorax* has recently been found to occur also in North Africa. Cattle, pigs and equines suffer most frequently, but other animals, including fowls and even man may also be affected. Pathology is essentially the same as in *C. bezziana.*
(Figures 275, 276)

Fig. 275 *Callitroga hominivorax* (syn. *Cochliomyia hominivorax*) the "screwworm"; adult (8–10 mm long) [19]

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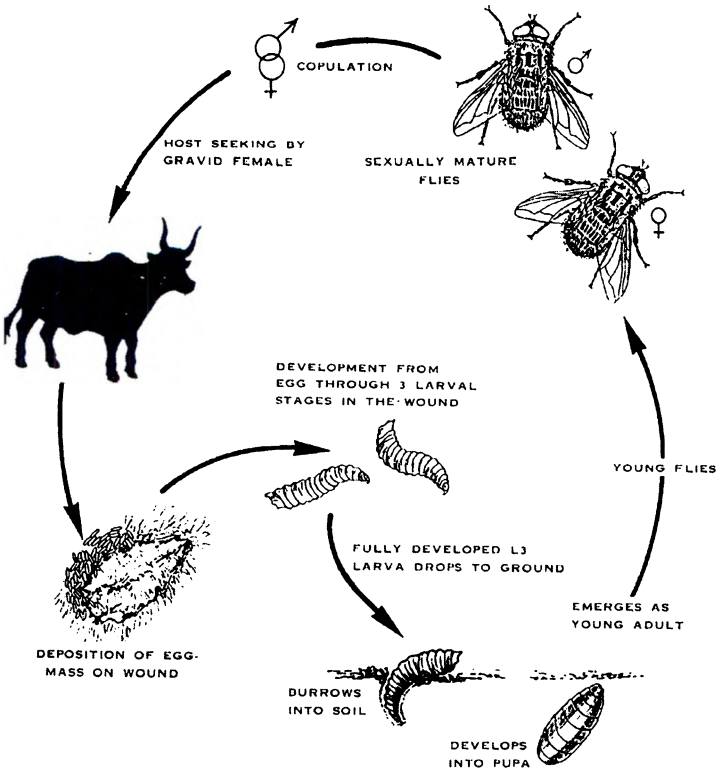

Fig. 276 Life cycle of *Callitroga hominivorax* [31]

The **non-metallic flies** are dull grey, yellow-brown or black and belong to the genera *Wohlfartia*, *Sarcophaga* and *Cordylobia*

Sarcophaga haemorrhoidalis
Red-tailed fleshfly

Remarks: The fly is very common in Africa south of the Sahara and is frequently found around human habitations. Flies of this species are larviparous and may lay their larvae in wounds or sores although larvae may also be deposited on faeces, carrion or fresh meat. Several animal species and man may be affected. The fly may cause myiasis, especially in sheep in some parts of the world.
(Figures 277, 278)

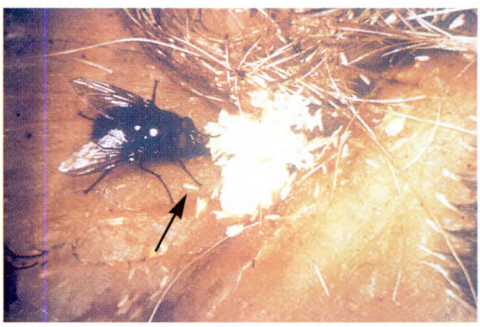

Fig. 277 *Sarcophaga* sp.; adult fly depositing its larvae on the skin of a cattle [8]

Fig. 278 *Sarcophaga* sp.; adult fly (13–15mm long) [4]

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Wohlfartia magnifica
Old World fleshfly

Remarks: This species occurs in North Africa. The fly attacks man and other animals. The larvae may be deposited into the external ear of man or in sores around the eyes.
(Figure 279)

Fig. 279 *Wohlfartia* sp.; adult fly (8–14 mm long) |4|

Cordylobia anthropophaga
Tumbu fly or "skin maggot fly"

Remarks: The fly is widely distributed in Africa south of the Sahara. It produces myiasis in man, dogs and other domestic animals. Eggs are laid around places where animals lie. After hatching the larvae penetrate the skin of the host, producing a boil of about 1 cm across with a hole in the centre. When mature the larvae wriggle out of the hole and pupate on the ground. Tumbu boils are found commonly on the ventral parts of the body but also on any other area of the body.
(Figures 280, 281)

Fig. 280 *Cordylobia anthropophaga* (Tumbu fly); stout fly with two black marks on the thorax; the face and legs are yellow (8–12 mm long) |27|

Fig. 281 *Cordylobia anthropophaga*; third-stage larva (12–28 mm long) |27|

HIPPOBOSCIDAE
The louse flies

Hippobosca equina Horse louse fly

Hosts: Horse and cattle
Geographic distribution: World-wide
(☞ HORSES AND DONKEYS, 5.1)
(Figures 282, 283)

Fig. 282 *Hippobosca equina* (horse louse fly; 7–9 mm long)

Fig. 283 *Hippobosca* sp.; adult louse flies on a cattle |4|

Hippobosca variegata

Hosts: Horse and cattle
Geographic distribution: Tropical Africa and oriental regions

Hippobosca rufipes Cattle louse fly

Hosts: Mainly wild and domestic Bovidae, less frequently horses
Geographic distribution: Tropical Africa (arid and semi-arid areas of Africa)
(Figure 284)

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Fig. 284 *Hippobosca rufipes* (cattle louse fly); this fly is very hard-bodied and difficult to squash. It has well-developed wings and strong legs ending in well-developed claws (8–11 mm long) [27]

Hippobosca maculata

Hosts: Horse and cattle
Geographic distribution: Tropical and subtropical areas of Africa
(Figure 285)

Fig. 285 *Hippobosca maculata*; adults (8 mm long) attached to the jugular vein region of a cattle [8]

Hippobosca camelina Camel louse fly

Hosts: Camel, horse and occasionally cattle
Geographic distribution: Northern Africa and Middle East

- **General features of *Hippobosca* spp.**

Location: The adult louse flies remain for long periods on their host and cluster in the perineal region, between the hindlegs to the pubic region, but may also bite on other parts of the body.
Species description: The flies live permanently on their host and feed on blood. Mainly cattle and horses are affected. They rarely fly and then usually not more than a few metres. They spend their whole time on the host and are strongly attached to their host and therefore difficult to dislodge. When disturbed they can quickly move sideways.
Symptoms: Infested animals scratch and rub, and skin-trauma are often seen as a consequence of heavy infestations.
Significance: These flies are a source of great irritation. They transmit the non-pathogenic *Trypanosoma theileri*, anthrax and other bacterial infections to cattle.
Diagnosis: Identification of flies located under the tail and between the hindlegs.
Therapy: ☞ below, THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 141
Prophylaxis: Regular grooming may be helpful to reduce louse fly infestations.

- **Therapy and prophylaxis of ectoparasites** (for arachnids and insects)

A great number of insecticides is used to control both arachnids and insects (Table 11). Methods of application include whole-body sprays, dips, dusts and topical application to the dermis and ears. Dips are more effective for ticks. Thorough treatment of the infested area is required, especially when ticks infest the ears or underside of the tail or mites infest localized patches on the skin. Impregnated ear tags, containing pyrethroids or organophosphates, are of increasing importance in fly control. Elimination of parasites or stages in the environment is difficult because many ectoprasites are capable of surviving in the environment for prolonged periods (e.g. some ticks can live on the ground up to 300 days without feeding). In areas where many ticks exist reinfection of the host occurs continuously and treatment therefore must be repeated regularly. Some ticks are not strictly species-specific and ticks normally adapted to horses can occasionally also affect cattle or other animals and they must be treated, too, if attempts are made to reduce tick infestations.
In subhumid areas the period of highest tick activity is the wet season and only few ticks are found on animals during the dry season. Con-

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sequently tick control is focussed to the wet season. In tropical areas most ticks are active throughout the year and must be controlled continuously.

Table 11 Some common compounds to control external parasites of cattle

Compound	Pest
Organophosphates	
Chlorpyrifos	Lice, horn flies
Coumaphos	Horn flies, lice, ticks, grubs, screw worms, mites
Crotoxyphos and dichlorvos	Horn flies, face flies, stable flies, house flies,lice, ticks
Dichlorvos	Horn flies, face flies, stable flies, house flies
Dichlorvos and tetrachlorvinphos	Horn flies, face flies, lice, ticks
Diazinon	Horn flies (including pyrethroid-resistant flies), spinose ear ticks, lice, mites, *Hypobosca* spp.
Dioxathion	Horn flies, lice, mites
Fenthion	Horn flies, lice, *Hypoderma* spp.
Malathion	Lice, keds, mites
Phoxim	Mites, lice, flies, myiasis, ticks
Phosmet	Grubs, lice, horn flies, ticks, mites
Stirofos	Horn flies, face flies, house flies, stable flies
Tetrachlorvinphos	Horn flies, lice, ticks
Trichlorfon (metrifonat)	Grubs, lice, mites
Carbamates	
Carbaryl	Mites, lice, ticks, flies
Promacyl	Mites, lice, ticks, flies
Chlorinated Hydrocarbons*	
Lindane	Screwworm larvae, mites, lice, ticks, flies
Methoxychlor	Mites, lice, ticks, flies
Toxaphen	Mites, lice, ticks, flies
Pyrethrins and Pyrethroids	
Cyfluthrin	Horn flies, horse flies
Cypermethrin	Horn and face flies, ticks
Deltamethrin	Flies, including tsetse flies
Fenvalerate	Horn and face flies, spinose ear ticks
Flumethrin	Flies, including tsetse flies, ticks
Permethrin	Horn flies, face flies, mites, ticks, lice
Pyrethrin	Horn flies, face flies, mites, lice
Avermectins	
Doramectin	Mites, lice, ticks, *Hypoderma* spp.
Ivermectin	Mites, lice, *Hypoderma* spp.

* some are no longer approved by some governments

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5.2 Eyes

Thelazia rhodesi Cattle eyeworm

Location: Conjunctival sac
Hosts: Cattle, rarely sheep and goat
Species description: Milky-white worms, males 8–12 mm long and females 12–18 mm. The cuticle is transversely striated. The spicules are 0.75–0.85 and 0.115–0.13 mm long. Intermediate hosts are *Musca larvipara* and *Musca amica*. Infected flies transmit the infective larvae via saliva to calves. Adult *Thelazia* appeared 20–25 days after infection. In some regions a high percentage of calves is infected but lesions are often absent.
Geographic distribution: World-wide
Symptoms: Conjunctivitis, clouded cornea, marked lacrimation. The affected eyes may be swollen and covered with exsudate and pus. Without treatment progressive keratitis and ulceration of the cornea may occur.
Significance: Infections with eyeworms may be very prevalent in some regions. Heavy infections may cause marked irritation, keratitis and weight loss of affected animals.
Diagnosis: This is made by the detection of worms in the conjunctival sacs. Examination of the lacrimal secretions may reveal eggs or first-stage larvae.
Therapy: Levamisole (5 mg/kg, sc.) and ivermectin (200 µg/kg, sc.) are active against *Thelazia* spp. Concurrent use of antibiotic ointment for secondary invaders is recommended.
Prophylaxis: Fly control measures against the face fly aid in the control of thelaziosis in both cattle and horses.
(Figures 286, 287, 288)

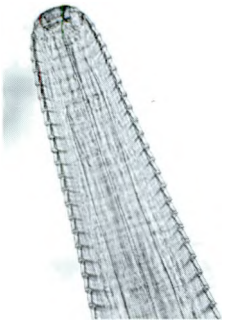

Fig. 286 *Thelazia rhodesi* (eyeworm); anterior end with typical cuticular striation

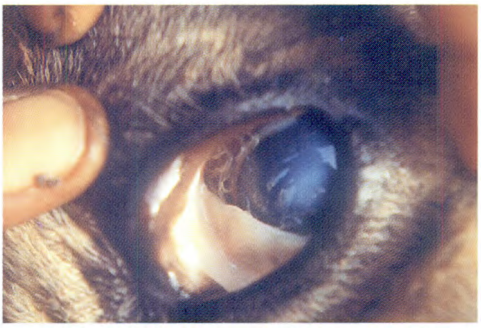

Fig. 287 *Thelazia rhodesi* in the eyes of a cattle

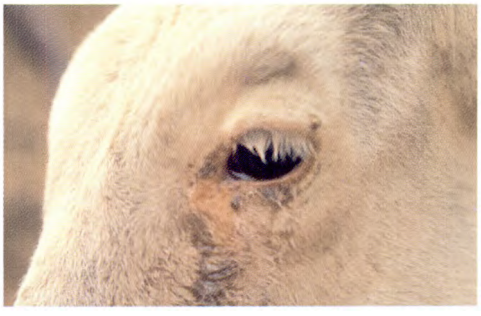

Fig. 288 N'Dama cattle with lacrimation due to *Thealzia rhodesi* infection

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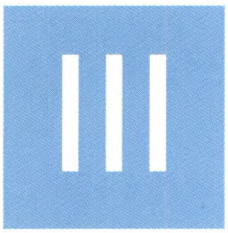

Parasites of Sheep and Goats

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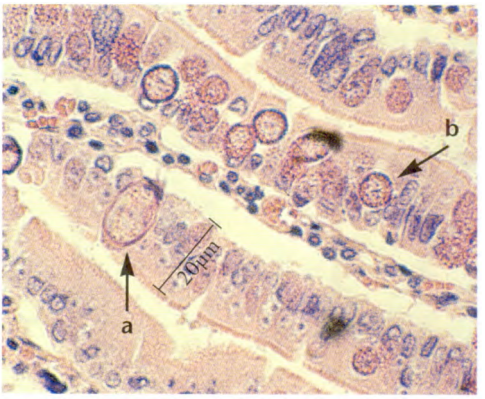

Fig. 289 *Eimeria* spp.; intestinal tissue section (sheep); heavy infection showing (a) oocysts (approx. 20 µm) and (b) gametocytes

PROTOZOA

- **Coccidiosis in sheep and goats**

Sheep and goats have their own species of Coccidia. Cross-transmission is not possible. Out of a spectrum of more than 10 species which occur world-wide in sheep and goats, generally only a few are highly pathogenic and only if predisposing factors increase the susceptibility of the host animal (stress at weaning, malnutrition, other diseases, etc.) or if the management conditions are such that animals are exposed to a very high infection risk (using highly contaminated feedlot confinements).

All the *Eimeria* spp. produce unsporulated oocysts which are passed in the faeces. Within 1–4 days sporulation occurs (oocysts with four sporocysts each containing two sporozoites). The sporulated oocyst represents the infective stage of *Eimeria* spp. The further development occurs in the epithelial cells and the cells of the lamina propria at species specific sites within the intestines. Oocysts occur 6–35 days after infection. Disease due to *Eimeria* spp. mainly occurs in young animals.
(Figures 289, 290)

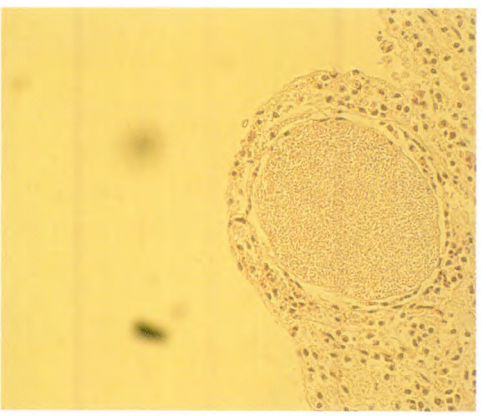

Fig. 290 *Eimeria* spp.; intestinal tissue section (sheep); macroschizont (100 µm in diameter)

Eimeria ovinoidalis (syn. *E. ninakohlyakimovae*), ***Eimeria ahsata, Eimeria bakuensis*** (syn. *E. ovina*) **and other *Eimeria* spp.**
Coccidiosis of sheep

Remarks: 15 species of *Eimeria* spp. have been described for sheep. Oocysts of similar appearance affect goats, but cross-transmission studies have confirmed their separate identity. *E. ovinoidalis* is the most pathogenic species. It affects the caecum and colon, causing severe enteritis, which may become haemorrhagic. Suckling lambs 4–7 weeks old are most commonly

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affected. *E. ahsata* is also highly pathogenic for sheep. The intestines of infected lambs are thickened and oedematous and the Peyer's patches and lower parts of the small intestine are inflated. Deaths may occur. Other *Eimeria* oocysts (e.g. *E. crandalis*) are often produced in large numbers by lambs without causing disease. *E. bakuensis* causes white patches on the mucosa and sometimes polyps but its pathogenic role is in debate.
(Figures 291, 292, 293)

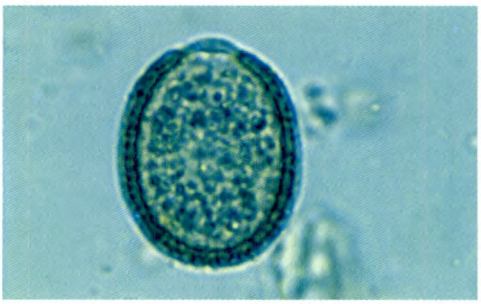
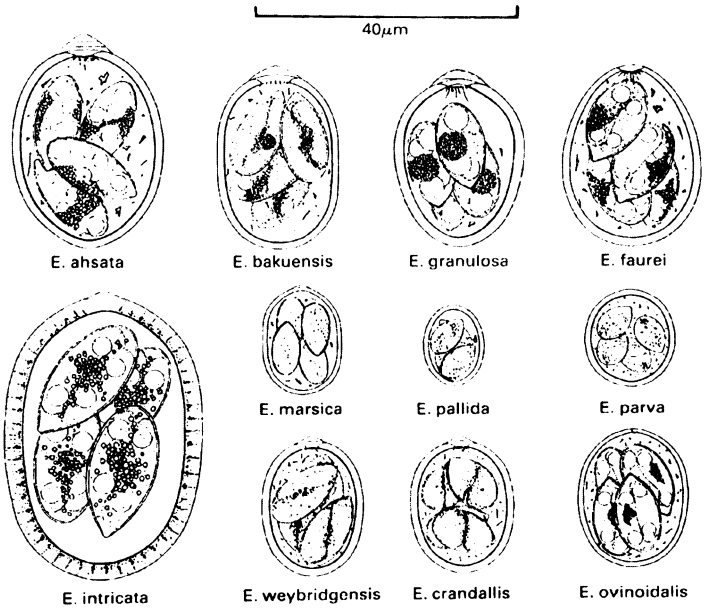


Fig. 291 Oocyst of *Eimeria intricata*



292 *Eimeria* species found in sheep (schematic) [1]

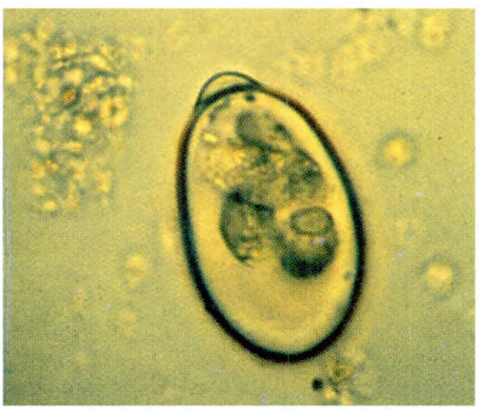


Fig. 293 Oocyst of Eimeria ahsata (39 × 25 μm) [15]

Eimeria arloingi, Eimeria christenseni, Eimeria ninakohlyakimovae and other *Eimeria* spp. Coccidiosis of goats

Remarks: Young goats commonly harbour a number of species of Coccidia and frequently produce oocysts at the rate of 10^5–10^6 per g of faeces without showing any clinical symptoms. Disease may occur following stress such as weaning, changes in diet, weather stress or regrouping, etc. *Eimeria arloingi, Eimeria christenseni* and *Eimeria ninakohlyakimovae* are the most pathogenic species in goats world-wide.
(Figures 294, 295)

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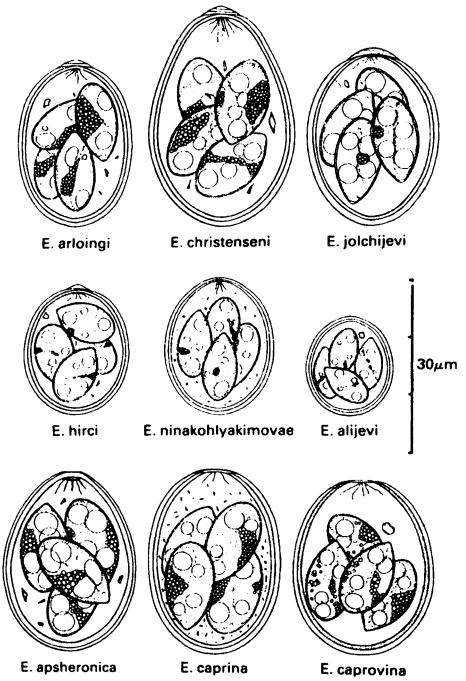

Fig. 294 *Eimeria* spp. found in goats [1]

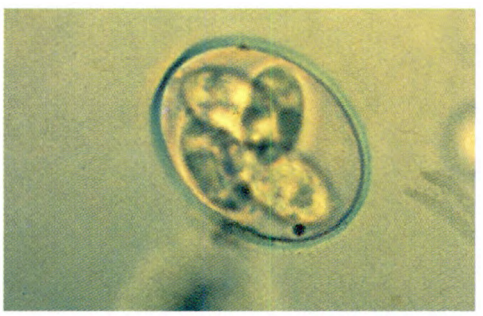

Fig. 295 Sporulated oocyst of *Eimeria ninakohlyakimovae* (24 × 19 µm) [15]

- **General features of coccidiosis in sheep and goats**

Symptoms: Diarrhoea (not always), dysentery, tenesm, exsiccosis; heavy infections cause fever, general depression, muscular spasms, CNS symptoms and finally death may occur. Secondary bacterial infections often aggravate the disease.

Significance: Coccidiosis may cause severe losses in small ruminants.

Diagnosis: This is difficult. Oocyst output can be very misleading because healthy lambs may produce more than 10^6 oocysts per g of faeces, whereas lambs dying of coccidiosis sometimes show very few oocysts. A satisfactory diagnosis can be made based on clinical history of the flock and autopsy lesions. Scrapings from the intestinal lesions usually reveal large numbers of gametocytes and oocysts. High oocyst counts of *E. ovinoidalis* from a number of scouring lambs support the diagnosis.

Therapy: Acute coccidiosis may be treated with trimethoprime/sulfonamide combinations. Lasalocid (15–70 mg/head/day, po.) may be used in sheep; amprolium can be used for clinical coccidiosis in sheep and goats; toltrazuril (15–20 mg/kg, po.).

Prophylaxis: Lasalocid (22–33 ppm, po.) and Monensin (1 mg/kg, po., daily during several days or 10–20 mg/kg feed) are highly effective against ovine coccidiosis. Amprolium (55 mg/kg, po. twice daily during 10 days) protects lambs against severe clinical disease. Sulfaguanidine (3 g/lamb daily or 0.1 mg/kg, po.), sulfaquinoxaline (2.5–4 g/kid daily or 8–70 mg/kg), sulfamerazine (65–130 mg/kg, po.) and nitrofurazon (70.4 mg/kg, po. daily for 7 days) control coccidiosis in lambs and goat kids. Decoquinate (0.5 kg/kg, po.) can be used in goats (☞ CATTLE, 1, Table 3, p.27).

Cryptosporidium parvum

Remarks: Diarrhoea in neonatal lambs and goat kids may occur due to *C. parvum* infections. Appropriate colostrum supply is of great importance to avoid cryptosporidial diarrhoea. (☞ CATTLE, 1).

Giardia duodenalis (syn. *G. caprae, G. ovis* and *G. quadrii*)

Remarks: This parasite occurs in sheep and goats and has been associated with diarrhoeic episodes in lambs and goat kids.

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Cysts measure 14 × 8 µm and may be demonstrated in faeces by the flotation or SAF method (☞ METHODS, 1.4 and 1.12). Vegetative forms (trophozoites) may be found in fresh faeces of diarrhoeic animals. (Figure 296)

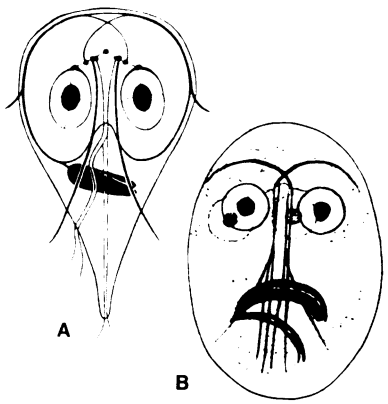

Fig. 296 (A) *Giardia* sp. trophozoite and (B) *Giardia bovis* cyst (16 × 10 µm) [14]

***Entamoeba* spp. (*E. ovis, E. dilimani* and *E. wenyoni*)**

Remarks: Several *Entamoeba* species are common in the intestine of sheep, goats and other ruminants. Little is known about their significance. They seem to be of low pathogenicity.

HELMINTHS

- **Trematoda eggs found in the faeces and adult trematodes living in the gastrointestinal tract**

Rumen flukes belong to the families of Paramphistomatidae and Gastrothylacidae each of which containing several species (☞ CATTLE, 1). *Bothriophoron bothriophoron, Calicophoron microbothrium, Calicophoron daubneyi, Cotylophoron cotylophorum, Carmyerius spatiosus* and *Carmyerius papillatus* are the most frequent rumen flukes in small ruminants in Africa. Adult flukes do not cause clinical disease although they are commonly found in the rumen and sometimes in large numbers. The immature worms attach to the duodenal mucosa by means of their large posterior sucker and may cause severe enteritis, necrosis and haemorrhage. Affected animals exhibit anorexia, polydipsia, unthriftiness and severe diarrhoea. Extensive mortality may occur, especially in lambs. For diagnosis and control ☞ CATTLE, 1.

Table 12 Recommended drugs against *Paramphistomum* spp. infections in sheep

Drug	Dosage (mg/kg) sheep	Immature flukes	Mature flukes
Niclosamide	90	+	
Rafoxanide	15	+	
Resorantel	65	+	+
Oxyclozanide	17	+	+

***F. gigantica* Tropical large liver fluke**
(☞ CATTLE, 1 and ■ 4.2)

***F. hepatica* Large liver fluke of temperate areas and high altitude regions in East Africa**

Location: Adult flukes in biliary ducts; eggs in the faeces (☞ CATTLE, 1 and ■ 4.2)

***Dicrocoelium hospes* African small liver fluke and *Dicrocoelium dendriticum* Small liver fluke**

Location: Adult flukes in biliary ducts; eggs in the faeces (☞ CATTLE, 1 and ■ 4.2)

***Eurytrema pancreaticum* Pancreas fluke**

Location: Adult flukes in pancreatic ducts; eggs in the faeces (☞ CATTLE, 1 and SHEEP AND GOATS, ■ 4.5)

***Schistosoma* spp. (*S. bovis, S. mattheei* and *S. curassoni*) Blood flukes**

Location: Adult flukes in mesenteric veins; eggs in the intestinal wall and faeces (☞ CATTLE, 1 and SHEEP AND GOATS, ■ 2).

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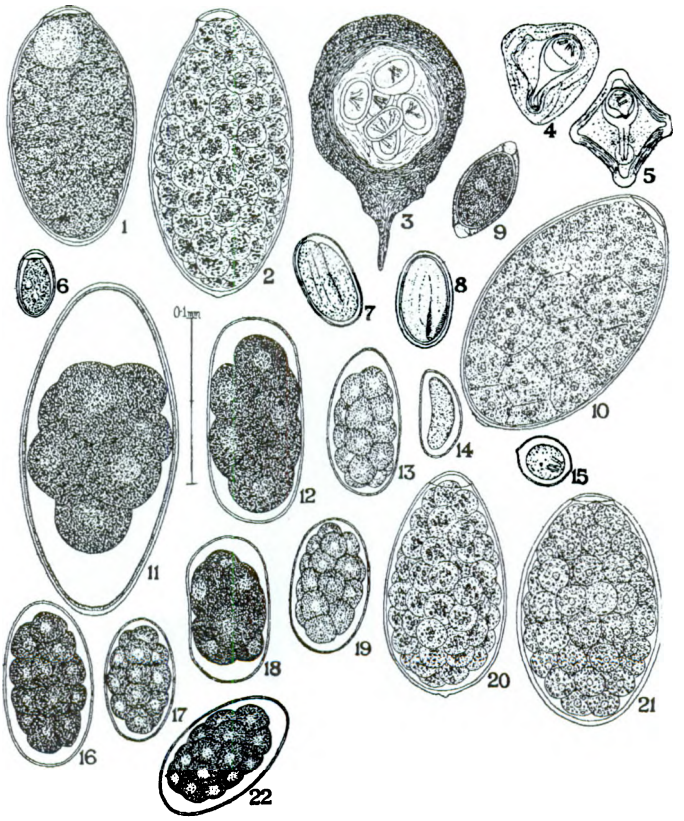

Fig. 297 Helminth eggs of sheep (schematic, not drawn to scale), (1) *Fasciola hepatica*, (2) *Paramphistomum cervi*, (3) *Thysaniezia ovilla*, (4) *Moniezia expansa*, (5) *M. benedeni*, (6) *Dicrocoelium dendriticum*, (7) *Strongyloides papillosus*, (8) *Gongylonema pulchrum*, (9) *Trichuris globulosa*, (10) *Fasciola gigantica*, (11) *Nematodirus spathiger*, (12) *Gaigeria pachyscelis*, (13) *Trichostrongylus* sp.*, (14) *Skrjabinema ovis*, (15) *Avitellina centripunctata*, (16) *Chabertia ovina**, (17) *Haemonchus contortus**, (18) *Bunostomum trigonocephalum**, (19) *Oesophagostomum columbianum**, (20) *Cotylophoron cotylophorum*, (21) *Fascioloides magna* and (22) *Ostertagia circumcincta**; * Strongyle-type eggs are difficult to differentiate in routine examinations [3]

- **Cestoda eggs found in the faeces and adult cestodes living in the gastrointestinal tract**

Moniezia expansa and *Moniezia benedeni*

Common tapeworms

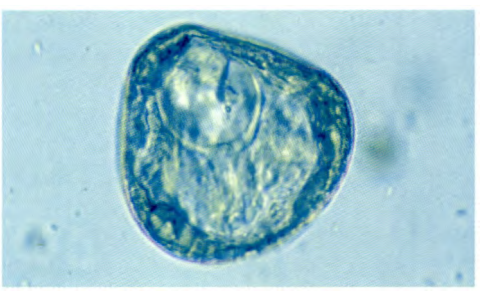

Fig. 298 Egg of *Moniezia* sp. (60–80 µm in diameter)

Remarks: *Moniezia* spp. infections in sheep are prevalent but are of relatively low pathogenicity. Heavy infections may result in unthriftiness and gastrointestinal disturbances. *Moniezia* spp. are easily seen at necropsy because of their size, but often the much smaller and much more pathogenic trichostrongylids (e.g. *Trichostrongylus colubriformis*) can not be seen with the naked eye. Therefore, many earlier observations which associated tapeworm infections with diarrhoea, emaciation and weight loss

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did not accurately consider the underlying infections with nematodes (☞ CATTLE, 1). For diagnosis, therapy and prophylaxis ☞ CATTLE, 1.
(Figures 298, 299, 300)

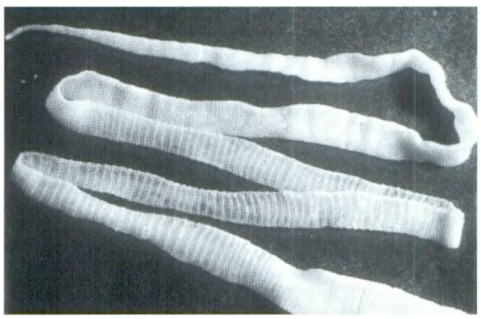

Fig. 299 *Moniezia* sp.; tapeworm with pronounced segments [32]

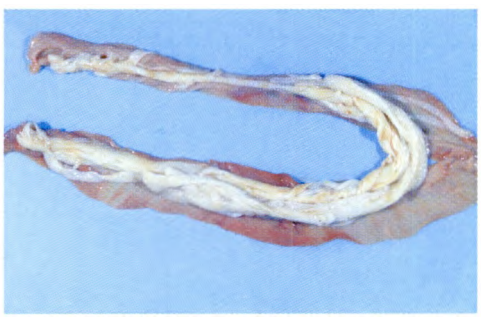

Fig. 300 *Moniezia* sp. found in the small intestine of a sheep [11]

Avitellina centripunctata

Remarks: This is a very common tapeworm of small ruminants and cattle in semi-arid and arid areas of Africa and Asia. Lambs acquire infections but many months elapse before the worms are adult. Adults are common in yearlings and older sheep. Lambs and kids may carry heavy burdens but often no obvious clinical signs may be found. However, clinical observations of pot-bellied suckling animals which improve after treatment are frequently reported. For diagnosis and control ☞ CATTLE, 1.
(Figure 301)

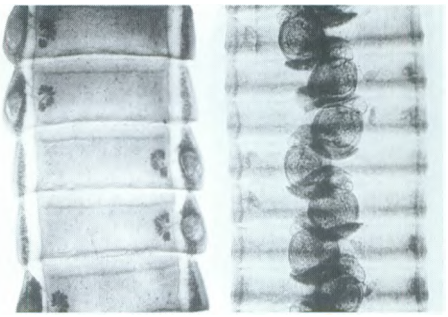

Fig. 301 Stained segments of *Thysaniezia ovilla* (left) and *Avitellina centripunctata* (right) [4]

Thysaniezia ovilla (syn. *T. giardi*)

Remarks: This species is frequently encountered in sheep and goats in many parts of Africa, especially in those with low rainfall. It occurs in the small intestine and is not very pathogenic but heavy infections occasionally produce clinical signs. For diagnosis and control ☞ CATTLE, 1.

Stilesia globipunctata

Location: Small intestine, duodenum and upper jejunum (☞ CATTLE, 1)
Hosts: Sheep, goat, dromedary, rarely cattle and other ruminants
Species description: Adults are 45–60 cm long and up to 2.5 mm wide. The scolex of the adult worm penetrates the mucosa of the duodenum and jejunum and causes a proliferative inflammation. The scolex is embedded in the mucosa while the posterior proglottids are free in the lumen. Heavy infections can cause death.
Geographic distribution: Arid areas of Africa

Stilesia hepatica

Remarks: This parasite occurs in the bile ducts (☞ CATTLE, 1 and SHEEP AND GOATS, ■ 4.2) and occasionally small intestine of sheep and goats in tropical and southern Africa. It is non-pathogenic but extremely prevalent (in 90–100% of sheep) in many parts of Africa. The condemnation of a large

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proportion of sheep livers at meat inspection is the major loss due to this parasite. The ovoid eggs measure 16–19 × 26 µm. These eggs or the proglottids may appear in the faeces of infected animals.

- **Preventive measures to control intestinal tapeworms**

***(Moniezia* spp., *Avitellina* spp., *Thysaniezia* spp. and *Stilesia* spp.) of ruminants**

Therapy: Niclosamid (45–75 mg/kg, po.) and praziquantel (3.75 mg/kg, po., sheep only) are specific cestocidal drugs. In addition the following benzimidazoles are effective against tapeworms: albendazole (3.8–5mg/kg, po.), fenbendazole (10 mg/kg, po.), oxfendazole (5 mg/kg, po.), mebendazole (10–20 mg/kg, po.), netobimin (20 mg/kg, po.), febantel (5–15 mg/kg, po.). Special attention should be given to the control of tapeworms in lambs to avoid losses in heavily infected populations (☞ CATTLE, 1).

- **Nematoda eggs found in the faeces, adult nematodes living in the gastrointestinal tract and first-stage larvae of lungworms**

Gongylonema pulchrum Gullet worm or zigzag worm

Remarks: This parasite occurs in the mucosae of the oesophagus and forestomachs of sheep, goats, less frequently cattle (☞ CATTLE, 1).

G. verrucosum Rumen gullet worm

Remarks: This is an apathogenic nematode, found in the mucosa of the forestomachs. This parasite may occasionally be found at necropsy (☞ CATTLE, 1).

Parabronema skrjabini

Remarks: This spirurid worm occurs occasionally in the abomasum of small ruminants. The life cycle includes *Stomoxys* spp. and *Lyperosia* spp. as intermediate hosts (☞ CATTLE, 1). For therapy and prophylaxis ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

Haemonchus contortus Large stomach worm, "twisted wire worm", "barber's pole worm"

Location: Abomasum
Hosts: Cattle, sheep, goat and other ruminants
Species description: *H. contortus* is one of the most prevalent and most pathogenic parasites of ruminants, particularly sheep (life cycle and morphology ☞ CATTLE, 1). It can rapidly kill young sheep. When high numbers of infective larvae are ingested at once, deaths can occur suddenly while the sheep still appear to be in good health. This is termed "acute prepatent disease" when eggs are not yet found in the faeces but high numbers of preadult *Haemonchus* specimens have started to suck blood. In this case the host was killed before the parasites were mature. Haemonchosis is always associated with anaemia as both fourth-stage larvae and adult worms suck blood. Chronic infections which result from continuous ingestion of third-stage larvae may produce oedema ("bottle jaw"), iron-deficiency anaemia, progressive wool breaks and death. *H. contortus* usually occurs together with other gastrointestinal nematodes. Sometimes, sheep infected with *H. contortus* may exhibit self-cure which is a sudden loss of the worm load. This phenomenon is rarely seen in animals kept under poor nutritional and hygienic conditions. Ingested larvae may arrest their development at the end of the rainy season (or summer) and survive the dry season (or winter) as inhibited larvae within their host. This phenomenon is also termed hypobiosis. This is an adaptation of the parasite to adverse environmental conditions, when the preparasitic development from the egg to the third-stage larva is impossible.

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Geographic distribution: World-wide, especially in warm areas

Symptoms: Anaemia, oedema ("bottle jaw"), rough coat, weakness, weight loss or retarded growth

Significance: *H. contortus* is a common parasite of sheep that may kill lambs rapidly. Chronic infections cause weakness, anaemia, making sheep raising uneconomical.

Diagnosis: Eggs of strongyle-type appear in faeces. However, in prepatent diseases, clinical signs (anaemia) and death occur before the worms are mature. Species identification only by identification of adult worms at necropsy.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figures 302, 303, 304, 305)

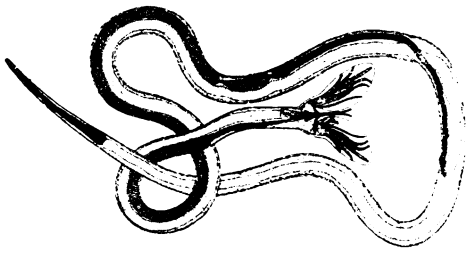

Fig. 302 *Haemonchus contortus*; male (10–20 mm × 400 µm) [9]

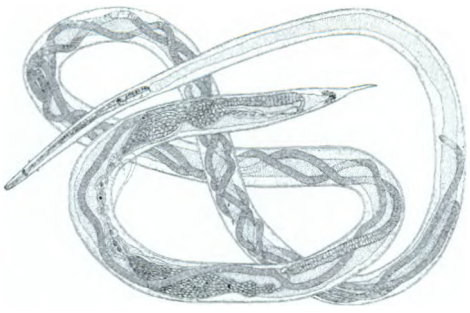

Fig. 303 *Haemonchus contortus*; female (18–30 mm × 500 µm) [9]

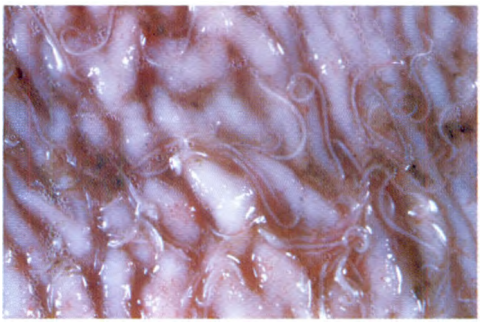

Fig. 304 *Haemonchus contortus*; adult worms on the abomasal mucosa

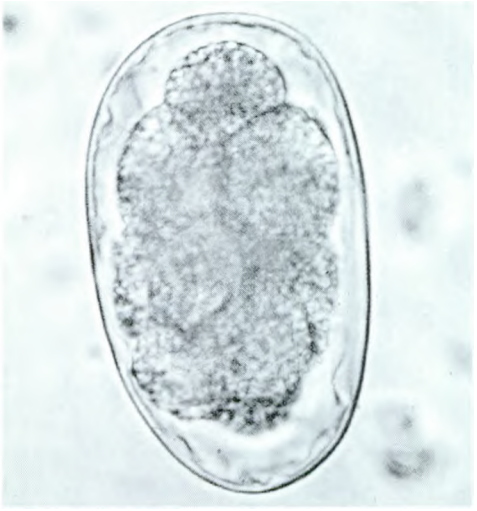

Fig. 305 Strongyle-type egg (74 × 44 µm) [11]

Mecistocirrus digitatus

Remarks: This is mainly a parasite of cattle and occurs occasionally in the abomasum of goats and very rarely of sheep (☞ CATTLE, 1). For therapy and prophylaxis ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

Trichostrongylus axei Stomach hair worm

Remarks: This trichostrongylid species occurs world-wide in the abomasum of sheep and goats. It is often found in mixed infections with other gastrointestinal nematodes (e.g.

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Ostertagia spp., or *Haemonchus contortus*). Adult worms penetrate the lining of the abomasum, causing irritation and inflammation. Wart-like swellings may occur and cause diarrhoea, weight loss and reduce appetite (☞ CATTLE, 1). For therapy and prophylaxis ☞ CATTLE, 1, p. 41; THERAPY OF NEMATODE INFECTIONS

***Trichostrongylus* spp. (*T. colubriformis, T. probolurus, T. vitrinus, T. capricola* and other *Trichostrongylus* species)** Small intestinal hair worm, bankrupt worm, black scours worm

Location: Small intestine

Hosts: Sheep and goat

Species description: These nematodes are small (up to 5.5 mm long), reddish-brown and without a specially developed head-end. There is no buccal capsule. A typical structure for all *Trichostrongylus* spp. is the excretory pore, situated near the anterior extremity. Males have bursae with large lateral lobes and the morphology of the spicules and the gubernacula is species-specific. Typical direct nematode life cycle. Infection occurs by ingestion of third-stage larvae. The prepatent period is 20–25 days.

Geographic distribution: World-wide

Symptoms: Weakness, progressive weight loss, depressed appetite, digestive troubles such as diarrhoea or constipation

Significance: *Trichostrongylus* species commonly occur with other gastrointestinal nematodes. Therefore losses due to *Trichostrongylus* spp. are not easy to measure. and their effects are additive. If coccidiosis accompanies trichostrongylosis, enteritis may occur.

Diagnosis: Strongyle-type eggs appear in the faeces. Adult worms may be found at necropsy.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figure 306, 307)

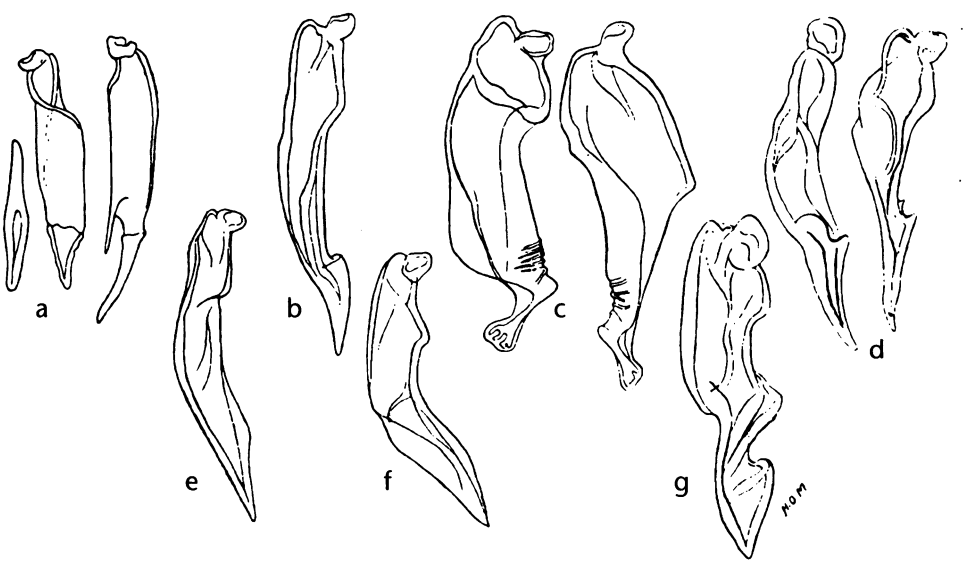

Fig. 306 Morphology and length of spicules of some *Trichostrongylus* species: (a) *T. axei* (85–95 and 110–150 µm), (b) *T. colubriformis* (135–156 µm), (c) *T. rugatus* (137–145 and 141–152 µm), (d) *T. falculatus* (100 µm), (e) *T. vitrinus* (160–170 µm), (f) *T. capricola* (130–145 µm) and (g) *T. probolurus* (126–134 µm) [3]

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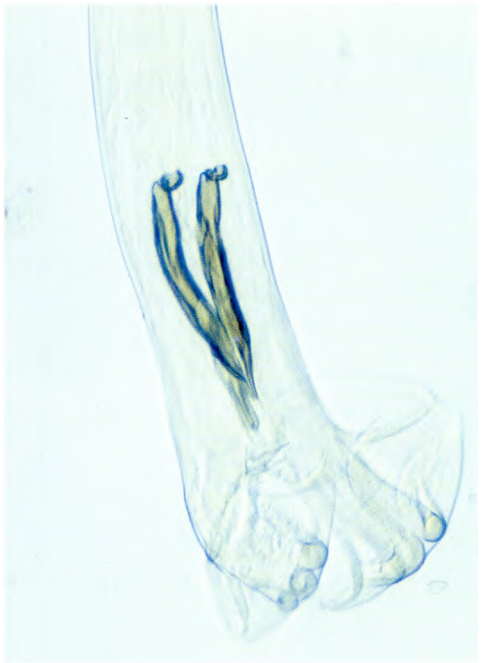

Fig. 307 *Trichostrongylus vitrinus*; bursa copulatrix and spicules (160–170 µm)

Ostertagia (syn. *Teladorsagia*) ***circumcincta,*** ***Ostertagia*** (syn. *Teladorsagia*) ***trifurcata,*** ***Ostertagia pinnata*** **and other** ***Ostertagia*** **spp.** Brown stomach worms

Location: Abomasum and rarely in the small intestine

Hosts: Sheep and goat

Species description: These species are brownish, thread-like nematodes up to 12 mm in length. Males are smaller with straight, slender spicules. All members of the genus *Ostertagia* have a small cephalic vesicle, cervical papillae and longitudinal cuticular ridges. The morphology of the bursa copulatrix and the spicules varies among the particular species. Typical direct nematode life cycle; infection is acquired by ingestion of infective third-stage larvae which may develop to adults within 17 days; arrested development (hypobiosis) occurs.

Geographic distribution: Widespread in moist, temperate regions; autochthonous infections are focal in Africa but *Ostertagia* spp. infections have repeatedly been reported from imported sheep and goats.

Symptoms: Profuse watery diarrhoea, progressive weight loss associated with hypoproteinaemia; death may occur.

Significance: These nematodes cause serious losses world-wide by producing weight loss and decreased wool production.

Diagnosis: Strongyle-type eggs appear in the faeces.

Therapy and Prophylaxis: ☞ CATTLE, 1, p. 42; THERAPY OF NEMATODE INFECTIONS

Pseudomarshallagia elongata

Remarks: This species was found in the abomasa of sheep and goats in Ethiopia. It is similar to *Ostertagia* spp. For therapy and prophylaxis ☞ CATTLE, 1 THERAPY OF NEMATODE INFECTIONS

Cooperia curticei, Cooperia pectinata, ***Cooperia punctata*** **and other** ***Cooperia*** **spp.**

Location: Small intestine

Hosts: Sheep and goat

Species description: GENERAL FEATURES OF COOPERIA spp. ☞ CATTLE, 1; *Cooperia* spp. have a direct life cycle; prepatent period is 15–20 days.

Geographic distribution: World-wide

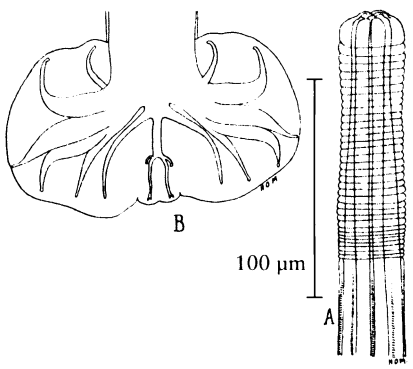

Fig. 308 *Cooperia* sp.; anterior end with enlarged cephalic vesicle (A) and bursa copulatrix (B) [3]

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Symptoms: Diarrhoea, weight loss and decreased wool growth

Significance: The effects of *Cooperia* spp. are additive to those of other gastrointestinal nematodes. Diarrhoea in lambs caused by heavy *Cooperia* infections may be fatal due to dehydration.

Diagnosis: Strongyle-type eggs appear in the faeces. Adult worms may be found at necropsy.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figure 308)

Paracooperia daubneyi

Location: Small intestine

Hosts: Sheep only

Species description: This genus resembles *Cooperia* spp. but differs in that the spicules are divided distally into three processes: one is serrate, one is winged and large and the third is slender and difficult to see. The gubernaculum is absent.

Geographic distribution: East Africa, Central Africa

Symptoms, Significance and Diagnosis: ☞ above *Cooperia* spp.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

Nematodirus spathiger, Nematodirus filicollis, Nematodirus battus and other *Nematodirus* spp. Thread-necked strongyles

Location: Small intestines

Hosts: Sheep and goat

Species description: General features of this genus ☞ CATTLE, 1. The life cycle is direct and typical for nematodes. Infection is acquired by ingestion of third-stage larvae, free-living or in eggs. Larval stages are the primary cause of disease. They damage the lining of the small intestine. Erosion of the intestinal mucosa occurs and pieces of it may become loose and be shed in the faeces.

Geographic distribution: *N. spathiger* and *N. filicollis* occur world-wide including East and Central Africa; *N. helvetianus* occurs in Europa, USA and in (Africa).

Symptoms: Loss of appetite, diarrhoea and weight loss. Wool growth is reduced in chronic infections.

Significance: *Nematodirus* infections may cause significant mortality in lambs, when present in high numbers.

Diagnosis: Characteristic large strongyle-type eggs appear in the faeces.

Cave: the eggs of the small ruminant hookworm *Gaigeria pachyscelis* are similar to those of *Nematodirus* spp.!

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figures 309, 310, 311)

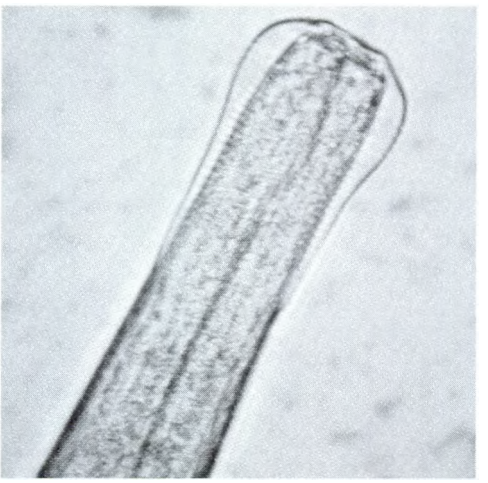

Fig. 309 *Nematodirus* sp.; anterior end with cephalic vesicle [32]

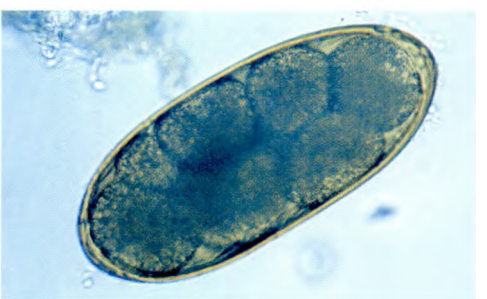

Fig. 310 Egg of *Nematodirus battus* (152–182 × 67–77 µm)

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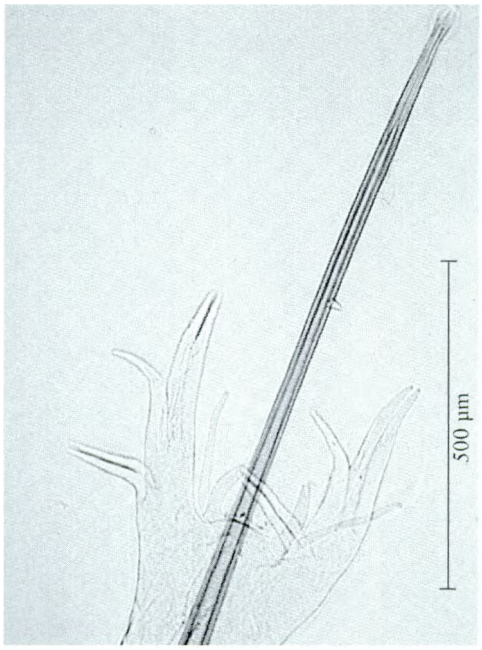

Fig. 311 *Nematodirus* sp.; bursa copulatrix with long spicula [32]

Impalaia tuberculata

Location: Abomasum and small intestine
Hosts: Dromedary, rarely sheep and cattle
Species description: These parasites belong to the Nematodirinae.
Geographic distribution: Central, eastern and southern Africa
Symptoms, Significance and Diagnosis: ☞ *Nematodirus* spp.
Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS; ☞ DROMEDARIES, 1.

Impalaia nudicollis

Location: Small intestine
Hosts: Sheep
Species description: The male is 5–9 mm long. The female is 11–13 mm long.
Geographic distribution: East Africa
Symptoms, Significance and Diagnosis: ☞ above, *Nematodirus* spp.
Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS (Figure 312)

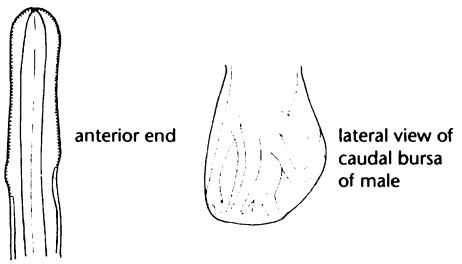

Fig. 312 *Impalaia nudicollis* (schematic) [16]

First-stage larvae of the following lungworms may be found in the faeces of sheep and goats:

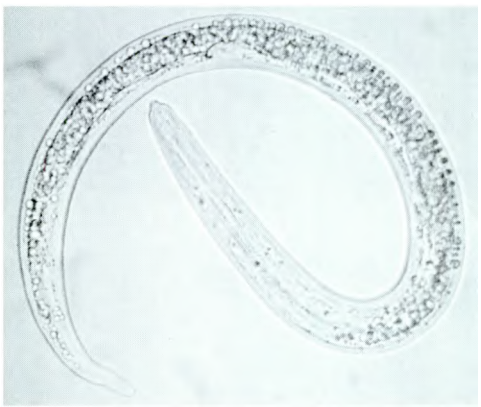

Fig. 313 *Dictyocaulus filaria*; first-stage larva (550–580 µm) [11]

DICTYOCAULIDAE Large lungworm

Dictyocaulus filaria Common lungworm
(☞ SHEEP AND GOATS, ■ 4.3; Fig. 313)

PROTOSTRONGYLIDAE Small lungworms

Protostrongylus rufescens Red lungworm
(☞ SHEEP AND GOATS, ■ 4.3)

Muellerius capillaris Hair lungworm
(☞ SHEEP AND GOATS, ■ 4.3)

Neostrongylus linearis
(☞ SHEEP AND GOATS, ■ 4.3)

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Cystocaulus ocreatus

(☞ SHEEP AND GOATS, ■ 4.3)
(Figures 314, 315, 316)

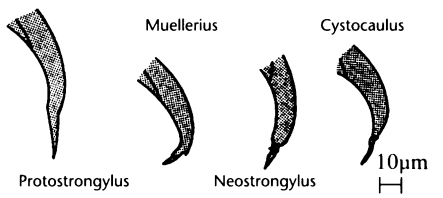

Fig. 314 The morphology of the tip (posterior end) of the lungworm larvae is relevant for the diagnosis [33]

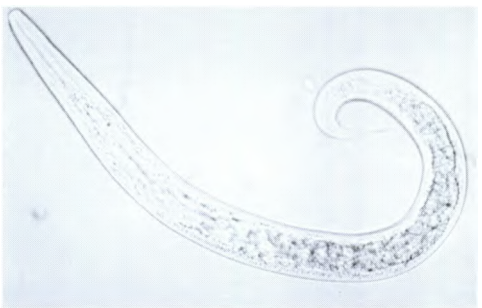

Fig. 315 *Protostrongylus rufescens*; first-stage larva (320–400 µm) [11]

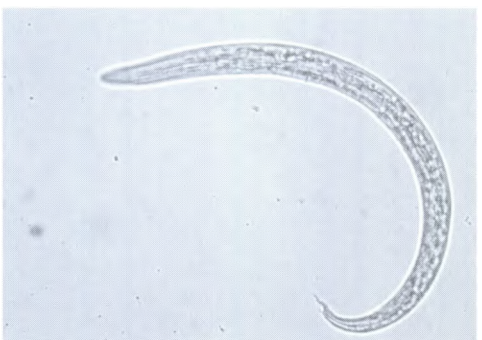

Fig. 316 *Muellerius capillaris*; first-stage larva (300–320 µm) [15]

Oesophagostomum columbianum

Nodular worm

Location: Colon
Hosts: Sheep, goat, camel and antelope
Species description: The male is 12–16.5 mm long and the female 15–21.5 mm and about 0.45 mm wide. The large cervical alae produce a marked dorsal curvature of the anterior part of the body. The cuticle forms a mouth collar which is separated from the rest of the body by a constriction. The anterior extremities of the cervical alae are pierced by marked cervical papillae. The male bursa contains two equal spicules, 0.77–0.86 mm long. The tail of the female is 500–600 µm long and ends in a fine point and the vulva is situated about 0.8 mm anterior to the anus. The eggs measure 73–89 × 34–45 µm. The life cycle is direct. After ingestion of the third-stage larvae they burrow into the wall of the gut anywhere between the small intestine and the rectum. Here they molt to the fourth-stage before leaving the gut wall and migrating to the large intestine, where they undergo their final molt and develop to the adult stage. Sometimes a number of larvae may remain in the mucosa for extended times. The prepatent period is about 41 days. *O. columbianum* larvae cause extensive nodule formation in the gut wall which impairs the ability of the intestine to absorb fluid and nutrients. Diarrhoea and weight loss may occur. Diarrhoea usually starts on the 6th day after a severe infection and coincides with the larvae leaving the nodules.
Geographic distribution: World-wide, particularly common in tropical and subtropical areas
Symptoms: In lambs the first sign is a persistent diarrhoea, which results in exhaustion and death unless the animals are treated. The faeces contain much mucus and sometimes blood. In chronic infections the animals show progressive emaciation, general weakness, and the skin becomes dry and the wool is unthrifty. The chronic oesophagostomosis in sheep is characterized by extreme emaciation, cachexia ending in complete prostration and death.
Significance: *O. columbianum* is a serious pathogen of sheep. 200–300 adult worms constitute a severe infection for lambs. Extensive nodule formation in the small and large intestine seriously affects the

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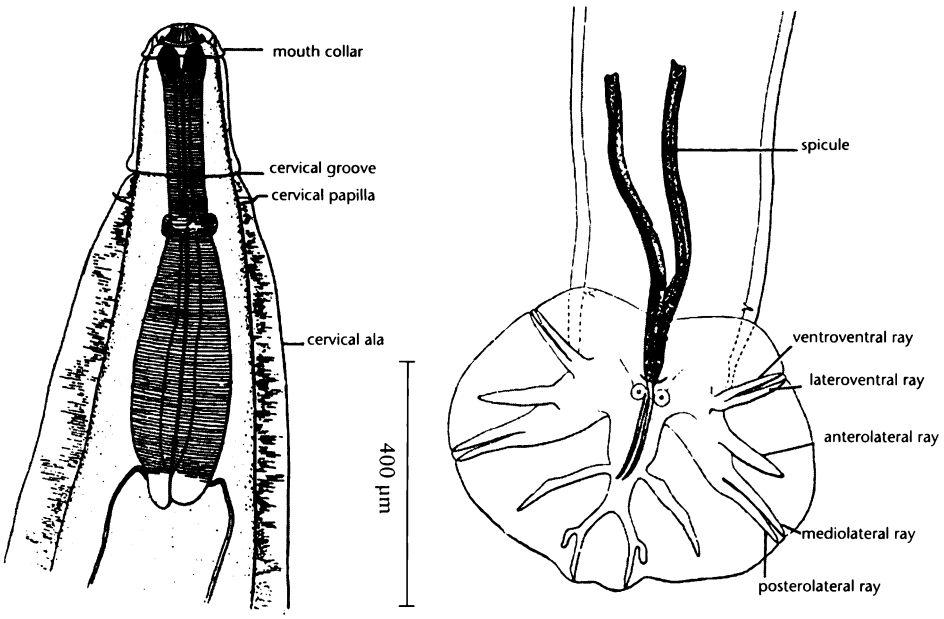

Fig. 317 *Oesophagostomum columbianum*; anterior end (left) and bursa copulatrix (right) [5]

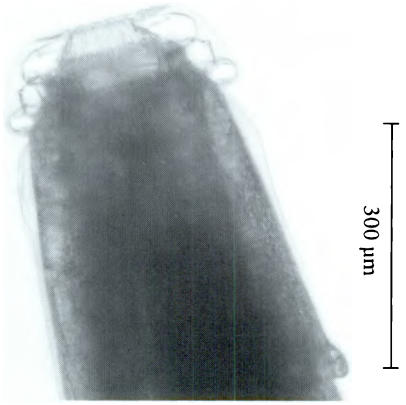

Fig. 318 *Oesophagostomum columbianum*; anterior end [8]

absorption, bowel movement and digestion. The nodules may rupture to the peritoneal surface causing peritonitis and peritoneal adhesions.

Diagnosis: Examination of diarrhoeic faeces may show fourth-stage larvae in acute cases or strongyle-type eggs in more chronic infections. Chronic infections without adult parasites are difficult to recognize and autopsy of a selected animal may be indicated.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figures 317, 318)

Oesophagostomum venulosum

Nodular worm

Remarks: This occurs in the colon of sheep, goat, deer and camel. The male is 11–16 mm and the female 13–24 mm long. There are no lateral cervical alae and the cervical papillae are behind the oesophagus. The spicules of the male are 1.1–1.5 mm long. The life cycle is similar to that of *O. columbianum* but the pathogenic effect is different. *O. venulosum* is relatively non-pathogenic and seldom causes nodules. Even in heavy infections the parasite does not cause significant clinical signs. Fog diagnosis, therapy and prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figures 319, 320)

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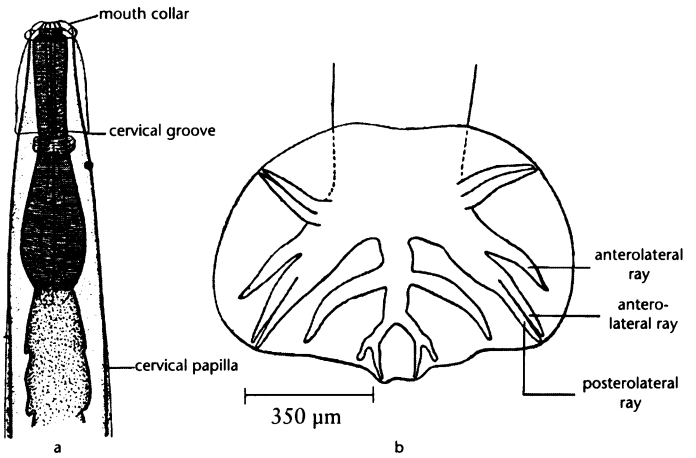

Fig. 319 *Oesophagostomum venulosum*; anterior end (a) and bursa copulatrix (b) [5]

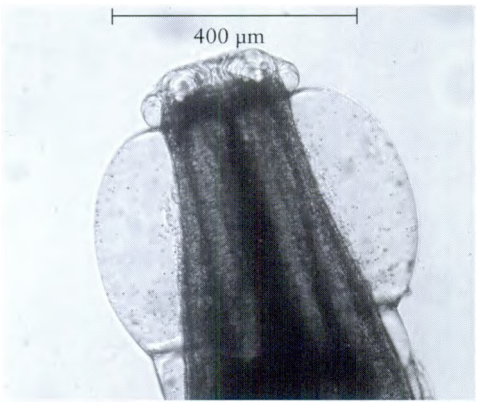

Fig. 320 *Oesophagostomum venulosum*; anterior end

Oesophagostomum multifoliatum

Remarks: This is a large-intestinal parasite of sheep and goats in eastern Africa. It has been reported from Nigeria, Kenya, Tanzania and Zimbabwe. The males are 12–14 mm and the females 14–17 mm long. This species differs from *O. columbianum* in that the cervical papillae are located just anterior to the end of the oesophagus and that the tail of the female is only 250–300 µm long. The eggs are large (150–170 × 68–80 µm) and similar to those of *Nematodirus* spp. No information is available on the life cycle and pathogenic effect of this species.

Chabertia ovina

Large-mouthed bowel worm

Location: Colon

Hosts: Sheep and goat

Species description: This nematode belongs to the Strongylidae and closely related to the horse strongyles. The males are 13–14 mm long and females are 17–20 mm long. The head end is characterized by a large buccal capsule which makes the identification of this parasite easy. The spicules are 1.3–1.7 mm long and a gubernaculum is present. The strongyle-type eggs measure 90–105 × 50–55 µm. The life cycle is direct. The ingested third-stage larvae undergo an extensive histiotropic phase in the wall of the small intestine prior to the third moult. 26 days after infection the larvae 4 may reach the colon. Fourth-stage larvae develop mainly in the lumen of the caecum and after the 4th moult immature adults then pass to the colon where patency occurs about 50 days after infection. The adult worms attach to the mucosa of the colon by means of their buccal capsule and then draw in a plug of mucosa. The worms suck blood only accidentally when a blood vessel is ruptured. The mucosa is inflamed, congested and swollen and covered with mucus in severe cases.

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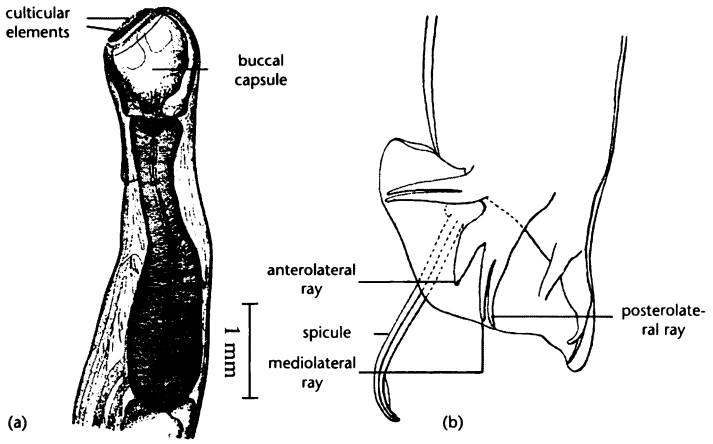

Fig. 321 *Chabertia ovina*; anterior end (a) and bursa copulatrix (b) [5]

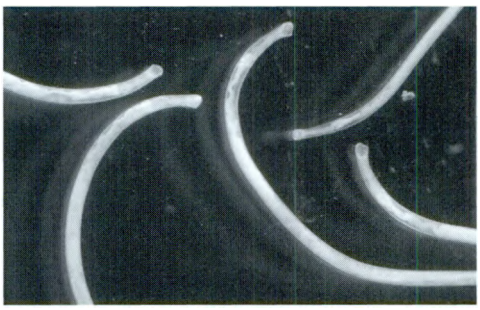

Fig. 322 *Chabertia ovina*; adults (13–20 mm long)

Geographic distribution: World-wide

Symptoms: Marked diarrhoea with blood and mucus may be seen in severely affected animals. Sheep lose condition, become anaemic and die following heavy infections.

Significance: *Chabertia* is significant only in heavily infected animals. Weight loss and reduced wool growth are the major results.

Diagnosis: Strongyle-type eggs appear in the faeces. Diagnosis can be confirmed either by identification of third-stage larvae following faecal culture or by demonstrating the adult worms in the colon at necropsy.

Therapy: Most of the benzimidazoles commonly used to treat gastrointestinal nematodes of ruminants are effective against *Chabertia ovina* (☞ CATTLE, 1 THERAPY OF NEMATODE INFECTIONS)

Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figures 321, 322, 323, 324)

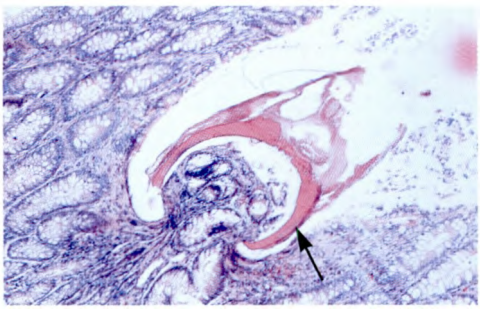

Fig. 323 *Chabertia ovina*; buccal capsule attached to the intestinal mucosa

Fig. 324 *Chabertia ovina* attached to the colon of a lamb

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Bunostomum trigonocephalum
Small ruminant hookworm

Location: Small intestine
Hosts: Sheep and goat
Species description: The male worms are 12–17 mm and the females are 19–26 mm long. The large buccal capsule is bent in a dorsal direction and bears at its ventral margin a pair of chitinous plates. At the base there is a pair of subventral lancets. There are no dorsal teeth in the buccal capsule. The spicules are slender and about 0.6 mm long. The eggs are 92 × 50 µm. Both the spicules and the eggs are much shorter than those of *Gaigeria pachyscelis*; direct life cycle; infection percutaneously or per os. Larvae migrate through the lungs and via the trachea to the oesophagus where they are swallowed to develop further in the small intestine. Prepatent period is 30–60 days. This parasite occurs in warm moist areas with a rainfall of more than 500 mm per year. Percutaneous infection is common. 4000 infective larvae can kill sheep without any previous clinical signs. At necropsy multiple haemorrhage may be found in the lungs. Adult worms cause anaemia and hypoalbuminaemia with oedema ("bottle-jaw"). 200–300 adult worms are fatal. In the small intestinal mucosa ulcers and erosions may be found which bleed after the worms have moved to another site of attachment. Usually the infection occurs together with other gastrointestinal nematodes, so the effect is additive.
Geographic distribution: World-wide
Symptoms: Anaemia, bottle jaw, weight loss. Chronic infections with only a few worms cause unthriftiness and thus great indirect losses within infected herds.
Significance: This is a prevalent and very pathogenic parasite of sheep in humid and moist areas. Heavy burdens are also found in older animals. Kraaling facilitates the spread of this parasite.
Diagnosis: This may be suspected from the clinical signs and the presence of strongyle-type eggs but the infection must be differentiated from other nematode infections that cause anaemia. However, only the demonstration of the adult parasites at necropsy is conclusive.
Therapy and Prophylaxis: ☞ CATTLE, § 1; THERAPY OF NEMATODE INFECTIONS
(Figure 325)

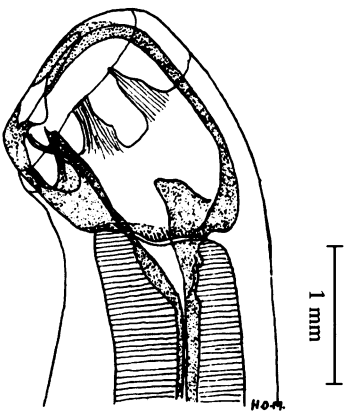

Fig. 325 *Bunostomum trigonocephalum*; anterior end with buccal capsule containing a pair of subventral lancets [3]

Gaigeria pachyscelis Hookworm

Location: Small intestine
Hosts: Sheep and goat
Species description: This parasite resembles in general *Bunostomum trigonocephalum*. Males are up to 20 mm long and females up to 30 mm. The buccal capsule contains a large dorsal cone, but no dorsal tooth and a pair of subventral lancets which have several cusps each. The male bursa has small lateral lobes joined together ventrally and a voluminous dorsal lobe. The spicules are slender with recurved unbarbed ends. They are 1.25–1.33 mm long. The eggs measure 105–129 × 50–55 µm (much larger than the eggs of *B. trigonocephalum*). The life cycle is direct and similar to those of other hookworms. Infection occurs through the skin but also by ingestion of infective larvae. Migration of the larvae via blood through the lungs, tra-

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chea, oesophagus and finally intestines where they mature about 10 weeks after infection. This is a very potent anaemia-causing pathogen. About 100 adult worms were found to be sufficient to cause death. The worms are usually found to be attached to the mucosa of the first part of the small intestine, frequently in groups of two to three. The worms are often surrounded by fresh blood. After primary infections there is a good immunity which prevents from reinfections.

Geographic distribution: India, Indonesia, Africa

Symptoms: Anaemia and sudden death may occur in heavy infections. In more chronic infections the usual signs of hookworm infection are seen.

Significance: The worms are potent blood-suckers and may be of great significance in endemic areas. The skin-penetrating larvae were suspected to introduce the organisms of footrot into the feet of sheep.

Diagnosis: This is made by demonstrating the characteristic large eggs in the faeces. These eggs are at first sight similar to *Nematodirus* spp. eggs. Demonstration of adult worms in the small intestine.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figure 326, 327)

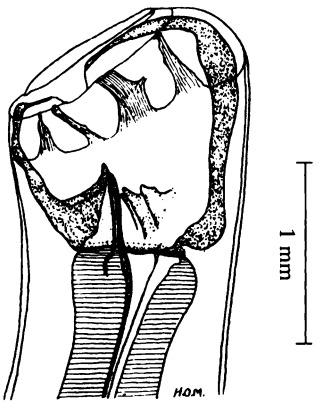

Fig. 326 *Gaigeria pachyscelis*; anterior end with buccal capsule [3]

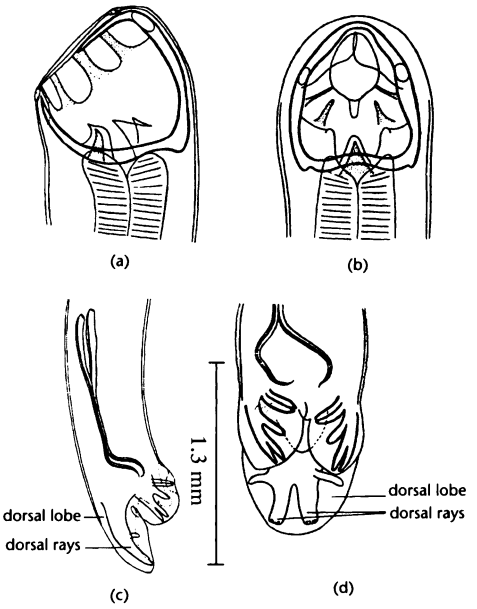

Fig. 327 *Gaigeria pachyscelis*; anterior end lateral (a), dorsal (b), tail end of a male lateral (c) and ventral view (d). The tail end shows prominent dorsal lobes compared with *B. trigonocephalum* [5]

Trichuris ovis and *Trichuris globulosa*

Whipworm

Location: Caecum

Hosts: Sheep, goat, camel, cattle and other ruminants

Species description: Nematodes with a long, slender neck and a much thicker hind end, giving them the appearance of a whip. Males are 50–80 mm long and have a curled tail. Females are 35–70 mm long. Direct nematode life cycle. Infective larvae develop within the eggs and infection is acquired by ingestion of eggs containing infective larvae (second-stage larvae). These infective eggs are very resistant to adverse conditions and may remain viable for years. Prepatent period is 1–3 months and varies markedly among the various species. Larval penetration into the intestinal wall may cause irritation, haemorrhage and may be instrumental for secondary bacterial infections. Heavy infections pro-

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duce thickening of the caecal wall and normal fluid absorption is impaired.

Geographic distribution: World-wide

Symptoms: Colitis, secondary infections and diarrhoea may occur in heavy infections.

Significance: Importance in sheep is unknown. High numbers of larvae cause haemorrhages and localised oedema when they penetrate the gut. The pathogenic effect of adult worms is low and generally additive to other worms.

Diagnosis: Demonstration of the characteristic, brown, barrel-shaped eggs with a transparent plug at either pole. Disease produced by immature stages may only be recognised at necropsy.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figures 328, 329, 330)

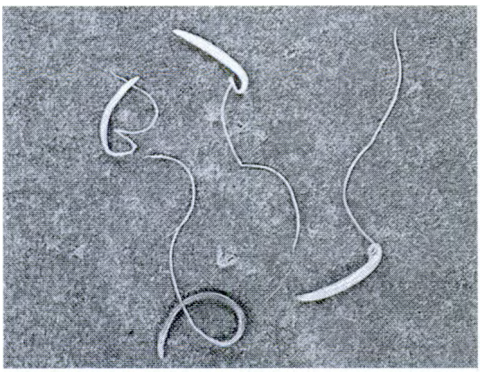

Fig. 328 *Trichuris* sp. (female: 35–70 mm and male: 50–80 mm) [32]

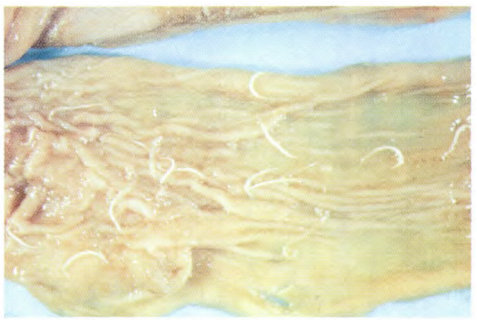

Fig. 329 *Trichuris* sp. attached to the large intestinal mucosa of a sheep [4]

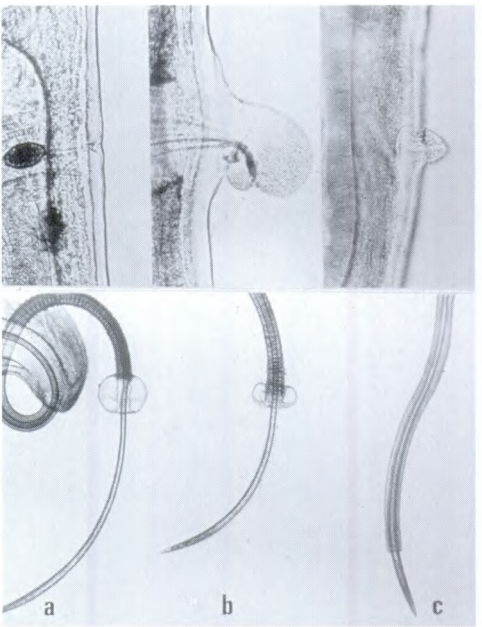

Fig. 330 *Trichuris* sp. of small ruminants; vulva (above) and spiculum (below) of *T. globulosa* (a), *T. ovis* (b) and *T. skrjabini* (c) [4]

Capillaria bovis

Remarks: This parasite occurs world-wide in the gastrointestinal tract of cattle, sheep and goats. The pathogenic role has not yet been defined conclusively, but it seems to be non-pathogenic (☞ CATTLE, 1).

Skrjabinema ovis and *Skrjabinema alata*

Small ruminant pinworms

Location: Caecum

Hosts: Sheep and goat

Species description: These small nematodes belong to the family of the *Oxyuridae*. They are small, about 3–8 mm in length and have 3 large lips and 3 small intermediate lips. The oesophagus terminates in a large spherical bulb. There is a single spicule in the male. The life cycle is direct. The eggs are embryonated when they are deposited by the female in the perianal region. Eggs drop off and are ingested. Larvae hatch in the small intestine and migrate to

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the large intestine where they mature in about 25 days after infection.

Geographic distribution: World-wide; in Africa: Kenya, Nigeria, Tchad, Réunion

Symptoms: Unknown

Significance: *Skrjabinema* spp. are non-pathogenic. They may be mistaken for other dangerous nematodes such as *Oesophagostomum columbianum*.

Diagnosis: Eggs containing larvae may be found on the skin surrounding the anus. Adult worms may be seen in the colon at necropsy. The typical oesophagus of *S. ovis* distinguishes it from fourth-stage larvae of *O. columbianum*.

Therapy and Prophylaxis: ☞ CATTLE, 1; THERAPY OF NEMATODE INFECTIONS

(Figures 331, 332)

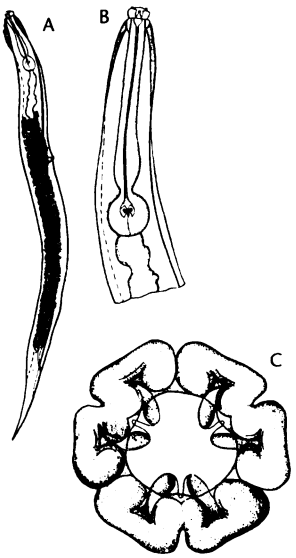

Fig. 332 *Skrjabinema ovis*; adult female (A), anterior end (B) and anterior view (C) [34]

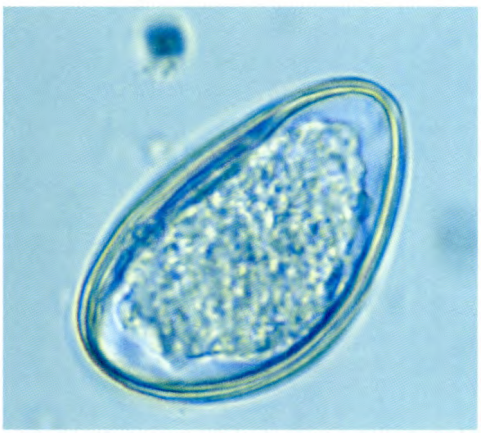

Fig. 331 Egg of *Skrjabinema ovis* (54–57 × 32–34 µm)

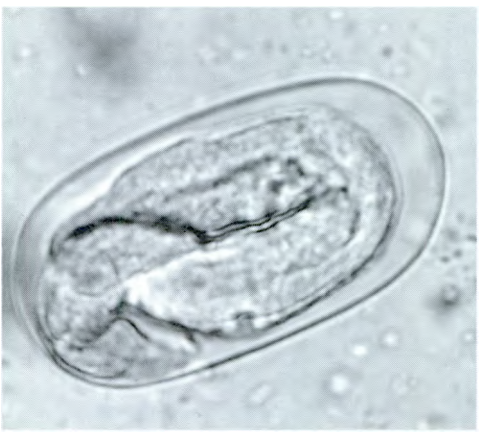

Fig. 333 Egg of *Strongyloides papillosus* (47–65 × 25–26 µm)

Strongyloides papillosus
Intestinal threadworm

Remarks: General description ☞ CATTLE, 1. Transmammary infection occurs. Larvae are present in the ewe's milk from the 8th to the 19th day after lambing. Eggs of *S. papillosus* may occur in the lamb's faeces as early as 21 days after birth. An infection dose of 11,000 larvae were found to be lethal to lambs and kids. Enteritis with diarrhoea or constipation followed by secondary bacterial infections were found in heavily infected sheep and goats. Anaemia due to the reduced absorption of proteins which are essential for haematopoiesis and low calcium, magnesium and phosphate levels were also observed in heavy infec-

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tions. General depression and paralysis was found just prior to death. Young animals are usually severely affected. Infections in older animals are rare. Kraaling facilitates the spread of *Strongyloides papillosus*.

Therapy and Prophylaxis: ☞ CATTLE, ■ 1; THERAPY OF NEMATODE INFECTIONS (Figure 333)

- **Therapy of nematode infections**

Gastrointestinal helminth infections are among the most important constraints of small ruminant rearing world-wide (☞ FURTHER READING). This is especially true in many tropical and subtropical regions. The parasites are highly adapted to the particular climatic conditions (e.g. arrested development during winter or dry season) and this should be considered for the control. As a rule sheep and goats have their own parasites, a feeding pattern which is different from cattle and therefore a different epidemiology as far as gastrointestinal helminths are concerned. The following basic health prevention should be carried out in both traditional and intensified sheep rearing: Vaccinations against the endemic viral and bacterial diseases (mainly: Peste des Petits Ruminants (PPR), Pasteurellosis, Anthrax, Blackleg (*Clostridium chauvoei*)). All the animals of a flock should further be treated with an anthelmintic during the rainy season either regularly every month when stocking rates are high or 3 weeks after the onset of the rains and once around 3–4 weeks before the end of the rains. At the end of the rainy season a high proportion of ingested nematode larvae tend to remain arrested in their development. These arrested larvae may be responsible for "dry season outbreaks" and early outbreaks in the following rainy season. For the last treatment of the rainy season an anthelmintic should be used which is also effective against the inhibited larvae. Most modern bendzimidazoles (fenbendazole, albendazole, oxfendazole, etc.) and probenzimidazoles (netobimin) and ivermectin are highly effective against inhibited larvae. Thiabendazole, levamisole, pyrantel tartrate are not effective against inhibited larvae. As a rule, animals should be kept worm-free during the rainy season so that they can make best use of the food resources in order to survive the dry season. (☞ CATTLE, ■ 1; THERAPY OF NEMATODE INFECTIONS and Table 5 p. 54)

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2 Stages in the blood and circulatory system

PROTOZOA

TRYPANOSOMATIDAE

Trypanosoma congolense, Trypanosoma vivax and *Trypanosoma brucei* Tsetse-transmitted trypanosomosis

Remarks: *T. congolense, T. vivax* and *T. brucei* may cause a chronic disease in sheep and goats, and may last from several weeks to several months. These three species belong to the *Salivaria*. The symptoms are mild and not very specific. Infected animals show fever attacks, rough and dull coat. Reproduction may be seriously impaired. The chronic trypanosome infection may render animals more susceptible to other diseases, especially when the animals live under poor nutritional conditions. The Sahelian breeds are very susceptible to trypanosomes but they avoid tsetse-infested areas. The indigenous dwarf breeds in the Guinean zone seem to be resistant to trypanosome infections and the disease is rarely detected in these breeds. A high percentage of animals kept in high tsetse-challenge areas are generally found to carry trypanosomes but these parasites are often of minor importance for the anaemia, weakness and unthriftiness. Very often these animals carry heavy burdens of gastrointestinal parasites and viral and bacterial diseases (Peste des Petits Ruminants (PPR), Pasteurellosis and other endemic diseases). Regular anthelmintic treatments during the rainy season and prophylactic vaccination against PPR are essential for a successful small ruminant production. The prophylactic use of trypanocidal drugs is generally not indicated in indigenous small ruminant breeds (☞ CATTLE, ■ 2). For therapy and prophylaxis ☞ Table 6 p. 56, Table 8 p. 59.

Trypanosoma melophagium

Remarks: This non-pathogenic trypanosome infects sheep. It is 50–60 µm in length and resembles *T. theileri* (☞ CATTLE, ■ 2). It belongs to the Stercoraria and is transmitted by the sheep ked, *Melophagus ovinus*, a pupiparous bloodsucking insect that permanently infests sheep. Infection occurs by contamination of the skin and if a ked is eaten by the sheep, the metacyclic stages penetrate the buccal mucosa. *T. melophagium* is widespread and usually demonstrable only by culture techniques but it may be also demonstrated microscopically in the peripheral blood.
(Figure 334)

Fig. 334 *Trypanosoma melophagium* (50–60 µm long) [8]

BABESIIDAE

Babesia motasi (syn. *Piroplasma motasi*) European piroplasmosis of small ruminants

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Hosts: Sheep and goat

Vector: *Haemaphysalis* spp. (*H. punctata; H. sulcata* and *H. parva*) in Europe, Mediterranean basin and northwestern Africa, *Dermacentor* spp. and *Rhipicephalus bursa* may also be involved.

Species description: This is a large form, measuring 2.5–4 × 2 µm, the pyriform stages resembling those of *Babesia bigemina*. The angle between the parasites is acute. The organisms occur singly or in pairs. The development cycle is similar to that of *B. bigemina*. Both transovarian and stage to stage transmission occur in *Rhipicephalus bursa*. Recovered animals are immune against *B. motasi* but not against *B. ovis* infections.

Geographic distribution: Southern Europe, Middle East, UIS, Southeast Asia, northwestern Africa

Symptoms: In the acute form high fever, haemoglobinuria, severe anaemia and prostration occur. Death is common. In the chronic form there are no characteristic signs and death is unusual.

Significance: *B. motasi* is a serious infection and frequently fatal in the acute form.

Diagnosis: This is based on clinical signs and the demonstration of the parasites in the peripheral blood. Highest parasitaemia levels are found during the time of acute fever.

Therapy: Diminazene aceturate (3.5 mg/kg, im.), imidocarb dipropionate (1.2–2.4 mg/kg, sc.) and quinuronium sulfate (1–2 mg/kg, sc. or im.) can be used against *B. motasi* infections.

Prophylaxis: The control of *Babesia* spp. in sheep and goats is similar to that in cattle and depends on regular tick control, the therapeutic and prophylactic use of drugs and attempts to immunize young animals with blood from older infected animals followed by a treatment with a babesiacidal drug (☞ CATTLE, ■ 2).

(Figures 335, 336)

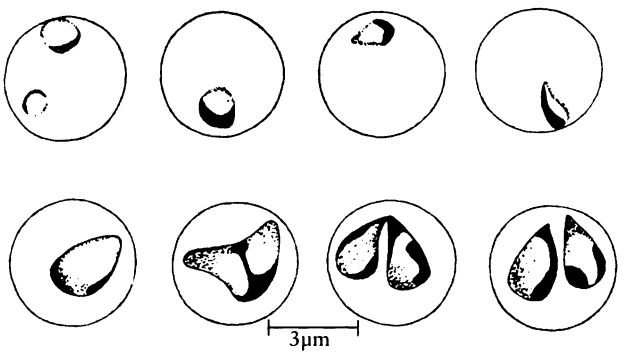

Fig. 335 *Babesia* species of sheep; *Babesia ovis* (upper row) and *Babesia motasi* (lower row) [3]

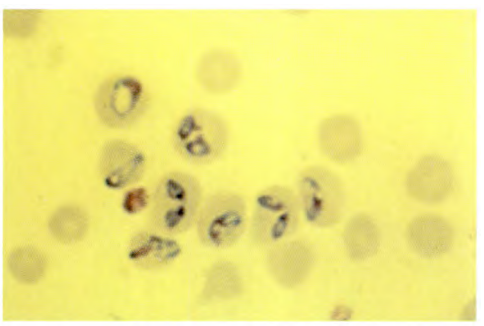

Fig. 336 *Babesia motasi*; stained bloodsmear [13]

Babesia ovis Tropical babesiosis of small ruminants

Hosts: Sheep and goat

Vector: *Rhipicephalus bursa* (Mediterranean basin and central-western Asia) and *Rhipicephalus evertsi* (tropical Africa) and probably other *Rhipicephalus* spp.

Species description: *B. ovis* is much smaller than *B. motasi*, being 1–2.5 µm in length. The majority of the organisms are round, occuring at the margin of the red cell. Pyriform

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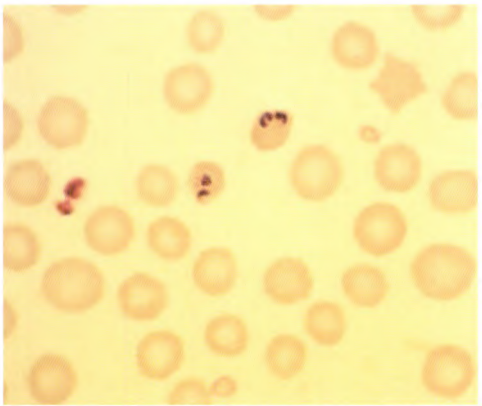

Fig. 337 *Babesia ovis*; stained bloodsmear [15]

organisms are comparatively rare. *B. ovis* is morphologically similar to that of *Babesia bovis*. *Rhipicephalus bursa* is a 2-host tick and transovarian and stage to stage transmission occurs in *R. bursa*. Recovered animals are immune to reinfection and there is no cross-immunity with *B. motasi*

Geographic distribution: Southern and central Europe, northern and tropical Africa, central-western Asia

Symptoms: Fever, jaundice, haemoglobinuria and anaemia may be seen in acute cases. The effects are usually less severe than those seen in *B. motasi* infections.

Significance: *B. ovis* may be an important cause of anaemia and icterus of small ruminants in endemic areas. It is considerably less pathogenic than *B. motasi*. In traditional management systems in endemic zones the disease passes often unnoticed.

Diagnosis: Demonstration of the organisms in stained blood smears and the clinical signs. In chronic infections about 1% of the erythrocytes are infected.

Therapy: Imidocarb dipropionate (1.2–2.4 mg/kg, sc.) is effective against *B. ovis* infections. Diminazene aceturate (3.5 mg/kg, im.) is only partially effective against *B. ovis*.

Prophylaxis: The control of *Babesia* spp. in sheep and goats is similar to that in cattle (☞ CATTLE, ■ 2 and *B. motasi*).

(Figure 337)

THEILERIIDAE

Theileria hirci (syn. *T. lestoquardi*)
Malignant theileriosis of small ruminants

Hosts: Sheep and goat

Vector: *Rhipicephalus bursa* and *Hyalomma anatolicum* and other species to be determined

Species description: This species is found in lymphocytes and erythrocytes of sheep and goats. The majority of the erythrocytic forms is round to oval (0.6–2 µm in diameter), about 20% are rod-shaped and a small percentage are *Anaplasma*-like. Binary or quadruple fission occurs in the erythrocytes. Schizonts occur in the lymphocytes of the spleen and lymph nodes. They range in size from 4–10 µm and contain up to 80 chromatin granules, 1–2 µm in diameter. Both macroschizonts and microschizonts occur. The disease may be highly fatal, mortality ranging from 50–100%. The infection is mild in young lambs and kids, due to maternal immunity. An acute form is more common, but subacute and chronic forms have been observed. The acute form resembles ECF.

Geographic distribution: North and East Africa, southern Europe, southern UIS, Asia.

Symptoms: In the acute form, high fever, listlessness, nasal discharge, jaundice, petechial haemorrhage in submucous, subserous and subcutaneous tissues may occur. The lymph nodes, spleen and kidneys are markedly enlarged. Haemoglobinuria may occur.

Significance: *T. hirci* is a very pathogenic species causing severe losses among small ruminant populations, mainly in the Mediterranean and North African regions.

Diagnosis: Is based on the detection of the organisms in blood smears or lymph nodes or spleen smears.

Therapy and Prophylaxis: ☞ CATTLE, ■ 2 (*T. parva*), parvaquone (1 × 20 mg/kg, im.), buparvaquone (2 × 2.5 mg/kg, im.) or the coccidiostat halofuginone (1 × 1.2 mg/kg, po.) may also be used to treat theileriosis

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in small ruminants. Chlortetracycline and oxytetracycline are relatively ineffective. Tick control is an essential part of the prophylaxis.
(Figures 114, 115, 338)

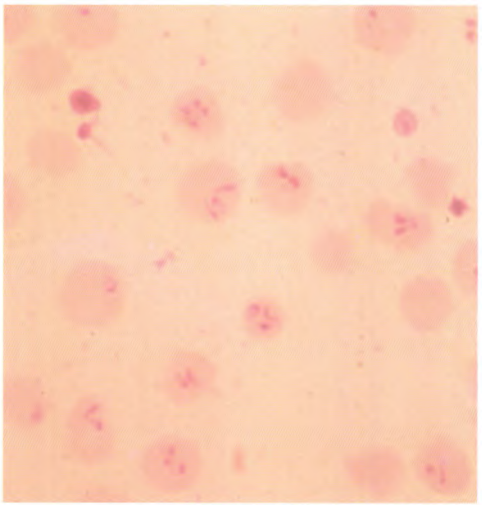

Fig. 338 *Theileria hirci*; erythrocyte forms (round forms 0.6–2 µm) [15]

Theileria ovis Benign theileriosis of small ruminants

Hosts: Sheep and goat
Vector: *Rhipicephalus bursa* in the Mediterranean basin and *Rhipicephalus evertsi* in tropical Africa
Species description: This species is found in lymphocytes and erythrocytes of sheep and goats and is much more widely distributed than *T. hirci*. The erythrocytic stages resemble those of *T. hirci* in shape and size but are much more sparse in infected animals, with less than 2% of the erythrocytes infected.
Geographic distribution: Africa, Asia, India, UIS, parts of Europe
Symptoms: The infection is usually mild and clinically inapparent.
Significance: The pathogenicity of this *Theileria* species is low and there is seldom mortality but the prevalence may be very high in endemic areas.
Diagnosis: Demonstration of the parasites in stained blood or lymph node smears. *T. ovis* is structurally indistinguishable from the more pathogenic *T. hirci*, but the small number of parasites present and their lack of pathogenicity may help to differentiate them.
Therapy and Prophylaxis: ☞ *T. hirci*; but therapy is generally not indicated.

Theileria separata Mild ovine theileriosis

Remarks: A mild theileriosis has be found in sheep. The vector is *Amblyomma variegatum*. It is distinguished from *T. ovis* by the presence of vela on some trophozoites. This species causes a mild and often clinically inapparent theileriosis in sheep.
(Figure 339)

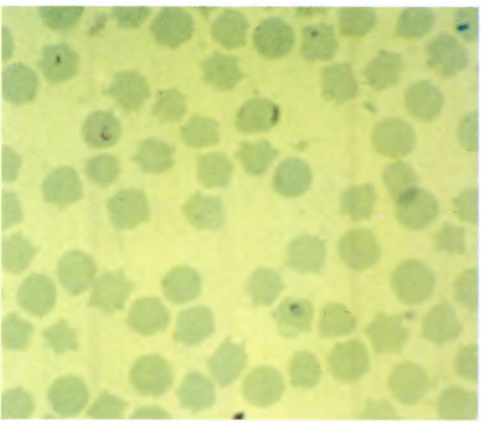

Fig. 339 *Theileria separata*; erythrocyte forms [13]

RICKETTSIALES

ANAPLASMATACEAE

Anaplasma spp.

Remarks: ☞ CATTLE, ■ 2 ANAPLASMATACEAE

Anaplasma ovis Tropical anaplasmosis of small ruminants

Remarks: It is comparable to *A. marginale* with 70% located marginally or peri-

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pherally. Its distribution corresponds to the most important vectors, *Rhipicephalus bursa* in the Mediterranean basin and *Rhipicephalus evertsi* in tropical Africa. Transplacental infection occurs.

Symptoms: Similar to those of *A. marginale* (☞ CATTLE, ■ 2)

Therapy and Prophylaxis: ☞ CATTLE, ■ 2 *A. marginale*

(Figure 340)

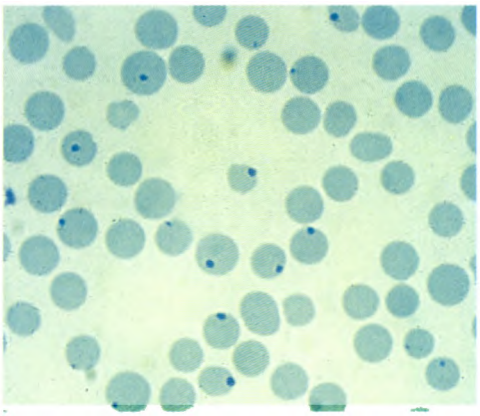

Fig. 340 *Anaplasma ovis*; stained bloodsmear [15]

Fig. 341 *Eperythrozoon* sp.; stained bloodsmear [15]

Eperythrozoon ovis

Remarks: This species occurs on the erythrocyte surface. It is a nonpathogenic form but it sometimes occurs frequently in sheep (☞ CATTLE, ■ 2). *Linognathus ovillus* and possibly also ticks (*Hyalomma marginatum* and *Rhipicephalus bursa*) are vectors of *E. ovis*.

(Figure 341)

EHRLICHIACEAE

Ehrlichia ovina Tropical ehrlichiosis of small ruminants

Remarks: The parasite occurs in mononuclear cells of sheep in North and Central Africa. Vectors are *Rhipicephalus bursa* in Mediterranean basin and *Rhipicephalus evertsi* in tropical Africa. Despite sometimes heavy parasitaemia *E. ovina* usually causes a mild disease similar to that of *E. bovis*.

Therapy and Prophylaxis: ☞ CATTLE, ■ 2 *E. bovis*.

(Figure 342)

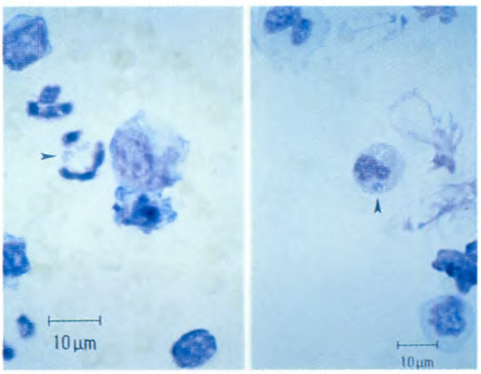

Fig. 342 *Ehrlichia* sp.; mass of elementary bodies (left) and initial body (right)

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Ehrlichia phagocytophila
European ehrlichiosis

Remarks: *E. phagocytophila* occurs in the neutrophilic and eosiniphilic granulocytes of cattle, sheep and other domestic and wild ruminants in Europe. Goats are not very susceptible. *Ixodes ricinus* transmits the disease by transstadial infection. The disease is characterized by fever which occurs after an incubation period of 4–11 days. The animals usually recover after this febrile phase. However, the most characteristic problems are abortion in pregnant ewes and cattle and progressive weight loss following infection may occur. *Ehrlichia phagocytophila* increases the susceptibility to secondary infections. At necropsy hypertrophy of the spleen and lymph nodes may be seen. The diagnosis is made by demonstrating the organisms in stained blood smears. Serological tests (IFAT) may be of value for epidemiological studies.

Cowdria ruminantium Heartwater, Tyewde

Remarks: The infection caused by *Cowdria ruminantium* is virulent, inoculable, and noncontagious. It is specific to ruminants and causes great losses in sheep, goats and cattle. The disease can only be transmitted by ticks. Its symptoms are characterised by gastroenteritis associated with exudative pericarditis, often followed by nervous disorders. The symptoms are almost identical in both cattle and small ruminants, except that the nervous symptoms are rarer in cattle and not as distinct. Digestive signs are more constant in cattle than in sheep and goats. Young calves (< 6 weeks), lambs and kids (< 1 week) are fairly resistant and may recover spontaneously. For symptoms, diagnosis, therapy and prophylaxis ☞ CATTLE, ■ 2.
(Figure 343)

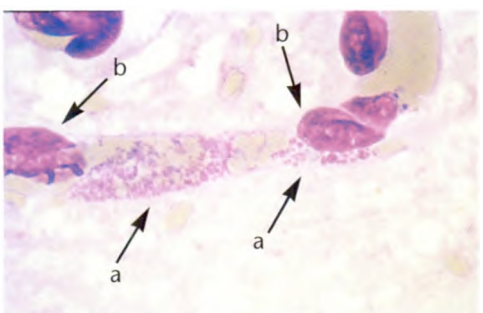

Fig. 343 *Cowdria ruminantium*; stained smear of a brain capillary; masses of organisms in endothelial cells (a) and nuclei of endothelial cells (b) [15]

Toxoplasma gondii and *Sarcocystis* spp.

Remarks: Merozoites may occur in the blood stream during the acute phase (☞ CATTLE, ■ 2).

HELMINTHS

- **Trematoda found in the blood and circulatory system**

Schistosoma spp. Blood fluke, Bilharziosis

Remarks: The following *Schistosoma* species are found in small ruminants in Africa: *S. bovis*, *S. mattheei*, and *S. curassoni*.
Sheep are considerably more susceptible to schistosomosis than cattle and die in large numbers, while cattle survive unless they have very large worm burdens. The infection may go unnoticed for some months in sheep while the animals lose mass at a progressive rate, eventually showing markedly sunken eyes, an anxious expression, intermittent diarrhoea which may contain specks of blood, anaemia and progressive weakness. This eventually leads to death. In South Africa, infected sheep may die of schistosomosis during winter time as a result of respiratory infections. The worms may survive for many years in the sheep. A chronic course and death 4 years after the infection occurred in sheep following *S. mattheei* infection. The disease in sheep is characterized by anorexia, muscle wasting, haemorrhage and plasma-leaking into the gastrointestinal tract and impaired diges-

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tion and food absorption. Low serum albumin and haemoglobin may be found following heavy infection. For therapy and prophylaxis ☞ CATTLE, ■ 2.

- **Nematoda found in the blood and circulatory system**

Onchocerca armillata

Remarks: This filarial worm may occasionally be found in the aorta of small ruminants. It is non-pathogenic (☞ CATTLE, ■ 2).

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3 Stages in the urogential system

No pathogenic parasites have been found so far in the urogential system of sheep and goats.

4 Stages in internal organs

4.1 Locomotory system

4.1.1 Muscles

PROTOZOA

***Sarcocystis* spp.** Sarcosporidiosis of small ruminants

Location: Cysts, containing the infective stage, called bradyzoites, occur in the striated musculature of sheep and goat.

Hosts: Sheep are intermediate hosts of four *Sarcocystis* species of which either the dog (*S. ovicanis* and *S. arieticanis*) or the cat (*S. ovifelis*) are final hosts. Goats are intermediate hosts of three *Sarcocystis* species in two of which the dog (*S. capracanis* and *S. hircicanis*) acts as final host.The final host of *S. moulei* is probably the cat. Details of the life cycle of *Sarcocytis* species are still unknown.

Species description: Cysts of several *Sarcocystis* species are found in the muscles of sheep and goats. (For general description and life cycle ☞ CATTLE, ■ 4.1.1). *S. ovifelis* produces big cysts, about 1 cm in length which are macroscopically visible. This species is non-pathogenic. Only those species in which the dog acts as final host are pathogenic. *S. ovicanis* is highly pathogenic for lambs and pregnant ewes. *S. capracanis* is equally pathogenic for kids and adult goats.

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Geographic distribution: World-wide

Symptoms: Anorexia, weakness and death may occur in lambs infected with *S. ovicanis*. A high proportion of pregnant ewes aborted, became anaemic and lost weight during heavy infections.

Significance: *Sarcocystis* spp. are common among small ruminant populations throughout the world. Economic losses during a fresh infection may be higher than expected. Once muscle cysts are formed the infection is usually inapparent.

Diagnosis: At necropsy of acutely ill animals the heart was the most severely affected organ, schizonts were found in epithelial cells. *S. ovicanis* stages have not been found in the placenta, fetus or uterine tissues following abortion (☞ CATTLE, ■ 4.1.1).

Therapy and Prophylaxis: ☞ bovine *Sarcocystis* spp. (☞ CATTLE, ■ 4.1.1)

(Figures 344, 345, 346, 347, Table 13)

Table 13 Sarcocystis species found in the muscles of sheep and goat

Species	Synonym	Pathogenicity			
		IH	DH	IH	DH
S. ovifelis	*S. gigantea**	sheep	cat	0	
S. ovicanis	*S. tenella*	sheep	dog	+++	0
S. arieticanis		sheep	dog	+++	0
S. medusiformis		sheep	cat	+	0
S. capracanis		goat	dog	+++	0
S. hircicanis		goat	dog	+	0
S. moulei		goat	cat	0	0

IH = intermediate host; DH = definitive host; *= macroscopic; 0 = non-pathogenic; + = low; ++ = moderate and +++ = high pathogenicity

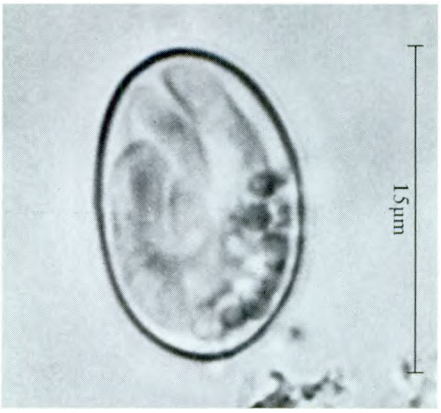

Fig. 345 Sporocyst of *Sarcocystis ovicanis* [4]

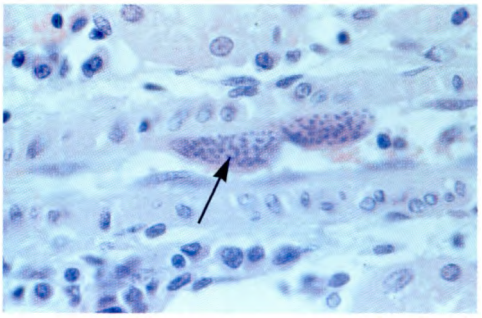

Fig. 346 *Sarcocystis capracanis*; schizonts with merozoites in endothelial cells (400×) [4]

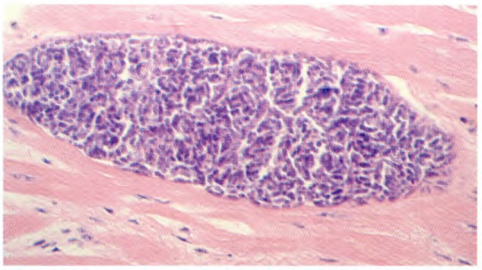

Fig. 344 *Sarcocystis* sp. HE-stained muscular cyst (150 µm long)

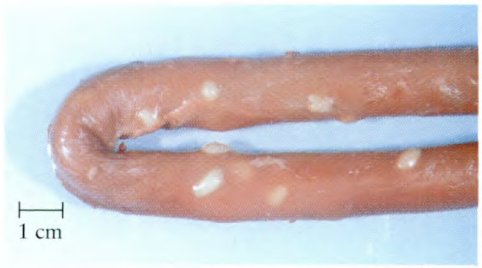

Fig. 347 *Sarcocystis ovifelis* (syn. *S. gigantea*); cysts on the outside of the oesophagus of a sheep

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Toxoplasma gondii

Remarks: *T. gondii* is an important cause of abortion in sheep and goats and responsible for high mortalities in lambs and young goats (> 10% of mortalities in lambs). Transmission occurs via infective oocysts from cats, congenitally and galactogenically. Abortion occurs mainly when pregnant goats and sheep acquire first infection during pregnancy. Animals normally do not show any clinical signs during the abortion. Infections are more serious in goats than in sheep. The placenta shows numerous yellowish-white necrotic foci in the villi. Tissue cysts of *T. gondii* may also occur in ruminants as they do in pigs (☞ SWINE, ■ 4.1). Muscle cysts are about 100 µm in diameter, containing merozoites which are 6–8 µm in diameter. Encephalomyelitis due to *T. gondii* infections may occur in sheep. Affected sheep have myelomalacia and schizonts appear in astrocytes. Infection is most often acquired orally by ingestion of sporulated oocysts from the faeces of cats.

Cave: Uncooked meat (mainly meat of pigs but also of sheep) is a potential source of Toxoplasma infection in man.

(Figure 348, 349)

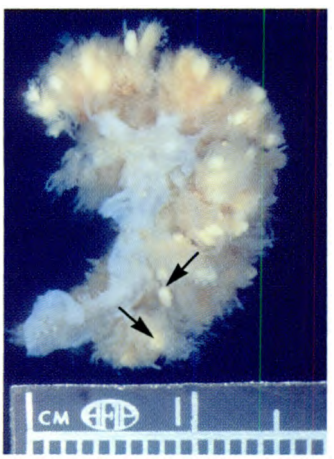

Fig. 348 *Toxoplasma gondii*; placenta of sheep showing cotyledon with numerous yellowish-white necrotic foci in villi [22]

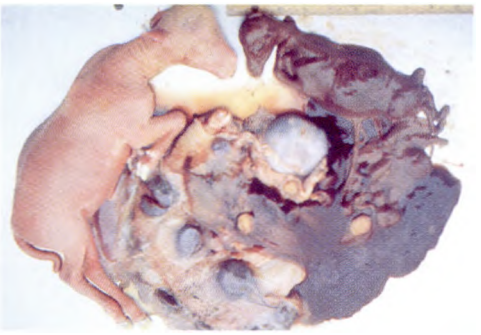

Fig. 349 *Toxoplasma* abortion of sheep; lesions characteristic of *Toxoplasma* are found in cotyledons of both lambs [22]

HELMINTHS

- **Cestoda larvae found in the muscles**

Cysticercus ovis Larvae of the canine tape worm Taenia ovis

Location: Cysticerci are found in the heart, diaphragm, masseters and other musculature.

Intermediate Hosts: Sheep and goat

Species description: The life cycle of *Taenia ovis* is similar to that of *Taenia saginata* (☞ CATTLE, ■ 4.1.1). Larvae (oncospheres) are disseminated with the blood and develop in skeletal and cardiac musculature. Mature cysticerci measure 10 × 5 mm and contain 1 protoscolex.

Geographic distribution: World-wide

Symptoms: Clinical signs are not usually seen.

Significance: This parasite is of importance as a cause of financial loss to the meat industry. Infected carcasses are unaesthetic and usually condemned at meat inspection.

Diagnosis: Detection of cysticerci in the striated musculature at meat inspection. *Cysticercus ovis* (possesses hooks) is macroscopically not distinguishable from *Cysticercus bovis* (no hooks).

Therapy: This is generally not indicated in the intermediate host. A consequent treatment of all dogs within a population would inevitably reduce the incidence of cysticerosis in small ruminants, but this is generally not feasible.

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Prophylaxis: Infected meat should not be fed to dogs. A highly protective recombinant vaccine may soon be available. The vaccination of small ruminants against *T. ovis* will then be the prophylactic measure of choice.
(Figure 350, 351)

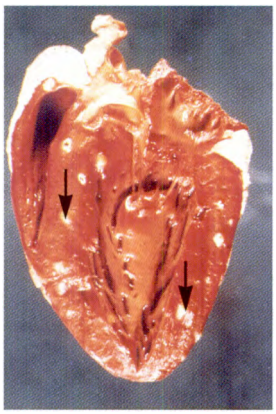

Fig. 350 *Cysticercus ovis*; heart muscle with cysts [10]

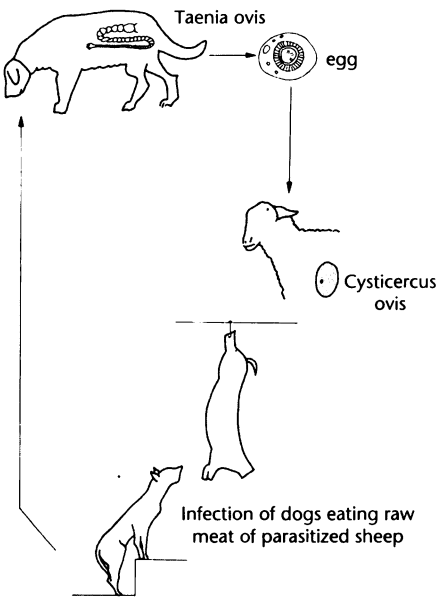

Fig. 351 Life cycle of *Cysticercus ovis* [16]

4.1.2 Tendons

HELMINTHS

- Nematoda larvae and adult nematodes found in the tendons

Onchocerca gutturosa (syn. *O. lienalis*)

Remarks: This parasite may sometimes be found in sheep (☞ CATTLE, ■ 4.1.2).

4.2 Liver

HELMINTHS

- Trematoda found in the liver

Fasciola gigantica Giant liver fluke

Remarks: This is the common liver fluke of domestic stock in Africa. It also occurs in Asia, the Pacific islands, southern USA, southern Europe and the Middle East. In Africa the parasite occurs in the bile ducts of cattle, sheep, goats, dromedary, horse, pigs and many other animal species (☞ CATTLE, ■ 4.2).

Fasciola hepatica Common liver fluke, liver fluke disease, liver rot

Remarks: This parasite occurs in the bile ducts of sheep, goat, cattle, horse, donkey, pig and many other animal species. The fluke is cosmopolitan in its distribution and the cause of fasciolosis, especially in sheep and cattle (☞ CATTLE, ■ 4.2). *F. hepatica* is a world-wide problem in sheep rearing systems. Fasciolosis in sheep is characterized by decreased wool growth, weight loss and liver condemnation at slaughter. *F. hepatica* may cause an acute form in sheep, associated with sudden deaths.

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Dicrocoelium spp. Lancet flukes

Remarks: *D. hospes* occurs in the bile ducts and gall bladder of sheep, goat, cattle and other ruminants from Egypt to Angola, particularly in the western half of the African continent. This parasite is closely related to *D. dendriticum*. In heavy infections, extensive hepatic cirrhosis and distension of the bile ducts may occur. Such sheep may be unthrifty (☞ CATTLE, ■ 4.2).
(Figures 352, 353, 354, 355)

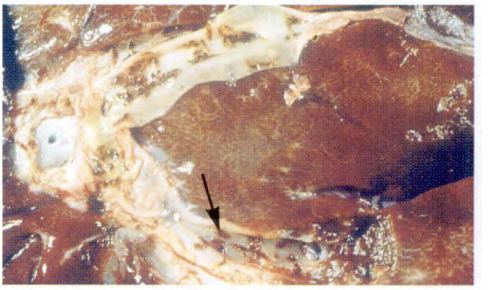

Fig. 352 *Dicrocoelium dendriticum*; adult flukes in bile ducts of a sheep

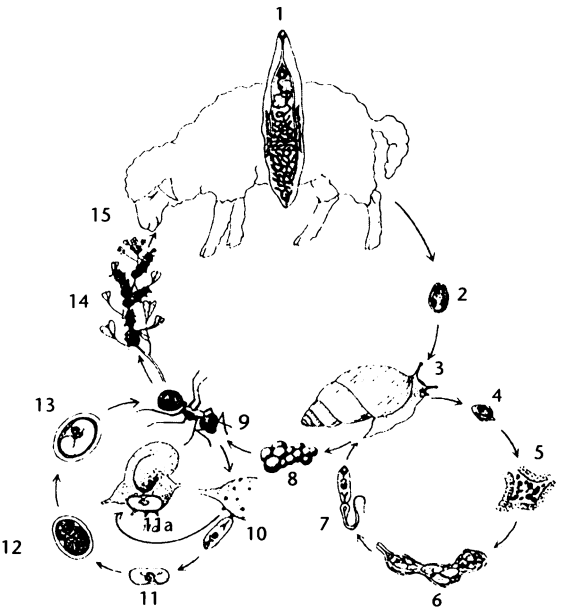

Fig. 353 Life cycle of *Dicrocoelium dendriticum*; (1) adult flukes in the liver of sheep, (2) eggs excreted with the faeces, (3)–(8) development in snails (first intermediate hosts), (9)–(13) development in ants (second intermediate hosts) and (14) ants containing the infective metacercariae in the brain attach to herbage

Fig. 354 Egg of *Dicrocoelium dendriticum* (36–45 × 25 µm)

Fig. 355 *Dicrocoelium dendriticum* infected ants remain attached to herbage

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- Cestoda found in the liver

Echinococcus granulosus (syn. *E. uniocularis*) Hydatid tapeworm, hydatidosis

Remarks: Hydatid cysts are often found in the liver and lungs of sheep where they are often multivesicular. Infected sheep usually do not show any clinical signs but constitute a major reservoir for new infections. Infected organs should never be fed to dogs, which are the principal hosts of *E. granulosus*. Sheep and goats and many other ungulates and man may act as intermediate hosts. Infection of the intermediate hosts are usually inapparent, except in man where hydatidosis is a severe condition and the function of the affected organ is frequently impaired (☞ CATTLE, ■ 4.2). (Figure 356)

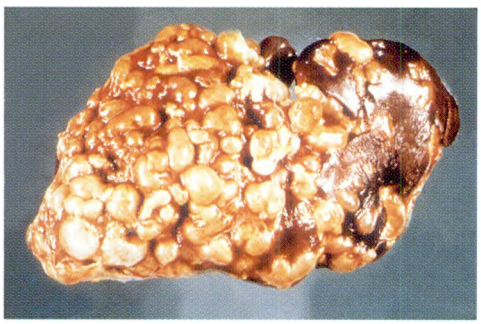

Fig. 356 *Echinococcus granulosus*; hydatid cyst in the liver of a sheep [10]

Stilesia hepatica

Location: Bile ducts and small intestine
Hosts: Sheep, goat, wild ruminants and rarely cattle
Species description: The life cycle is not known but probably involves oribatide mites. It is non-pathogenic. Heavy infections are often seen in apparently perfectly healthy sheep. Affected livers may sometimes show cirrhosis and thickened walls of the bile ducts. Although the bile ducts may be almost occluded, or even form sac-like occlusions, no icterus or clinical signs are seen. Adults have a large scolex with prominent suckers and measure 20–50 cm in length and up to 2 mm in width.
Geographic distribution: Tropical and southern Africa, Asia
Symptoms: Infections are often clinically inapparent.
Significance: The condemnation of a large proportion of sheep livers at meat inspection is the major loss due to this parasite. The parasite itself does not impair the raising of sheep.
Diagnosis: Proglottids or ovoid eggs may appear in the faeces. The proglottids are short and the eggs measure 16–19 × 26 µm.
Therapy: Praziquantel (15 mg/kg, po.) can be used for treatment.
Prophylaxis: Unknown
(Figures 37, 43, 357, 358)

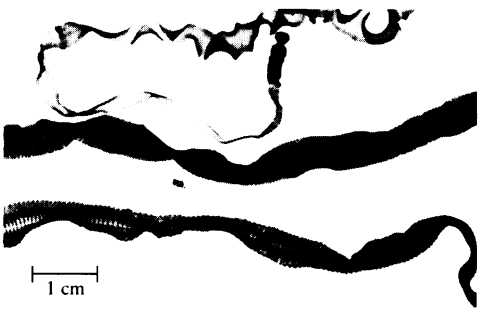

Fig. 357 *Stilesia hepatica*; scolex and proglottids (20–50 cm × 2 mm) [8]

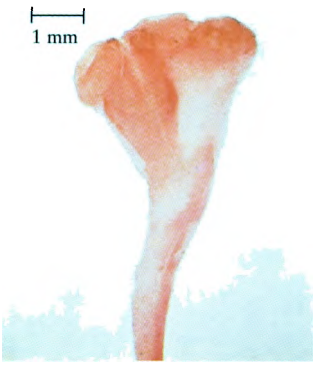

Fig. 358 *Stilesia hepatica*; scolex [4]

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Cysticercus tenuicollis

Remarks: Migrating oncospheres may occasionally be found in the liver parenchyma and thin necked cysts may be found on the serosa of the liver (☞ SHEEP AND GOATS, ■ 4.4).

Linguatula serrata "tongue worm"
(☞ CATTLE, ■ 4.2)

4.3 Respiratory system

HELMINTHS

- **Cestoda found in the respiratory system**

Echinococcus granulosus (syn. *E. unilocularis*)
Hydatid tapeworm, hydatidosis

Remarks: Larvae (Hydatid cysts) of *E. granulosus* are often found in the lungs and liver of cattle and other domestic animals (☞ CATTLE, ■ 4.2).

- **Nematoda found in the lungs and trachea**

SYNGAMIDAE

Mammomonogamus laryngeus

Remarks: This parasite may occasionally be found in the larynx of small ruminants (☞ CATTLE, ■ 4.3).

Mammomonogamus nasicola

Remarks: This parasite may occasionally be found in the nasal cavities of small ruminants (☞ CATTLE, ■ 4.3).

DICTYOCAULIDAE

Dictyocaulus filaria Large lungworm of small ruminants, parasitic bronchitis

Location: Bronchi and bronchioles

Hosts: Sheep, goat, dromedary and some wild ruminants

Species description: The male is 3–8 cm long and the female is 5–10 cm long. The worms have a milk-white colour and the intestine shows as a dark line. There are very small lips and a very small, shallow buccal capsule. The spicules of the male bursa are stout, dark-brown, boot-shaped and 0.4–0.64 mm long. The eggs measure 112–138 × 60–90 µm. The life cycle is direct and similar to that of *D. viviparus* (☞ CATTLE, ■ 4.3). The prepatent period is 24–28 days. In infected animals the bronchial mucosa and the peribronchial tissue are inflamed and infiltrated with mononuclear cells. Localized pneumonia, atelectasis and compensatory emphysema may be seen.

Geographic distribution: World-wide, especially in warm moist areas; *D. filaria* was reported from Tchad, Sudan, Ethiopia, Kenya, Mozambique, Zimbabwe, Malawi and South Africa

Symptoms: Infected animals may cough and show nasal discharge. Dyspnoea, rapid respiration and abnormal lung sounds can be heard on auscultation. Young animals are mainly affected, but the disease may occur at all ages and is usually chronic. The body temperature is not elevated unless pneumonia and secondary infection develops.

Significance: Lungworm infections may cause serious losses in sheep in endemic areas. Lung damage often persists for long periods and animals are unthrifty and susceptible to secondary infections.

Diagnosis: This is made by demonstrating first-stage larvae in fresh faeces. Eggs or larvae may be found in the sputum or nasal discharge, but during the prepatent period their absence is not significant. At necropsy atelectatic areas of variable size may be seen in the lungs. The bronchi in the affected parts contain the worms and large amounts of mucus, which are mixed with blood and desquamated epithelial cells, leucocytes and eggs of the worms.

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Therapy: ☞ CATTLE, ■ 4.3 THERAPY OF *DICTYOCAULUS VIVIPARUS*

Prophylaxis: In endemic areas, young sheep must be kept off areas where infected sheep have been grazing. Infected animals should be treated with anthelmintics. For further prophylactic measures ☞ also CATTLE, ■ 4.3. The prophylactic use of irradiated *D. filaria* larvae has been successfully used in some countries but the vaccine is not commercially available.

(Figures 313, 359)

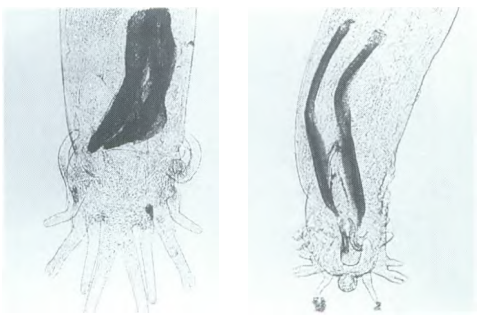

Fig. 359 *Dictyocaulus filaria* (left) and *Protostrongylus rufescens* (right); posterior end of males [4]

PROTOSTRONGYLIDAE

Protostrongylus rufescens Red lungworm; ***Muellerius capillaris*** Hair lungworm; ***Neostrongylus linearis*** **and** ***Cystocaulus ocreatus***

These four species are generally referred to as small lungworms of small ruminants.

Location: Adult worms occur in small air passages (small bronchioles, bronchioli) and in some species even in the alveoli and the pulmonary parenchyma and the subpleural tissue (e.g. *Muellerius capillaris*). The eggs are found in the bronchioli and alveoli.

Hosts: Sheep, goat and some wild ruminant species

Species description: The life cycle of all small lungworms includes an intermediate host which may be one species of land snails of the genera *Helicella, Theba, Abida, Zebrina, Arianta, Succinea, Helix, Cepaea, Monacha,* etc. or nude slugs such as *Limax, Agriolimax, Arion*, etc. The first-stage larvae which are passed in the faeces penetrate the foot of the snail to develop to the infective third-stage in about 12–14 days. The final host becomes infected by swallowing the snail with its food. The larvae are liberated in the gut of the ruminant host and penetrate the intestinal wall and pass via the mesenteric lymphatic glands to the lungs. Further development is similar to that of *Dictyocaulus* species. *Muellerius capillaris* and *Protostrongylus rufescens* are frequently encountered in Europe but they are also found in many parts of Africa. Differentiation can easily be made by the examination of the first-stage larvae which are passed in the faeces of animals with patent infections. As a general rule, small lungworms cause extensive alterations of the lungs but generally little or no clinical signs. In some areas almost 100% of the adult sheep and goats are infected and show massive lung reactions. These are not easily distinguishable between the different lungworm species. The worms live in the small air passages and cause inflammation. The resulting exudate fills the alveoli and the inflammatory and allergic process spreads to the peribronchial tissue. The alveolar epithelium is desquamated and infiltration with round cells and proliferation of the surrounding connective tissue occurs. The result is a small focus of lobular pneumonia and the number of such foci depends on the number of parasites present. In *Muellerius capillaris* infections, greyish nodules up to 2 cm in diameter may be seen on the pleural surface. These "brood nodules" consist of necrotic masses, resulting from leucocytes and pulmonary tissue and masses of eggs, and they are surrounded by a wall of connective tissue and giant cells. These nodules may calcify. An adenoma-like proliferation of the bronchial epithelium and a hypertrophy of the smooth muscles around the alveoli and bronchioli is often seen. *Muellerius capillaris* is usually not found in lambs or kids under six months of age. Thereafter the prevalence increases with age and

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may reach 100% in goats older than 3 years. *Muellerius capillaris* may cause interstitial pneumonia and clinical signs. It seems that this species is relatively pathogenic for goats.

Geographic distribution: Europe, Africa, Australia, North America

Symptoms: Animals show no definite clinical signs, but heavy infections affect the general health, and the weakened lungs are susceptible to secondary infections which may produce pneumonia.

Significance: Small lungworms are extremely prevalent and often not associated with clinical disease. However, if the lesions are large and animals kept under poor conditions severe problems may result due to secondary infections.

Diagnosis: This is based on the identification of first-stage larvae in the faeces by means of the Baermann apparatus. The larva of *P. rufescens* is 0.25–0.32 mm long. The tip of its tail has a wavy outline but no dorsal spine. The larva of *M. capillaris* is 0.23–0.3 mm long. The tail of the larva has an undulating tip and a dorsal spine. At necropsy the nodules in the lungs are typical and may support the diagnosis (☞ Fig. 314, p. 158).

Therapy: The therapy of the small lungworms in sheep and goats is difficult. Most of the modern benzimidazoles (albendazole 5 mg/kg, po., fenbendazole 5 mg/kg, po., febantel 5 mg/kg, po., mebendazole 10–20 mg/kg, po., oxfendazole 5 mg/kg, po., netobimin 7.5 mg/kg, po. and levamisole 5.0 mg/kg, sc., are partially effective against the adult worms. Ivermectin (200 µg/kg, sc.) is highly effective against adults of *Protostrongylus rufescens*.

Prophylaxis: This is difficult because of the ubiquitous presence of the intermediate hosts. Often, regular anthelmintic treatment with a broad spectrum compound controls both gastrointestinal and heavy lungworm infestations. Snail control on pastures with molluscicides is ineffective and ecologically not justifiable.

(Figures 360, 361, 362, Table 14)

Table 14 Comparison of prepatent periods and patency periods of different lung worm species

Genus	Prepatent period	Patency
Protostrongylus spp.	4–5 weeks	2 years
Neostrongylus spp.	2 months	2 years
Cystocaulus spp.	1 month	5–6 years
Muellerius spp.	1 month	5–6 years
Dictyocaulus spp.	24–28 days	2–3 months

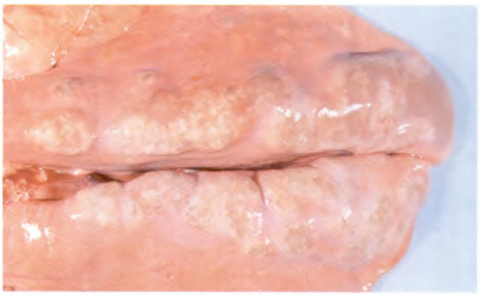

Fig. 360 *Muellerius capillaris*; brood nodules in the lungs (dorsal view)

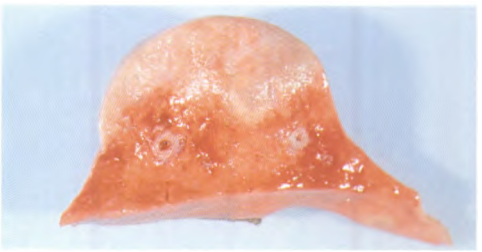

Fig. 361 *Muellerius capillaris*; brood nodules in the lungs (section)

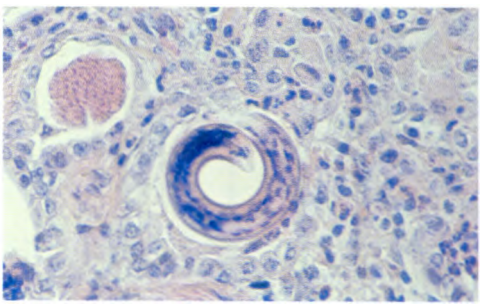

Fig. 362 *Muellerius capillaris*; tissue section showing the parasites and the massive cellular infiltration of the lungs (250 ×)

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- **Insecta larvae found in the respiratory system**

Oestrus ovis Sheep nasal flies, sheep nasal bot, nasal myiasis

Location: Larvae live in nasal passages and frontal and nasal sinuses

Hosts: Sheep and goat

Species description: Adult flies are about 12 mm long. The body is greyish-brown, with many small black spots on the thorax, which is covered with fine, light brown hair. The female flies produce live larvae which are deposited around the nostrils of sheep and occasionally goats. These larvae crawl into the nasal passages where they remain for a variable period, ranging from 2 weeks in summer to 9 months during the colder part of the year. They attach themselves with the oral hooks to the mucous membranes, causing irritation, and then enter the frontal sinuses where they develop into second- and third-stage larvae. The mature larvae are sneezed out by the host and pupate on the ground for 3–9 weeks before becoming adults. The mature flies live for about 2 weeks. Usually 4–15 larvae are present in infested sheep.

Geographic distribution: World-wide

Symptoms: Sneezing and nasal discharges and fly worry are the consequences of *O. ovis* infestations. Decreased appetite, restlessness and poor gains are the results of nasal bot fly strikes. Respiration may be impaired by the larvae and the thickening of the nasal mucosa. Occasionally the larvae may penetrate the bones of the skull and enter the cerebral cavity. These sheep show a characteristic behavior (stagger).

Significance: The larvae cause irritation and excessive secretion. Infected sheep sneeze frequently and secondary bacterial infections are common. Sheep try to avoid attacks by the adult fly by pressing their noses between other sheep or they may run from place to place, stamping their feet and shaking their heads continuously. Consequently their time spent with feeding is considerably reduced.

Diagnosis: Clinical signs may help, but must be differentiated from other conditions with sneezing and nasal discharges. Sometimes larvae may be found on the ground after a severe sneezing attack. Very often the diagnosis can only be made at necropsy when the skull is opened longitudinally. Nasal myiasis often produces clinical signs which are similar to those seen in CNS disorders (restlessness, shaking of the head, etc.).

Therapy: Ivermectin (200 µg/kg, sc.) is highly effective. Ruelene (110 mg/kg, po.) administered as a drench should afford good control. Rafoxanide (7.5–10 mg/kg, po.) as a drench or bolus, trichlorfon (75 mg/kg, po. or as a drench) and nitroxynil (15–20 mg/kg, sc.) have been reported to be effective.

Prophylaxis: Fly repellents may be used but are not very effective.

(Figures 363, 364, 365)

Fig. 363 *Oestrus ovis;* the sheep nasal fly (10–12 mm) [19]

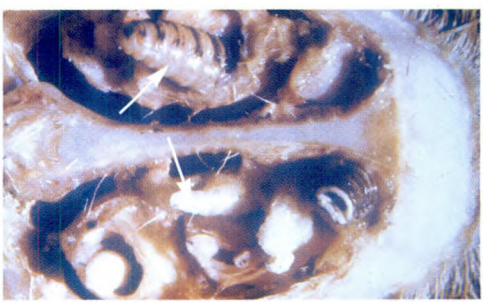

Fig. 364 Conchae of a sheep with *Oestrus ovis* larvae (20–30 mm × 7–10 mm) [8]

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Fig. 365 *Oestrus ovis* infestation causing nasal discharge [15]

4.4 Abdominal cavity

HELMINTHS

- **Cestoda found in the abdominal cavity**

Cysticercus tenuicollis Larval stage of the canine tapeworm *Taenia hydatigena*

Location: Cysticerci are found attached to the omentum, the intestinal mesentery and to the serosal surface of abdominal organs, especially the liver. Adult tapeworms occur in the small intestine of dogs and other Canidae.

Hosts: Sheep and other domestic and wild herbivores including pig and horse, act as intermediate hosts. Dog, wolf, hyaena and other wild carnivores are the final hosts.

Species description: Adult tapeworms are 75–500 cm long and have two rows of 26 and 46 rostellar hooks. Gravid proglottids measure 12 × 6 mm. The eggs are oval and 36–39 × 31–35 µm. Infections of the dogs are usually inapparent. Infections of the intermediate hosts are acquired by ingestion of proglottids or eggs passed in the faeces of the dog. Ingested eggs hatch in the small intestine and oncospheres reach the liver via the portal vein. Here the embryos break out of the portal system and migrate through the liver parenchyma and towards the serosal surface for up to 30 days. They cause haemorrhagic tracts. The cysticerci develop subserosal from about 18 –28 days after infection. They reach maturity between 34 and 53 days and are found attached to the greater omentum, mesentery, liver and other serosal surfaces of abdominal organs. Mature cysticerci are up to 6 cm long and have a thin, long neck. They contain a single scolex. The life cycle is closed, if carnivores ingest cysticerci. The prepatent period in the dog is 51 days and dogs may remain infected for more than one year. The prevalence of infection in sheep is high. In some regions more than 80% of the slaughtered sheep and goats show cysticerci. Migration of young cysticerci through the liver parenchyma causes haemorrhagic and later fibrotic tracts.

Geographic distribution: World-wide

Symptoms: Infections in intermediate hosts are usually inapparent. Heavy infections may cause traumatic hepatitis when large numbers of larvae migrate through the liver. Death may result often due to haemorrhage. This may occasionally occur in young lambs and pigs. Animals during that time often show unthriftiness. Mature cysticerci in the peritoneal cavity usually cause no harm.

Significance: Losses occur through condemned livers and organs.

Diagnosis: This is based on finding the cysticerci at slaughter. Cysticerci are covered by a layer of serosa. If this layer is broken the typical thin-necked organism appears. The diagnosis antemortem is difficult. Serological tests may be unspecific. Haemorrhagic tracts may be seen in the liver when

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masses of larvae have passed through the parenchyma.

Therapy: This is not indicated in intermediate hosts. Dogs may be treated as described in *Echinococcus granulosus* (☞ CATTLE, ■ 4.2).

Prophylaxis: Removed cysticerci at slaughter should not be fed to dogs. Dogs should in general not be allowed to feed on condemned meat and organs.

(Figures 179, 180, 366)

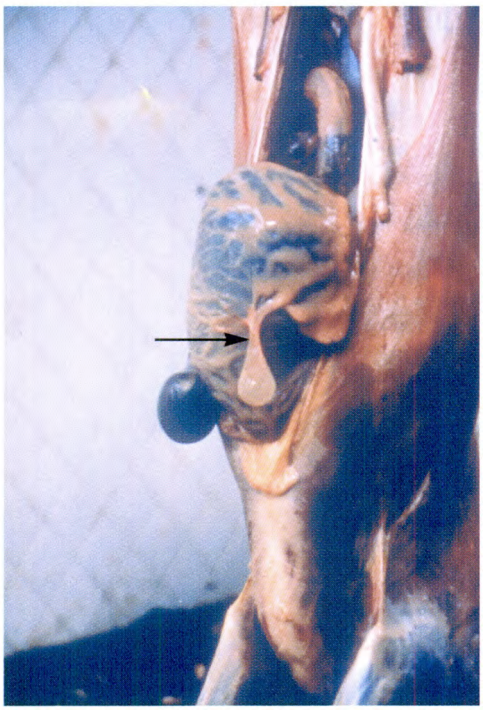

Fig. 366 *Cysticercus tenuicollis*; thin-necked cysts attached to the serosa [10]

- Nematoda found in the abdominal cavity

Setaria labiatopapillosa

Remarks: This parasite occurs in the peritoneal cavity of cattle, deer, giraffe and other ruminants. Sheep and goats may occasionally be abnormal hosts in which erratic microfilariae may invade the CNS and produce nervous symptoms. The diagnosis of microfilariae-associated (mainly *S. digitata*) neurological disturbances can only be made at necropsy by microscopical examination of CNS sections (☞ CATTLE, ■ 4.4).

4.5 Pancreas

HELMINTHS

- Trematoda found in the pancreas

Eurytrema pancreaticum

Pancreatic fluke

Location: Pancreatic ducts, bile ducts, occasionally duodenum

Hosts: Sheep, goat and cattle

Species description: These flukes are 8–16 mm long and 5–8.5 mm wide. The bodies are thick with large suckers. The eggs measure 40–50 × 23–34 µm. The life cycle is indirect with two intermediate hosts. Land snails of the genera *Bradybaena* and *Cathaica* serve as first intermediate hosts. Grasshoppers of different genera act as second intermediate hosts. Infective metacercariae develop in 3 weeks after infection of the grasshopper. Sheep and goats are infected by eating infected grasshoppers. After ingestion the immature flukes migrate via the pancreatic duct into the pancreas. The prepatent period in sheep and cattle is 80–100 days. Infection produces a catarrhal inflammation with destruction of the duct epithelium. Eggs may penetrate into the walls of the ducts causing granuloma. Occasionally fibrosis may produce atrophy of the parenchyma.

Geographic distribution: Eastern Asia, Brazil, Madagascar, Réunion

Symptoms: Severely infected animals may be poor in condition. Heavily infected sheep show chronic wasting condition and increased mortality.

Significance: This parasite is not a serious threat unless a herd is heavily infected.

Diagnosis: There are no obvious clinical signs. *Dicrocoelium*-like eggs appear in the faeces (☞ Cattle, 1).

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Therapy: The therapy is is difficult. Praziquantel (20 mg/kg, po. for 2 days) has been reported to be effective.

Prophylaxis: As for *Dicrocoelium*, the control of intermediate hosts is not feasible.

(Figure 367)

Fig. 367 *Eurytrema pancreaticum*; adult fluke (8–16 × 5–8.5 mm; stained) [4]

4.6 Central nervous system

PROTOZOA

Toxoplasma gondii

Remarks: Tissue cysts of this parasite may occasionally be found in the brain of sheep and cattle (☞ CATTLE, ■ 4.6). These cysts contain merozoites (6–8 µm long) and are infective for the final host (cat). Encephalomyelitis due to *T. gondii* infections may occur in sheep. Affected sheep have myelomalacia and schizonts appear in astrocytes (☞ SWINE, ■ 4.1).

Neospora caninum

Remarks: Thick-walled tissue cysts of this parasite may occasionally be found in the brain of sheep and cattle (☞ CATTLE, ■ 4.6).

HELMINTHS

- Cestoda cysts found in the central nervous system

Coenurus cerebralis

Larval stage of the canine tapeworm *Taenia multiceps* (syn. *Multiceps multiceps*) "gid", "stuggers", "sturdy"

Location: The larval stage, a coenurus, develops in the brain and spinal cord of sheep and goats. In the goat the cysts may also reach maturity in other organs, intramuscularly and subcutaneously.

Hosts: Sheep and goat; adult *Taenia multiceps* occurs in the small intestine of the dog, fox, coyote, hyaena and other Canidae.

Species description: Adult tapeworms in dogs are 40–100 cm in length. Eggs measure 29–37 µm in diameter. Larvae develop in the brain into a large fluid-containing cyst, measuring 5–6 cm in diameter. The cyst contains several hundred protoscolices invaginated in clusters on the cyst wall. *Coenurus* cysts cause increased intracranial pressure which is the cause of the neurological symptoms.

Geographic distribution: World-wide; in Africa *C. cerebralis* was reported from Kenya, Ethiopia, Sudan, Angola, Tchad, Zaire, Congo, Senegal, northern and southern Africa.

Symptoms: Ataxia, hypermetria, blindness, head deviation, stumbling and paralysis. In sheep, palpation of the skull caudal to the horn buds may reveal rarefaction of the skull.

Significance: *C. cerebralis* may account for remarkable losses among small ruminants in endemic areas. The disease must be differentiated from other conditions associated with nervous signs.

Diagnosis: Clinical symptoms are not specific. Diagnosis is often made at necropsy.

Therapy: The therapy is difficult. However, surgery to remove the cysts which are located on the surface of the brain, has a reasonable chance of success and may be justified in valuable animals.

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Prophylaxis: Dogs associated with livestock should not be fed the heads of infected animals and should be dewormed regularly. Praziquantel (5 mg/kg, po. or 5.7 mg/kg, sc. or im.), bunamidine hydrochloride (25–50 mg/kg, po.), nitroscanate (50 mg/kg, po.) and the combination of febantel/praziquantel/pyrantel are sufficiently effective against adult *Taenia multiceps* in dogs. Some benzimidazoles administered for a few days may eliminate both nematodes and cestodes: fenbendazole (50 mg/kg, po. during 3 days) or mebendazole (2 × 100 mg/animal/day during 5 subsequent days for dogs > 2 kg body weight).
(Figure 368)

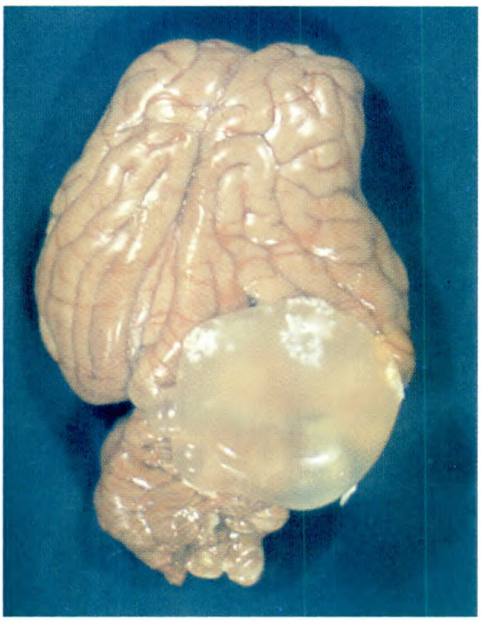

Fig. 368 *Coenurus cerebralis*; cyst in the brain of a sheep

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5 Stages on the body surface

5.1 Skin and coat

5.2 Eyes

5.1 Skin and coat

PROTOZOA

Besnoitia besnoiti (syn. *Sarcocystis besnoiti)*

Remarks: *Besnoitia* cysts are found in the dermis, subcutaneous tissue and fascia and in the laryngeal and nasal mucosae of cattle, goat, wildebeest, impala and kudu. Sheep may be experimental intermediate hosts. Definitive hosts are domestic and wild cats. *Besnoitia besnoiti* is a protozoan disease of the skin, subcutis, blood vessels and mucous membranes. These *Toxoplasma*-like organisms multiply in endothelial cells and produce characteristic large, thick-walled cysts filled with bradyzoites. The *Besnoitia* cysts may reach up to 600 µm in diameter (☞ CATTLE, ■ 5.1).

HELMINTHS

• Nematoda found in the skin

Onchocerca spp.

Remarks: Very little is known about filarial worms of the subcutaneous tissue of sheep and goats but there is some evidence that several species may occur. They seem to be of little significance and do not require control.

ARTHROPODS

Arthropods are divided into two main groups:

Arachnida
– *Ticks*
– *Mites*

Insecta
– *Lice*
– *Fleas*
– *Dipterida*

• Arachnida found in/on the skin

– **Ticks**

Sheep ticks are of great economic importance, but not to the extent that they are in cattle. These parasites cause tick worry, blood loss and damage at feeding sites. Wounds left from tick bites are susceptible to additional attack by blowflies. Infested sheep itch, bite and scratch causing self-inflicted wool damage and skin trauma which may become secondarily infected. Heavy tick burdens may result in anaemia, loss of appetite and weight loss (☞ Directly Noxious Effects). Some tick species cause tick paralysis (☞ Tick Toxicoses). Apart from the direct noxious effects ticks may act as vectors of a number of diseases in sheep such as louping ill, rickettsial tick-borne fever, Nairobi sheep disease, spirochaetosis, heartwater and Q fever. Ticks may therefore provoke disease in 3 different ways:

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- **Direct noxious effects**

In sheep and goats ticks of the following genera and species have a direct noxious effect (☞ CATTLE, ■ 5.1):

IXODIDAE ("hard ticks")
Amblyomma spp.
Boophilus spp.
Dermacentor spp.
Haemaphysalis spp.
Hyalomma spp.
Ixodes spp.
Rhipicephalus spp.

ARGASIDAE ("soft ticks")
Otobius megnini The spinose ear tick

- **Transmission of diseases**

Some of the tick-borne parasitic infections are as follows (☞ SHEEP AND GOATS, ■ 2; general aspects ☞ CATTLE, ■ 5.1).

Babesia ovis
Tropical babesiosis of small ruminants

Remarks: It is transmitted by *Rhipicephalus bursa* and *Rhipicephalus evertsi.*

Babesia motasi *(syn. Piroplasma motasi)*
European piroplasmosis of small ruminants

Remarks: It is transmitted by *Haemaphysalis* spp. (*H. punctata, H. sulcata* and *H. parva*), *Dermacentor* spp. and *Rhipicephalus bursa.*

Theileria hirci *(syn. T. lestoquardi)*
Malignant theileriosis of small ruminants

Remarks: It is transmitted by *Rhipicephalus bursa* and *Hyalomma anatolicum* and other species to be determined.

Theileria ovis Benign theileriosis of small ruminants

Remarks: It is transmitted by *Rhipicephalus bursa* and *Rhipicephalus evertsi.*

Anaplasma ovis Tropical anaplasmosis of small ruminants

Remarks: It is transmitted by *Rhipicephalus bursa* and *Rhipicephalus evertsi* in tropical Africa; transplacental infection occurs.

Eperythrozoon ovis

Remarks: This species occurs on the erythrocyte surface. It is transmitted by *Linognathus ovillus* and possibly also by ticks (*Hyalomma marginatum* and *Rhipicephalus bursa*).

Cowdria ruminantium Heartwater, Tyewde

Remarks: The infection is transmitted by *Amblyomma hebraeum* and *Amblyomma variegatum* (☞ CATTLE, ■ 2).

Ehrlichia ovina Tropical ehrlichiosis of small ruminants

Remarks: The parasite is transmitted by *Rhipicephalus bursa* in Mediterranean basin and *Rhipicephalus evertsi* in tropical Africa.

Ehrlichia phagocytophila European ehrlichiosis

Remarks: *Ixodes ricinus* transmits the disease by trans-stadial infection.

- **Tick Toxicosis**

Tick paralysis

Lambs and calves are particularly susceptible to tick paralysis. *Ixodes rubicundus* (the Karoo paralysis tick) mainly affects sheep and goats and *Rhipecephalus evertsi* causes the "spring lamb paralysis" in lambs but also in calves. Worldwide several species of the genera *Amblyomma, Dermacentor* and *Ixodes* are associated with tick paralysis in sheep (Table 15). This condition can be fatal within several days if the parasites are not removed. The paralysis is caused by toxins injected by female ticks while they suck blood. Cases of tick paralysis are reported from most regions

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of the world. The most common tick species which cause tick paralysis are listed in Table 15 (☞ CATTLE, ■ 5.1)
(Figures 369, 370)

Fig. 369 *Ixodes ricinus*; engorged female and male [4]

Fig. 370 *Haemaphysalis punctata*; female (3 × 2 mm) [4]

Table 15 The most common tick species causing tick paralysis in sheep

Tick species	Occurrence
Ixodes rubicundus	dry regions,southern Africa
Ixodes ricinus	Western Europe
Ixodes gibbosus	Mediterranean basin
Haemaphysalis punctata	Mediterranean basin
Haemaphysalis sulcata	Mediterranean basin
Rhipicehalus evertsi	East and southern Africa

Sweating sickness
It is mainly a disease of young calves but also sheep in eastern, central and southern Africa. The causative agents are certain strains of *Hyalomma truncatum* which produce an epitheliotropic toxin. Calf mortality may reach 70% (☞ Cattle, ■ 5.1).

General toxicosis
Toxicosis, associated with general disorders occurs with *Ornithodoros savignyi* (sand tampan) in young calves and lambs, especially when there are many bites. Animals show cutaneous oedema, haemorrhage, rapidly progressing weakness and prostration. Death can occur within 6 hours. Toxicosis may occur in recumbent animals, during rest (☞ Cattle, ■ 5.1).

- **Description of ticks affecting sheep and goats**

IXODIDAE ("hard ticks")

A general description of each genus is made in CATTLE, ■ 5.1.

Table 16 Hard tick genera which are important for small ruminants:

Tick species	Geographic distribution
Amblyomma spp.	Central and southern Africa
Boophilus spp.	Warm climates throughout the world
Dermacentor spp.	North and southern Africa
Haemaphysalis spp	Throughout Europe, Asia, Africa
Hyalomma spp.	Southern UIS, Iran, Palaearctic region
Ixodes spp.	North and southern Africa
Rhipicephalus spp.	Africa south of the equator

ARGASIDAE ("soft ticks")

Ornithodoros moubata The eyeless tampan

Remarks: This tick causes much trouble by feeding on sheep at their resting place in the pastures. It lives in the native huts and in the sand under trees where animals and human beings frequently seek shelter. In huts they live in cracks in the floor or under

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loose soil, from which they emerge at night to feed. In burrows they bury themselves in the earth lining, coming out to feed when a suitable host is available. Adult females lay batches of about 100 eggs and brood over the eggs in the sand. Development includes a larval stage which remains quiescent until it has moulted to the nymphal stage. Several nymphal stages are passed through and the nymphs, like the adults, attack their host for short periods to feed. This tampan sucks blood on its hosts. It is extremely resistant to both starvation and desiccation (survival time off the host up to 5 years).

Significance: In sheep *O. moubata* mainly causes irritation. It is also known to transmit the following diseases: *Borrelia duttoni* (relapsing fever of man), African swine fever, Q fever, *Borrelia anserina* and *Aegyptianella pullorum* in fowls.

(Figure 371)

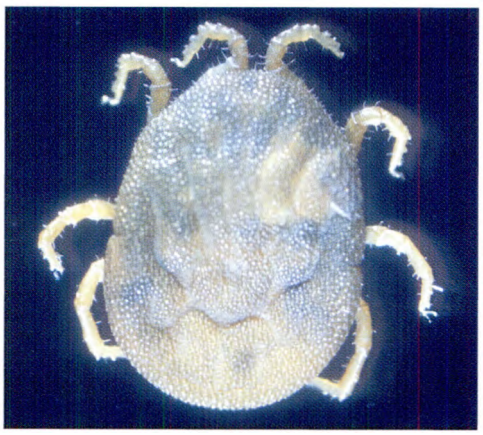

Fig. 371 *O. moubata*; dorsal view (8 mm long) [8]

Ornithodoros savignyi The sand tampan

Remarks: Its habits are similar to those of *O. moubata*. To the naked eye it is indistinguishable from the eyeless tampan. It occurs on man, camel, cattle, sheep and goats and many other animals (including birds). On standing cattle it feeds on the legs, especially, just above the hoofs.

Significance: *O. savignyi* is a major pest of domestic stock in areas in which it occurs. Its bites are extremely painful and it secretes toxins in its saliva that frequently cause death especially of calves, kids and lambs (tick toxicosis). Adult animals may also die from these toxic effects, especially when they have been attacked by large numbers of ticks. The sand tampan may cause severe allergic reactions in man.

Otobius megnini Spinose ear tick

Remarks: Larvae and nymphae feed deep in the external ear canal. Adults are non-parasitic and live on the ground. The spinose ear tick is a serious threat to sheep. Damage caused by the blood-sucking larvae and nymphs on the inside of the ears may result in marked debilitation, depressed appetite, restlessness and massive loss of weight in sheep. Weakness and anaemia may occur in heavy infestations. A waxy exudate is found in the ears (☞ CATTLE, ■ 5.1).

- **Tick control in sheep and goat**

(☞ CATTLE, ■ 5.1 THERAPY AND PROPHYLAXIS OF ECTOPARASITES [arachnids and insects])

– Mites

Psoroptes ovis The sheep scab mite

Location: Most areas of the body, especially those heavily wooled and haired such as shoulders, sides and back are affected.

Hosts: Sheep

Species description: The *Psoroptes* spp. occurring on domestic animals are all morphologically very much alike (☞ CATTLE, ■ 5.1). Mites pierce the skin to obtain lymph and cause an inflammatory reaction, resulting in the exudation of serum which coagulates on the skin surface. The irritation caused by the mites also makes the sheep bite and pull at the skin, thus aggravating the condition. In

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many countries sheep scab is a notifiable disease and controlled by the national veterinary authorities.

Geographic distribution: World-wide

Symptoms: Continuous scratching and rubbing occurs in affected flocks. Wool breaks may occur, leaving the skin exposed. Depression, loss of appetite, weight loss and death can occur in heavy infestations.

Significance: Psoroptic mange is highly contagious and spreads rapidly in flocks. Wool loss, weight loss and death may cause enormous losses throughout the world in sheep rearing enterprises.

Diagnosis: The diagnosis is based on identification of mites in skin scrapings.

Therapy: Ivermectin (200 µg/kg, sc.) is effective against psoroptic mange in sheep, although 2 injections with 7-day interval are required to eliminate mites. One injection is effective against *Sarcoptes* spp. and *Psorergates* spp. A single dipping in toxaphene (0.5–0.6%) eliminates all mites that affect sheep except *Psoroptes ovis*. Other approved dips effective against *Psoroptes ovis* and *Chorioptes ovis* are coumaphos (0.3%) and phosmet (0.2–0.25%), diazinon, fenvalerate and flumethrin. It is of fundamental importance that the entire flock is treated.

Prophylaxis: ☞ CATTLE, ■ 5.1 *Psoroptes bovis* (Figures 372, 373)

Fig. 372 infested with *Psoroptes ovis*; initial lesions on the back

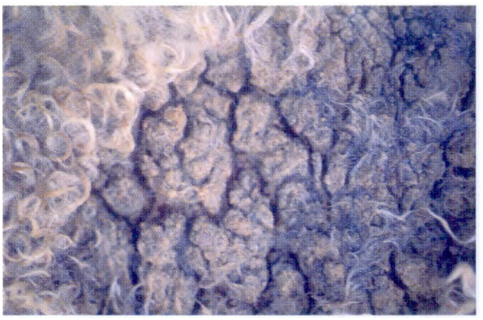

Fig. 373 Skin fissures caused by *Psoroptes ovis*; these wounds are often complicated by myiasis

Psoroptes caprae

Remarks: Some authors consider *P. caprae* to be a valid species of goats. Psoroptic mange of goats (ear mange of goats) especially of some breeds (Angora goats) in some parts of the world is considered to be *Psoroptes cuniculi*, which is known to infest the ears of domestic rabbits. Any of the acaricides approved for use as sheep dips will eliminate ear mange of goats.

Chorioptes ovis Chorioptic mange, "leg mange", "foot scab"

Location: Chorioptic mange is most often found on the hindlegs and between the toes, or on the scrotum of rams.

Hosts: Sheep

Species description: This is the most frequent type in sheep. General genus description is given in CATTLE, ■ 5.

Geographic distribution: World-wide

Symptoms: Infested sheep scratch and rub their feet. Localized scabs may be seen. The lesions are less severe and localized than in *Psoroptes ovis* infections.

Significance: Foot scab is not a major problem in sheep raising, but chronic infections of flocks may cause considerable indirect losses, due to restlessness, scratching, weight losses and secondary skin infections.

Diagnosis: The diagnosis is based on identification of mites in skin scrapings.

Therapy: ☞ above *Psoroptes ovis*

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Prophylaxis: ☞ above *Psoroptes ovis* (Figure 374)

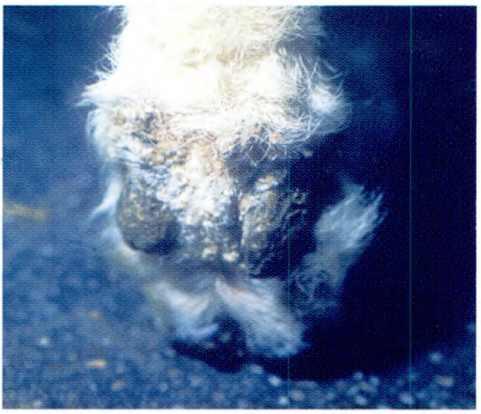

Fig. 374 Foot of a sheep infested with *Chorioptes ovis*; posterior view [15]

Sarcoptes ovis Sarcoptic mange

Location: This mite occurs mainly on non-wooly skin, starting usually at the head and face.

Hosts: Sheep

Species description: This species is rarely found. Genus description ☞ Cattle, ■ 5; *Sarcoptes bovis*. Female adults burrow tunnels in skin and lay eggs. All stages of sarcoptic mites are very susceptible to drying and can survive for only a few days off sheep.

Geographic distribution: World-wide

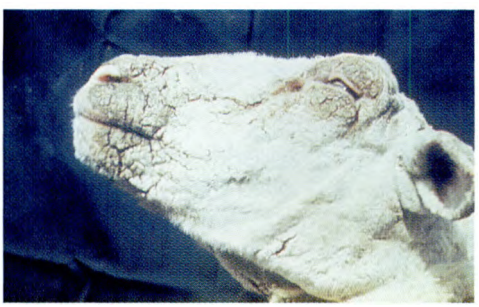

Fig. 375 Head of a sheep infested with *Sarcoptes ovis* [4]

Symptoms: Affected skin is thickened and crusts may be seen. Scratching and rubbing followed by wool loss may occur.

Significance: Sarcoptic mange is rarely a serious problem in wooled sheep.

Diagnosis: This is based on identification of mites in skin scrapings.

Therapy: ☞ above *Psoroptes ovis*

Prophylaxis: ☞ above *Psoroptes ovis* (Figure 375)

Psorergates ovis Itch mite, "Australian-itch"

Location: Skin all over the body

Hosts: Sheep

Species description: This is a very minute mite, one-third the size (189 × 189 µm) of psoroptic mites. The mite is spherical and has paired claws and legs which are arranged radially. Especially fine-wooled sheep are affected. The adult mites spread by contact and are most often transferred between shorn sheep.

Geographic distribution: Australia, USA, New Zealand, South Africa

Symptoms: These mites cause severe irritation, leading to rubbing and biting at the fleece of sheep. Newly infected animals show more signs than chronically infested animals.

Significance: Itch mite is a serious problem in Merino sheep in Australia, causing great losses in wool production due to fleece derangement and low wool quality and quantity. Itch mites are also of great significance in South Africa.

Diagnosis: This is difficult because of the small size of the mites. Microscopical examination of skin scrapings may reveal these small mites. Sodium hydroxide liquefies debris in scrapings and may be helpful to detect the mites.

Therapy: Ivermectin (200 µg/kg, sc.) is highly effective against *Psorergates ovis*. A single injection usually kills all the mites. Dipping and spraying (☞ above *Psoroptes ovis*), especially after shearing is an important control measure.

Prophylaxis: Yearly dipping after shearing for

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the control of the parasites will normally suppress the itch mite population. This will keep the infestation rate low but eradication is difficult to be achieved.
(Figure 376)

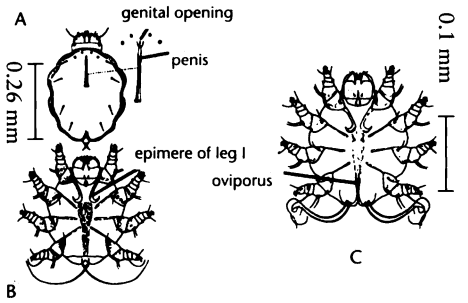

Fig. 376 *P. ovis* (schematic); (A) dorsal view of male with enlargement of penis and genital opening; (B) ventral view of a male; (C) ventral view of female [29]

Demodex spp. *(D. ovis* and *D. caprae)*
Demodectic mite

Location: Hair follicles and sebaceous glands of skin. Favoured areas are eyelid, prepuce of male and vulva of female. Often-affected areas are also skin of the neck, shoulder, thorax, and flank.
Hosts: Sheep and goat
Species description: The nodules produced by this cigar-shaped mite (genus description ☞ CATTLE, ■ 5.1) range in size from pinhead to hazelnut, contain a thick, waxy, greyish material that can easily be expressed. Numerous demodectic mites are found in this material. There is some evidence that *Demodex* are normally found in and on the healthy skin and that they may resume increased significance when the host animal is immunosuppressed.
Geographic distribution: World-wide
Symptoms: Nodules occur in the skin. The nodules of goats appear as cysts with mild inflammation in the surrounding tissue.
Significance: Demodectic mange in goats may cause considerable losses due to damage of the hides.
Diagnosis: Numerous demodectic mites are found in the contents of skin nodules. However, there is some evidence that *Demodex* may be present in normal healthy skin without apparent signs.
Therapy: There is no satisfactory treatment. Incision of the nodules and disinfection with tincture of iodine was described to give the best therapeutic results in valuable goats. Organophosphates may also be used.
Prophylaxis: Unknown
(Figure 377)

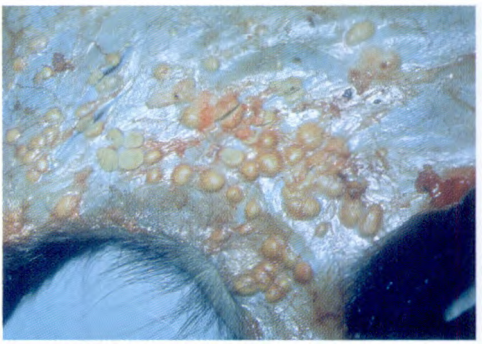

Fig. 377 *Demodex caprae* in subcutaneous nodules of a goat (0.5–1.5 cm in diameter) [4]

Raillietia spp. *(R. auris* and *R. caprae)*

Location: Middle and inner ear
Hosts: Sheep (*R. auris*), goat (*R. caprae*) and cattle
Species description: The mites feed on epidermal cells and wax but not on blood.
Geographic distribution: North America, Australia, East Africa, Europe.
Symptoms: Infestations are usually inapparent but otitis media and interna with nervous signs, including head shaking, head rotation to the affected side, circling, general incoordination, can be found in a progressive stage of the infestation.
Significance: *Raillietia* spp. infections can cause signs similar to those found in other CNS infections and should therefore be excluded.

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Diagnosis: It is almost impossible to diagnose *Raillietia* spp. antemortem; demonstration of the mites in the middle or inner ear at necropsy.

Therapy: Ivermectin may eliminate the mites but the diagnosis is difficult. Acaricides, applied topically, may also be effective.

Prophylaxis: Unknown (☞ CATTLE, ■ 5.1)

- **Insecta found on the skin**

– **Lice**

MALLOPHAGA Chewing lice

Lepikentron ovis Ovine chewing louse **and** ***Bovicola*** (syn. *Damalinia*) ***caprae*** Caprine chewing louse

Hosts: Sheep and goat

Location: Skin of the neck, withers and root of the tail

Species description: *Lepikentron ovis* is a small, wingless insect, 1–2 mm long. The head is broad and flat with mouth parts adapted to chewing. *Bovicola caprae* parasitises the common goat and *Bovicola crassipes* and *Bovicola limbatus* are found mainly on Angora goats.

Geographic distribution: World-wide

Symptoms: Infested sheep bite, scratch and show a poor body condition. Louse worry may produce low fleece quality.

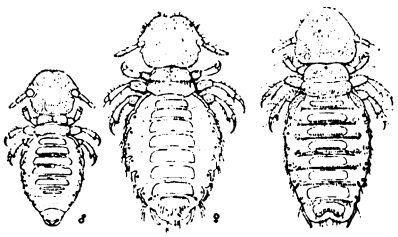

Fig. 378 Chewing lice (Mallophaga) of sheep and goats; (left) *Bovicola caprae* male (1 mm) and female (1.5–2 mm) and (right) *Bovicola bovis* (1.5–2 mm) [35]

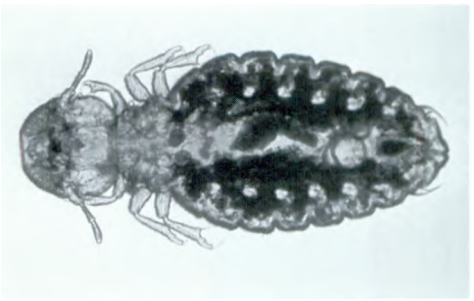

Fig. 379 *Bovicola caprae* (1–2mm) [4]

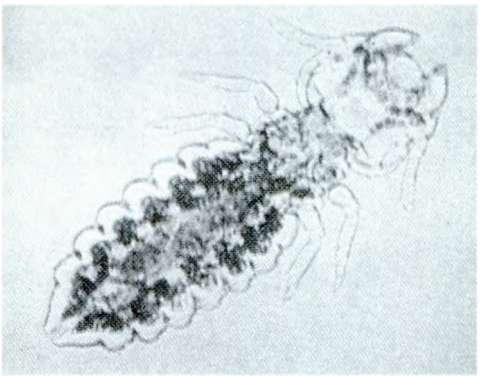

Fig. 380 *Lepikentron ovis* (1–2 mm) [32]

Significance: Louse worry may cause decreased food intake, reduced wool growth and the overall profitability may significantly be reduced.

Diagnosis: Nits and adults may be seen in fleece and on skin.

Therapy: ☞ CATTLE, ■ 5.1 THERAPY OF CHEWING LICE OF CATTLE

Prophylaxis: ☞ CATTLE, ■ 5.1

(Figures 378, 379, 380)

ANOPLURA Blood sucking lice of sheep and goats

Linognathus ovillus The sheep body louse, face louse

Remarks: It occurs chiefly on the face of sheep in Australia, New Zealand and probably many other parts of the world (☞ CATTLE, ■ 5.1).

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Linognathus pedalis The sheep foot louse

Remarks: It occurs on legs, belly and feet where no wool exists.

Linognathus africanus The African sheep louse, the blue louse

Remarks: It occurs mainly on the face of sheep.

Linognathus stenopsis The goat sucking louse

Remarks: It occurs mainly on goats. (Figure 381)

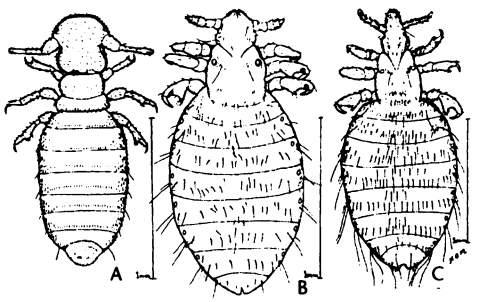

Fig. 381 Sheep lice; (A) *Lepikentron ovis*, (B) *Linognathus pedalis* and (C) *Linognathus africanus* [3]

- **General features of blood sucking lice of sheep and goats**

Species description: These are wingless insects with mouth parts adapted for piercing skin and sucking blood. The head is long and narrow. The entire life cycle is spent on the host. Lice may survive in warm environments for up to 25 days in the absence of their host.

Geographic distribution: *Linognathus* spp. occur in many parts of the world.

Symptoms: Scratching, rubbing and biting are seen in heavy infestations. Wool breaks and general unthriftiness, matted, dull fleece with tufts of loose wool may indicate lice infestation.

Significance: Heavily infested animals have markedly reduced weight gain and wool growth.

Diagnosis: Presence of nits on wool and adult lice on skin.

Therapy: ☞ ■ CATTLE, 5.1 THERAPY OF SUCKING LICE OF CATTLE p. 129

Prophylaxis: This is difficult wherever direct contact between the animals of the herd is possible. If sheep and goats are kept in sheds these may also be fumigated.

– Fleas

No fleas have been found on the body surface of sheeps and goats.

– Dipterida

CULICIDAE Mosquitoes

Remarks: With a single pair of wings, they belong, like flies, to the order Diptera. The main genera are *Anopheles, Culex* and *Aedes*. They are important vectors of diseases of sheep (e.g. Rift Valley fever, Lumpy skin disease, Blue tongue) (☞ CATTLE, ■ 5.1).

SIMULIIDAE

Simulium spp. Black flies, midges

Remarks: Simulids cause severe irritation to livestock when they occur in large numbers and herds and flocks will stampede, often with disastrous results. Bites are inflicted on all parts of the body, giving rise to vesicles which burst exposing the underlying flesh. Skin wounds with secondary infections and myiasis may result. Black flies cause severe irritation of livestock and certain areas of the tropics are rendered uninhabitable by simulids. (☞ Cattle, ■ 5.1)

CERATOPOGONIDAE

Culicoides spp. Biting midges

Remarks: Very small size (1–3 mm long). Adult female midges attack cattle, sheep, poultry, horse, man and other species,

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causing marked irritation by penetration of the skin with their proboscis. The bites cause intense itching. Biting midges cause massive irritation of livestock and transmit the Blue tongue virus to sheep (☞ CATTLE, ■ 5.1).

TABANIDAE

Tabanus spp., *Haematopota* spp. and *Chrysops* spp. Horse flies

Remarks: Several species of horse flies attack sheep and goats. They may cause irritation (stampeding of flocks may result) and transmit a number of diseases to livestock (besnoitiosis, anaplasmosis, trypanosomosis and anthrax; ☞ ■ CATTLE, 5.1).

MUSCIDAE

Musca spp., *Lyperosia* spp. and *Haematobia* spp. Muscid flies

Remarks: Muscid flies annoy livestock especially during the summer or rainy season. They swarm around farm livestock. The resulting irritation is incessant and much of the energy of the animals is lost to defend against fly attacks. In addition to the nuisance the flies often carry pathogens on their feet and bodies. Some also act as intermediate hosts for other parasites. Two genera need to receive special attention: *Musca* and *Haematobia*.

Musca domestica Common house fly

Remarks: This is a non-biting muscid. It is attracted to wounds and other moist parts of the body, especially the eyes where it may provoke an ulcerative dermatitis. *M. domestica* is known to transfer pathogenic bacteria mechanically from one wound to another (☞ CATTLE, ■ 5.1).

Haematobia minuta and other *Haematobia* spp. Horn flies, buffalo flies

Remarks: These are biting muscids. The adults of this fly live almost permanently on cattle, buffalo sheep and other animals. They congregate on the back where their bites cause severe irritation so that the cattle rub themselves raw. *Haematobia* spp. cause intense worry and irritation to animals, the bite being very painful. Serious blood loss may occur when large numbers attack and loss of condition, reduced performance is a common result. The flies cause sores at the basis of the horns, on the poll, ears, neck, withers and tail root (☞ CATTLE, ■ 5.1).

Stomoxys calcitrans Stable fly

Remarks: *S. calcitrans* also belongs to the muscids. It attacks almost all livestock species. It is a biting muscid and occurs worldwide. Both sexes of this fly are bloodsuckers and can become extremely irritating pests of man and domestic animals. Their salivary secretions cause toxic reactions with an immunosuppressive effect, rendering the host more susceptible to diseases. *S. calcitrans* acts as vector of *Trypanosoma evansi*, *Anthrax*, *Dermatophilus congolensis*, the "lumpy wool" in sheep and other pathogens (☞ ■ CATTLE, 5.1). Its painful bite causes intense worry and irritation to animals. It may produce toxic reactions and immunosuppression. Blood loss may be marked following continuous, heavy attacks.

GLOSSINIDAE

Glossina spp. Tsetse flies

Remarks: Tsetse flies are active during the day time and hunt by sight and smell. Both sexes suck blood and are equally capable of transmitting trypanosomes. Trypanosomosis in domestic animals is caused by *T. congolense*, *T. vivax*, *T. brucei* and *T. simiae* (☞ CATTLE, ■ 5.1).

Oestrus ovis Sheep nasal bot, nasal myiasis (☞ SHEEP AND GOATS, ■ 4.3).

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CALLIPHORIDAE The blowflies and their allies

They are highly important in many domestic animal species and man. The adults are free-living and the larvae are parasitic maggots which develop in the tissue of their host, causing a condition called myiasis. The larvae may be laid into preexisting wounds. Myiasis is often a secondary skin problem. Calliphoridae can be divided into groups: **metallic** and **non-metallic flies.**

Metallic flies are green, blue or purplish coloured and belong to the genera *Chrysomya, Lucilia* and *Calliphora* (blowflies, bottle flies).

***Lucilia cuprina* and other *Lucilia* spp.**
Green-bottle or copper-bottle flies

Remarks: *L. cuprina* is the predominant cause of sheep blowfly strike in South Africa and Australia. It causes myiasis in sheep. It is the most important primary blowfly initiating strikes on living sheep. Sheep are only attacked by primary blowflies if moisture is present in the fleece with resulting bacterial decomposition of the wool and superficial skin layers known as "fleece-rot". The odour arising from such processes attracts the primary blowflies, which liquefy the tissue and extend the process. The larvae of the primary blowflies, which initiate the attack, create conditions attractive to secondary blowflies.
(Figures 382, 383)

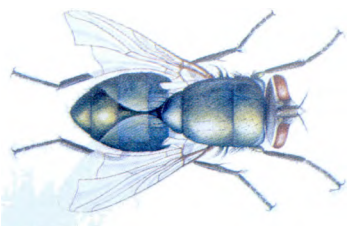

Fig. 382 *Lucilia cuprina*; adult (8–10 mm) [27]

Fig. 383 *Lucilia cuprina*; praepupae in a skin wound of a sheep (10–20 mm)

Lucilia sericata Green bottle fly

Remarks: *L. sericata* is the predominant cause of sheep blowfly strike world-wide. *Lucilia* spp. lay eggs in areas of skin with constant moisture (the breech and tail). *Lucilia* spp. are the first flies to strike living sheep. Prolonged wet weather may cause

Fig. 384 *Lucilia sericata*; adult (8–9 mm; left) and pupa, larva, eggs (right) [4]

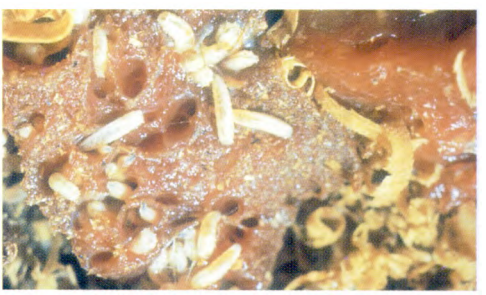

Fig. 385 *Lucilia sericata*; larvae (1.5–2 cm) in a skin wound

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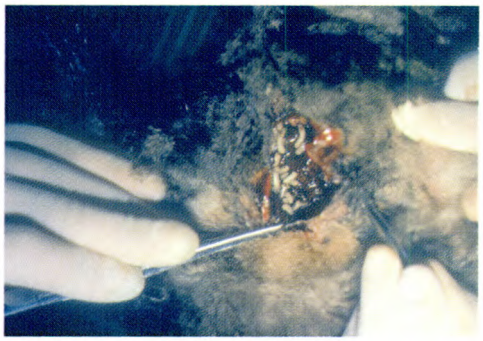

Fig. 386 *Lucilia sericata*; myiasis of a skin wound [11]

Lucilia to strike sheep in the wool of the back. These flies are responsible for more losses than any other single parasite of sheep.
(Figures 384, 385, 386)

Chrysomya chloropyga Green-tailed blowfly

Remarks: This is probably the second important primary blowfly of sheep in South Africa. *Chrysomya marginalis* may also act as a primary blowfly.
(Figure 387)

Fig. 387 *Chrysomya chloropyga*; adult (10–12 mm) [10]

***Chrysomya albiceps* and other *Chrysomya* spp.**
(Figure 388)

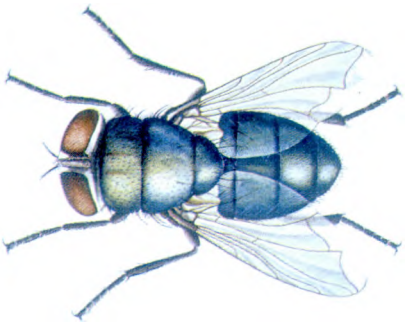

Fig. 388 *Chrysomya albiceps* [27]

***Calliphora* spp.** Bluebottle flies

Remarks: Large flies (12 mm) metallic blue in colour with stout bodies. Adult flies attack existing skin wounds, e.g. from castration, tail docking or wounds affected by primary blowflies. They may extend these wounds in which bacterial infections have already started. They cause severe myiasis. *Calliphora* blowflies may kill sheep and cause great losses world-wide.

Callitroga hominivorax (syn. *Cochliomyia hominivorax*) "American screwworm"

Remarks: *C. hominivorax* has recently been found to occur also in North Africa. Cattle, pigs and equines suffer most frequently, but other animals, including fowls and even man may also be affected. Pathology is essentially the same as in *C. bezziana* (☞ CATTLE, ■ 5.1).

Chrysomyia rufifacies Screwworm fly, carrion fly

Remarks: Flies are 8–10 mm long and bluish-green. Larvae are up to 14 mm long with thorn-like spines. This blowfly strikes sheep after primary infestations which are established by *Lucilia* spp. and *Calliphora* spp. They may weaken and kill sheep. If sheep are struck by *Chrysomyia* spp., death will usually follow.

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They may expand the pre-existing skin lesions enormously. The wound is infected by secondary bacterial invaders and is usually foul-smelling. Resistance of *Chrysomyia* to several insecticides has been reported.

The non-metallic flies are dull grey, yellow-brown or black and belong to the genera *Wohlfartia* and *Sarcophaga* (flesh-flies).

Sarcophaga haemorrhoidalis Red-tailed flesh-fly

Remarks: The fly is very common in Africa south of the Sahara and is frequently found around human habitations. Flies of this species are larviparous and may lay their larvae in wounds or sores although larvae may also be deposited on faeces, carrion or fresh meat. Several animal species and man may be affected. The fly may cause myiasis, especially in sheep.

Wohlfartia magnifica Old World flesh-fly

Remarks: It occurs in North Africa. The fly attacks man and other animals. The larvae may be deposited into the external ear of man or in sores around the eyes.

Cordylobia anthropophaga Tumbu fly or "skin maggot fly"

Remarks: The fly is widely distributed in Africa south of the Sahara. It produces myiasis in man, dogs and domestic animals. Eggs are laid around places where animals lie. After hatching the larvae penetrate the skin of the host, producing a boil of about 1 cm across with a hole in the centre. When mature the larvae wriggle out of the hole and pupate on the ground. Tumbu boils are found commonly on the ventral parts of the body but also on any other area of the body.

HIPPOBOSCIDAE The louse flies

Melophagus ovinus Sheep ked

Location: Skin on the neck, shoulders and belly
Hosts: Sheep
Species description: The sheep ked is a wingless fly living permanently on sheep. The legs are strong with claws at the end. Adult females deposit larvae on wool using an adhesive material. Larvae remain in place and molt to the pupal stage. Pupae last 19–23 days in summer and 36 days in winter. These pupae are resistant to treatment. The ked is a blood sucker and can cause anaemia if it is present in high numbers. They cause severe irritation so that infested sheep rub, bite and scratch themselves continuously. The piercing mouth parts of keds open wounds susceptible to further bacterial and parasitic (myiasis) infections. *Trypanosoma melophagium* is a non-pathogenic trypanosome which infects sheep. It belongs to the Stercoraria and is transmitted by the sheep ked, *Melophagus ovinus*. Infection occurs by contamination of the skin and if a ked is eaten by the sheep, the metacyclic stages penetrate the buccal mucosa.
Geographic distribution: World-wide
Symptoms: Intense itching from irritation causes sheep to rub, bite and scratch themselves and tearing the fleece. Heavy infestation may cause anaemia.
Significance: The sheep ked causes marked irritation with reduced grazing and fleece quality. It may further cause anaemia when present in high numbers.
Diagnosis: Adults and pupae may be seen on sheep.
Therapy: Insecticides which are also used to treat lice infestations may be applied. Dips, sprays, pour-on formulations may be used. Organophosphates and pyrethroids applied as dips, sprays or pour on are highly effective. (☞ CATTLE, ■ 5.1 THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 141.

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Prophylaxis: Shearing removes pupae and adults and should be carried out before lambing. A subsequent insecticidal treatment may eliminate the remaining ked (☞ CATTLE, ■ 5.1 THERAPY AND PROHYLAXIS OF ECTOPARASITES p. 141).

(Figure 389)

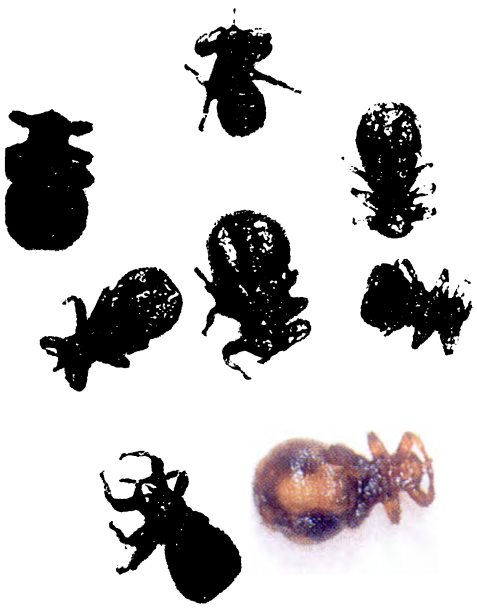

Fig. 389 *Melophagus ovinus* (3–5 mm) [32]

5.2 Eyes

Thelazia rhodesi Eyeworm

(☞ CATTLE, ■ 5.2)

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IV

Parasites of Horses and Donkeys

CONTENTS

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PROTOZOA

Eimeria leuckarti (syn. *Globidium leuckarti*)
Globidiosis

Location: Small intestine
Hosts: Horse and donkey
Species description: *E. leuckarti* oocyst is one of the largest of the genus *Eimeria* (80–88 × 55–59 µm) oval, thick wall, dark brown, distinct micropyle. Sporulation time is 20–22 days at 20°C.
Geographic distribution: World-wide
Symptoms: Acute and chronic diarrhoea
Significance: Little is known but serious diarrhoeic episodes have been attributed to massive infection with this parasite mainly in foals.
Diagnosis: *E. leuckarti* is too dense to rise in the flotation fluid usually used in the diagnostic laboratory and must be looked for by the sedimentation technique.
Therapy: Sulfonamids such as sulfadimidine (sulfamethazine, 220 mg/kg, po. or iv.), sulfadimethoxine (55 mg/kg, po.), sulfathiazole (66 mg/kg, po.) and the poorly soluble sulfaguanidine can be used against globidiosis.
Prophylaxis: Regular change of the feeding and holding area or disinfection of the stable reduces the infection risk.
(Figure 390, Table 1)

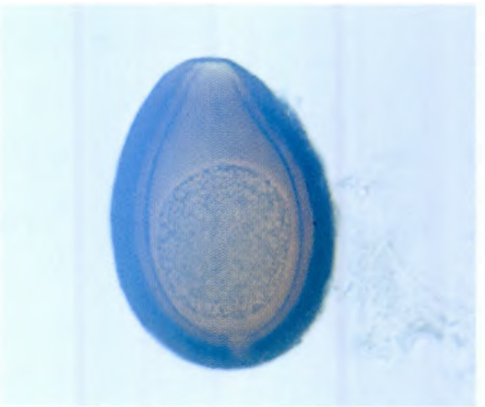

Fig. 390 Oocyst of *Eimeria leuckarti* (80–88 × 55–59 µm) [4]

Cryptosporidium parvum

Remarks: This species has only been found in immunodeficient foals (☞ CATTLE, ■ 1).

PROTOZOA
(Figure 391)

- Trematoda eggs found in the faeces and adult trematodes living in the gastrointestinal tract

Gastrodiscus aegyptiacus
Equine intestinal fluke

Location: Colon and small intestine
Hosts: Equine, pig and warthog
Species description: *G. aegyptiacus* belongs to the family Paramphistomatidae. Intermediate hosts are snails of the genus *Bulinus* and *Cleopatra*. Infection is acquired by eating metacercariae with vegetation.
Geographic distribution: Throughout Africa and India

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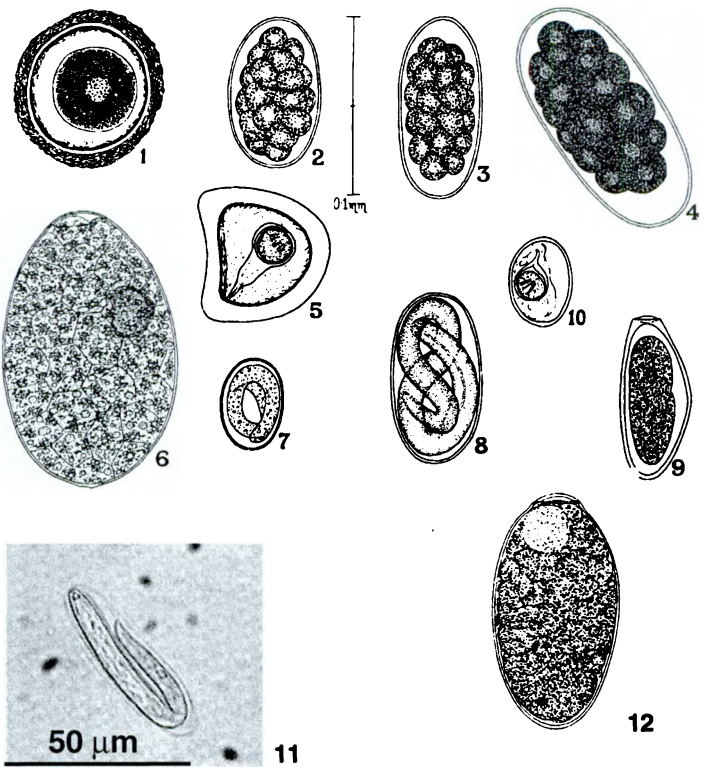

Fig. 391 Eggs of helminths found in equines [3]:

1 *Parascaris equorum*
2 *Strongylus* spp.
3 *Trichonema* spp.
4 *Triodontophorus tenuicollis*
5 *Anoplocephala* spp.
6 *Gastrodiscus aegyptiacus*
7 *Strongyloides westeri*
8 *Dictyocaulus arnfieldi*
9 *Oxyuris equi*
10 *Paranoplocephala mamillana*
11 *Habronema* spp.
12 *Fasciola hepatica*

Symptoms: Heavy infections are accompanied by diarrhoea, anaemia, oedema, emaciation and marked weakness. With the usual chronic mild infection there is no apparent effect on the host.

Significance: Adults have a relatively low level of pathogenicity. High numbers of immature flukes can cause severe problems. High numbers of *G. aegyptiacus* were found in horses in Central Africa.

Diagnosis: The diagnosis is based on the presence of eggs or immature flukes in the fluid faeces, and history of being kept on marshy pastures.

Therapy: The following compounds are effective against trematodes and can be considered for treatment: oxyclozanide, diamphenethide, rafoxanide, nitroxynil, albendazole, closantel, triclabendazole, netobimin and clorsulon. Some of these are active against immature flukes: diamphenethide (100 mg/kg), nitroxynil (15 mg/kg), closantel (10 mg/kg), triclabendazole (10 mg/kg) and clorsulon (7 mg/kg).

Prophylaxis: In addition to treatment of infected horses, wet pastures and swamps should be avoided. Horses should be watered with fresh water at bore holes.

(Figure 392)

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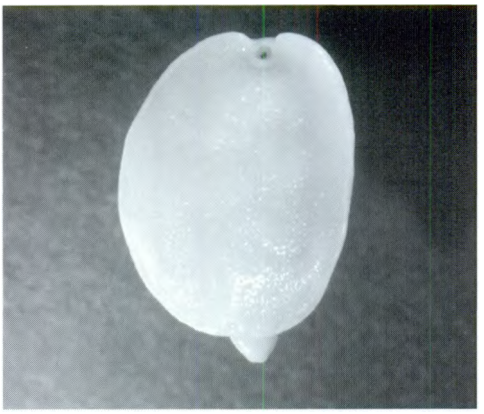

Fig. 392 *Gastrodiscus aegyptiacus*; ventral view (9–17 mm long)

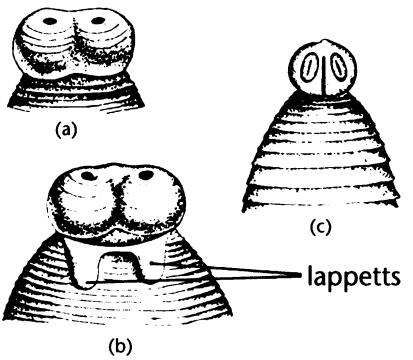

Fig. 393 Anterior end of equine tapeworms (schematic): *Anoplocephala magna* (a), *A. perfoliata* (b) and *Paranoplocephala mamillana* (c) [5]

Schistosoma bovis and *Schistosoma mattheei* Blood flukes

Location: Adult flukes in the mesenteric and portal veins, eggs occur in the faeces. (☞ HORSES AND DONKEYS, ■ 2)

- **Cestoda eggs found in the faeces and adult cestodes living in the gastrointestinal tract**

Anoplocephala magna Equine tapeworm

Location: Small intestine
Hosts: Horse and donkey
Species description: *A. magna* is the largest tapeworm of equines. It is up to 80 cm long and 2 cm wide and has a typical tapeworm shape.
Significance: *A. magna* in high numbers can cause a catarrhal or haemorrhagic enteritis.
(Figures 393, 394, 395, 396, 397)

Characteristics	*A. magna*	*A.perfoliata*	*P.mamillana*
Length	80 cm (long)	8 cm (short)	0.6–5.0 cm (minute)
Width	2 cm	0.8–1.4 cm	0.5 cm
Sucker	Round	Round	Slit-like
Ear-shaped lappets posterior to suckers	Absent	Present	Absent

Fig. 394 Description of equine tapeworms [5]

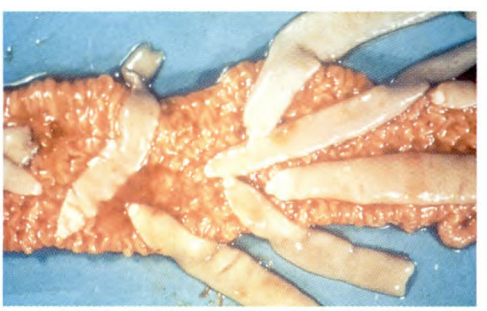

Fig. 395 *Anoplocephala magna*; adult tapeworms in the small intestine of a horse [15]

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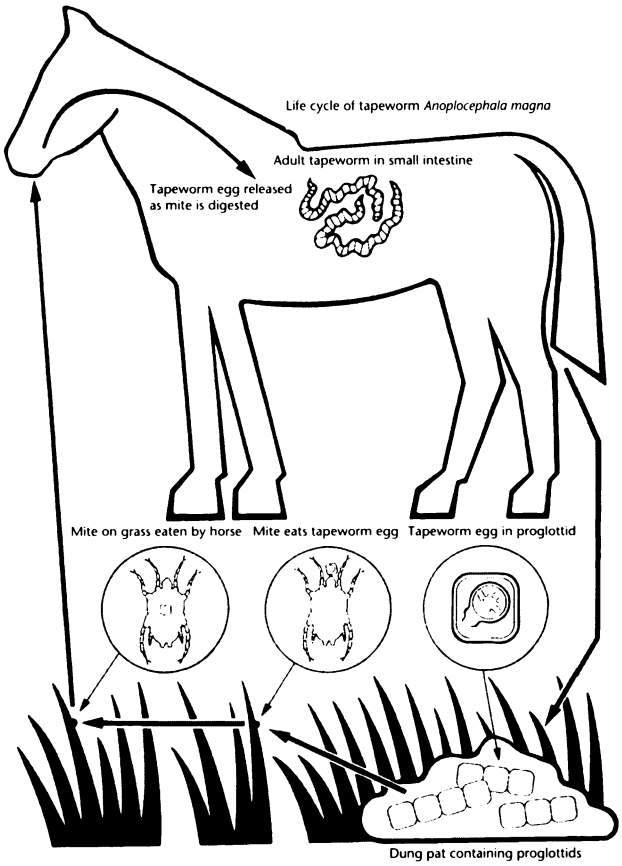

Fig. 396 Life cycle of equine tapeworms [36]

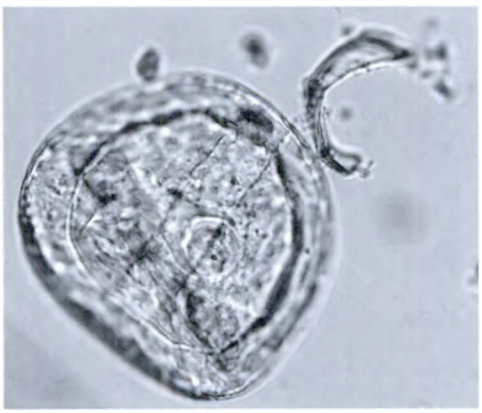

Fig. 397 Egg of *Anoplocephala* spp. (70–80 µm)

Anoplocephala perfoliata

Tapeworm of the ileocaecal-valve

Location: Large intestine, ileocaecal valve (found on either side)

Hosts: Horse and donkey

Species description: *A. perfoliata* varies from a few mm to 8 cm in length (some authors indicate length up to 30 cm) and looks at first sight more like a trematode. Closer inspection shows that the body is segmented, but the proglottids (segments) are crowded together. The scolex of *A. perfoliata* differs from the others in that there are prominent swellings (lappets) behind the suckers (Figure 393).

Significance: *A. perfoliata* normally causes little clinical effect but the site of attachment is often inflamed and ulcerated. Partial

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occlusion of the ileocaecal orifice may cause colic.
(Figure 398)

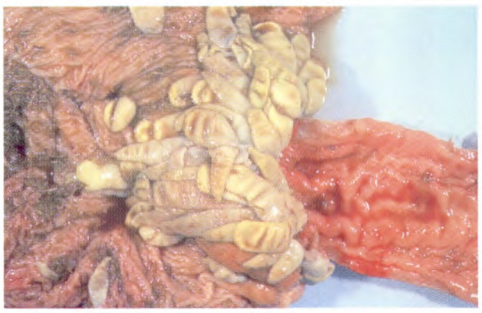

Fig. 398 *Anoplocephala perfoliata*; adult tapeworms attached to the ileocaecal valve

Paranoplocephala mamillana
Dwarf equine tapeworm

Location: Small intestine (jejunum, ileum) rarely stomach
Hosts: Horse
Species description: It is the smallest of the three equine tapeworms with a length of only 4 cm.
Significance: *P. mamillana* is seldom responsible for ill health.
(Figures 391, 393, 399)

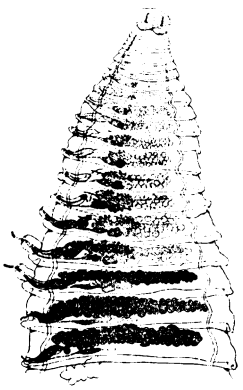

Fig. 399 *Paranoplocephala mamillana*; head and immature proglottids [9]

- **General features of *Anophocephala magna*, *A. perfoliata* and *Paranoplocephala mamillana***

Species description: Life cycle: Oribatid mites are intermediate hosts of all three species. Infection occurs via ingestion of infected mites. Adult tapeworms develop within 4 to 6 weeks after infection (Figure 396).
Geographic distribution: World-wide
Symptoms: Light infections are not apparent. Large numbers cause haemorrhagic or ulcerative enteritis, seen especially with *A. magna*. Fatal intestinal blockage may be caused by *A. perfoliata*, clustering at the ileocaecal junction. Unthriftiness, colic and diarrhoea may be caused by heavy infections.
Diagnosis: Identification of square, thick-shelled eggs containing the pyriform apparatus or segments in the faeces, or the presence of adult worms at necropsy.
Therapy: Anoplocephalidae are known to be relatively resistant to treatment. However, some of the modern anthelmintics, at increased dosage rates, are partially effective against equine tapeworms. Pyrantel pamoat (13.2 mg/kg, po.) at twice the label dosage is effective against *A. perfoliata*. Niclosamide (88 mg/kg, po.), fenbendazole (3 × 10 mg/kg, po.), resorantel (65 mg/kg, po.) and dichlorphen (20 mg/kg, po.), bithionol (7 mg/kg, po.) or mebendazole (20 mg/kg, po.) are safe and can also be used to treat equine tapeworms.
Prophylaxis: Prophylaxis is difficult as oribatid mites occur everywhere with densities of up to 20,000 per m^2.

- Nematoda eggs found in the faeces, adult nematodes living in the gastrointestinal tract and first-stage larvae of *Dictyocaulus arnfieldi* and *Probsmayria vivipara*

Gongylonema pulchrum Gullet worm

Location: Mucosa of the oesophagus
Hosts: Horse, donkey and ruminants
Species description: *G. pulchrum* lies embed-

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ded in the oesophageal mucosa in a zigzag fashion and reaches 14.5 cm in length. Eggs are passed in the faeces and hatch after being eaten by dung beetles in which the larvae develop to the infective stage. Infection of the horse occurs by eating the infected beetles (☞ CATTLE, 1).

Geographic distribution: World-wide

Symptoms: Generally inapparent infections although chronic irritation of the mucosa occurs.

Significance: *G. pulchrum* is of little clinical importance.

Diagnosis: Eggs in the faeces or adults are seen at necropsy in longitudinal rows of circular raised thickenings on the worm's forward end.

Therapy: Not indicated

Prophylaxis: Manure removal and normal sanitary measures are sufficient to reduce the infection risk.

Parascaris equorum Horse roundworm

Location: Small intestine

Hosts: Horse and donkey

Species description: Typical direct nematode life cycle. Within the egg the second-stage larvae develop to infectivity on pasture in 6 weeks. When swallowed, larvae hatch and penetrate the intestinal wall and are carried by the blood to the lungs, where they migrate up to the trachea. They are then coughed up and swallowed again and mature in the small intestine. *P. equorum* are whitish worms up to 30 cm long with 3 prominent lips. The principal source of infection for young foals is contamination of pasture, paddock or stall with eggs from foals of the previous year.

Geographic distribution: World-wide

Symptoms: Adult worms in heavy infections can cause bile duct and intestinal obstructions and occasionally gut perforation. Damage is more pronounced in foals. Acute parascariasis is also accompanied by severe enteritis, resulting in alternating constipation and foul-smelling diarrhoea. Large numbers of larvae breaking into the lungs cause a "summer cold" with coughing, fever and anorexia. Mature horses rarely show clinical signs, as previous infections confer good resistance.

Significance: Highly important, mostly in foals younger than 6 months in which parascariosis causes severe debilitation, retarded growth and even death.

Diagnosis: Identification of round, pitted, thick-walled eggs in the faeces.

Therapy: Piperazine (88 mg/kg, po.) and thiabendazole (100 mg/kg, po.) are effective against adult worms. Cambendazole (20 mg/kg), febantel (6 mg/kg), fenbendazole (7.5 mg/kg), mebendazole (10 mg/kg), pyrantel pamoat (19 mg/kg) and ivermectin (0.2 mg/kg) for oral administration are effective against adult and larval stages of *P. equorum*.

Prophylaxis: As foals become infected with *P. equorum* soon after birth, treatment and/or prophylaxis should start when foals are about 8 weeks old and repeated at 6–8- week intervals, until they are yearlings.

(Figures 400, 401)

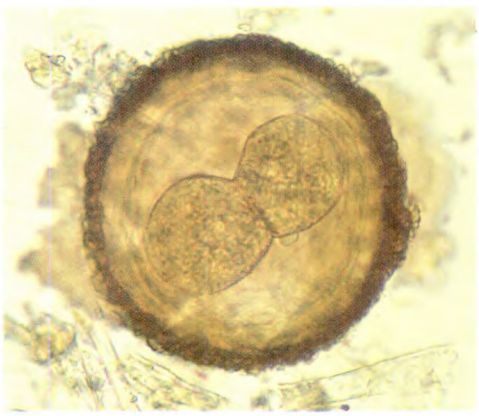

Fig. 400 Egg of *Parascaris equorum* (90–100 µm in diameter), partially developed

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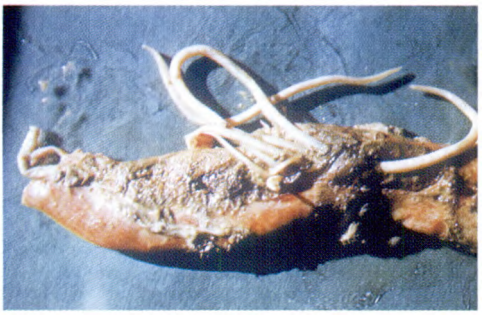

Fig. 401 *Parascaris equorum*; adult worms (males are 15–28 cm long, females are 16–50 cm long) found in the small intestine of a foal at necropsy

Strongyloides westeri

Equine intestinal threadworm

Location: Small intestine

Hosts: Horse and donkey

Species description: *S. westeri* is a hair-like, 8–9 mm long nematode. Only the parthenogenic females are parasitic. Free-living males and females reproduce sexually outside the host. Infection is acquired by ingestion of larvae, skin penetration or via the milk of the mare. Ingestion of larvae and skin penetration involves migration to the lungs. There are two possible ways of development, a homogonic cycle involving adult females in the host producing eggs that do not require fertilisation to develop. These eggs are passed in the faeces and then develop to infective third-stage larvae.

In the heterogonic cycle adult threadworms in the intestine lay eggs which develop into a different type of larvae. These larvae can develop to adult males and females which can live outside the host. The fertilized eggs of this type yield in infective larvae wich are ingested by the host. Migrating larvae can cross the mammary glands to infect nursing foals.

Geographic distribution: World-wide

Symptoms: Foals with heavy burdens show acute diarrhoea, weakness and emaciation. Older animals may harbour large burdens without clinical manifestations. Migration of *S. westeri* larvae through the lungs can cause severe haemorrhage and respiratory distress. Skin penetration may result in irritation and dermatitis.

Significance: Highly important in neonatal foals. Severe diarrhoea which can be so severe as to cause death in foals. The most important route of transmission is galactogenic.

Diagnosis: Identification of thin-shelled eggs (45 × 38 µm) containing a larva.

Therapy: Cambendazole (20 mg/kg, po.), thiabendazole (75 mg/kg, po.), ivermectin (0.2 mg/kg), fenbendazole at increased dosage rate (50 mg/kg, po.), oxibendazole (15 mg/kg, po.) and other benzimidazoles generally at increased dosage rates.

Prophylaxis: Reduction of densities of the free-living larvae by removal of the faeces and provision of dry quarters. Pre-foaling treatments to the mare reduce transmammary infection.

(Figures 402, 403, 404)

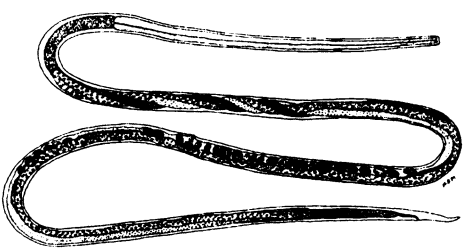

Fig. 402 *Strongyloides westeri*; adult parasitic female (0.1–9 mm long) [3]

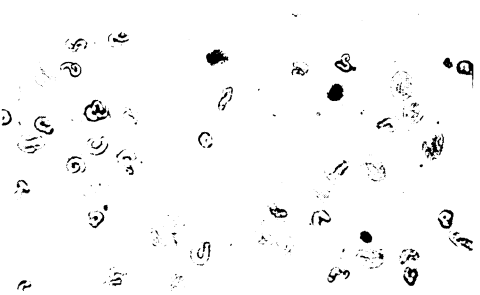

Fig. 403 Eggs of *Strongyloides westeri* (40–50 × 30–40 µm) [15]

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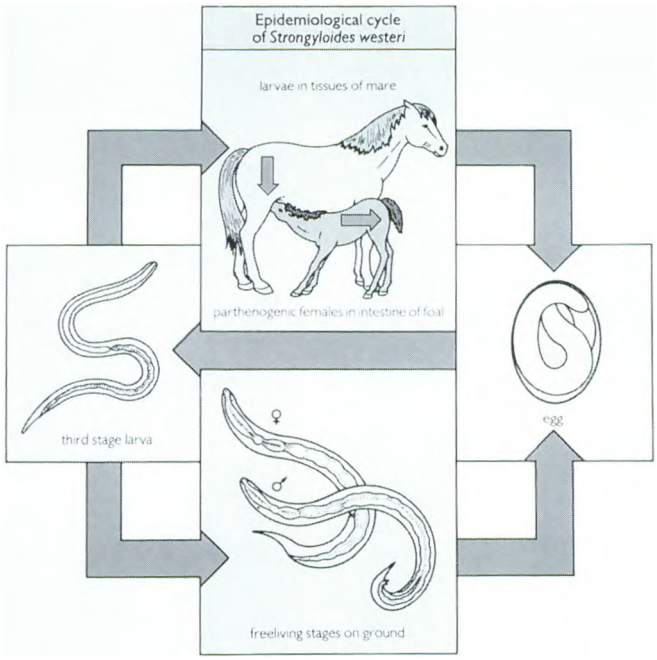

Fig. 404 Life cycle *Strongyloides westeri*; infection may also be acquired galactogenically and percutaneously (not shown on this figure) [37]

Oxyuris equi Equine pinworm

Location: Large intestine (caecum, colon)
Hosts: Horse and donkey
Species description: Adult female worms migrate out of the anus and lay eggs on the perianal skin. The eggs are covered with sticky fluid. Infective larvae develop within 3–5 days inside the eggs. The horse may be infected accidentally by licking the eggs with the itchy fluid or by ingesting embryonated eggs with feed or water. Adult pinworms are of little significance in the intestine, but cause perineal irritation and intense pruritus following egg laying.
Geographic distribution: World-wide
Symptoms: Loss of condition, poor appearance, biting and scratching at the perineal region are often seen in horses infected with *O. equi*. Irritation in the perineal region causes wounds open to secondary infections and myiasis. Dull hair coat and loss of hair is known as "rat-tail".
Significance: Most of the clinical significance results from the intense itching caused by the sticky fluid with which the eggs are attached to the rump.
Diagnosis: Presence of masses of whitish-yellow eggs around the anal region.
Therapy: Most of the broad-spectrum drugs recommended for the strongyles (☞ below) and trichlorfon (35 mg/kg, po.) are effective against pinworms.
Prophylaxis: Regular treatment will eliminate the intestinal worms and frequent change of bedding will restrict reinfection.
(Figures 405, 406, 407)

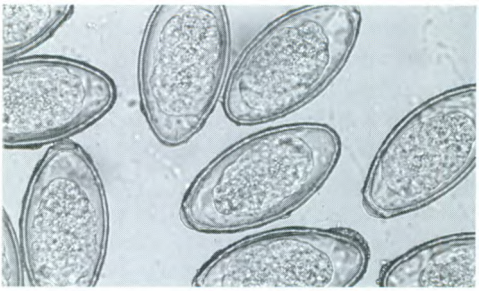

Fig. 405 Eggs of *Oxyuris equi* (85–95 × 40–45 µm)

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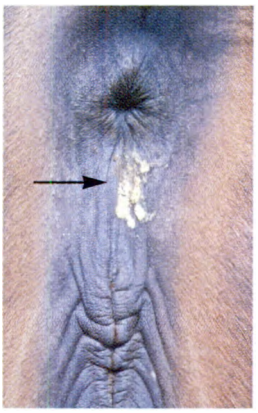

Fig. 406 Masses of yellowish eggs of *Oxyuris equi* around the perineum of a horse

Fig. 407 *Oxyuris equi*; adults (males are 0.9–1.2 cm long, females are 4–12 cm long) [37]

Probstmayria vivipara Small pinworm

Location: Colon
Hosts: Horse and donkey
Species description: It is a tiny pinworm only 2–3 mm long. Complete life cycle takes place in the host.
Geographic distribution: World-wide, except some Western European regions
Symptoms: Inapparent infections are common.
Significance: It is not clinically important. Millions of this pinworm may be present in infected horses with no clinical signs.
Diagnosis: First-stage larvae may be found in the faeces. Larvae may be collected with the Baermann apparatus. *P. vivipara* may also be found at necropsy.
Therapy: Is not indicated but most benzimidazoles and dichlorvos are effective.
(Figure 408)

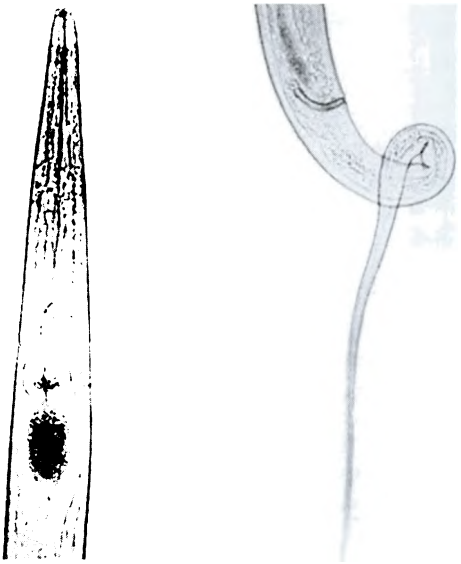

Fig. 408 *Probstmayria vivipara*; anterior end (left), posterior (right) of an adult female (2.2–3.8 mm) [38]

- **Stomach worms**

Habronema muscae, *H. majus* (syn. *H. microstoma*) and *Draschia megastoma* (syn. *H. megastoma*) Equine stomach worms, summer sores

Location: Adult worms of *H. muscae* and *H. majus* occur on the horse stomach mucosa under a layer of mucus. *D. megastoma* is in tumour-like swellings of the stomach wall. Larvae may be found in non-healing skin wounds (☞ HORSES AND DONKEYS, ■ 5.1).
Hosts: Horse and donkey
Species description: Adult worms are found in the stomach. Larvae or embryonated eggs produced by the adult worms are passed in

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the faeces and ingested by the housefly (*Musca* spp.) or stablefly (*Stomoxys* spp.) maggots which develop in manure. Larval development occurs within the maggot and the infective third-stage larvae is reached at the time the adult fly emerges from the pupa. Infective third-stage larvae are deposited by the intermediate hosts on pre-existing wounds (cutaneous habronemosis) or on moisture of the genitalia or eyes (ocular habronemosis) and migrate into the host's tissue. These irritations cause a granulomatous reaction. Larvae are deposited by the flies around the nostril, lips and wounds of horses as the flies feed. The cycle is closed when larvae or infected flies are ingested by the horse.

In temperate areas cutaneous wounds tend to heal during the winter but often recur in subsequent warm seasons (summer sores) when flies are active again. In hot, humid zones *D. megastoma* is mainly responsible for both, gastric tumour-like lesions and cutaneous habronemosis.

Geographic distribution: World-wide

Symptoms: Mild digestive disorders may result from gastric habronemosis. Granulomatous lesions on the skin and eyes resistant to conservative wound treatment are characteristic for cutaneous habronemosis (☞ HORSES AND DONKEYS, ■ 5.1)

Significance: Adult *Habronema* are of little concern but ocular or cutaneous larvae cause annoyance and wounds with a protracted healing, open for subsequent problems (e.g. myiasis). *D. megastoma* produces tumour-like swellings in the stomach (up to 10 cm in diameter) which may rupture and cause fatal peritonitis or block the passage.

Diagnosis: Antemortem diagnosis is difficult since the thin-shelled eggs or larvae are easily missed in faecal examinations. Worms and eggs may be found in gastric lavage. The almost transparent adult worms can be found during postmortem examination. Non-healing, reddish brown, greasy skin granuloms that contain rice-grain-sized, calcified material is indicative for cutaneous habronemosis. Larvae in lesion scrapings can sometimes be found.

Therapy: High dosages of the following benzimidazoles cured gastric habronemosis: thiabendazole (3 × 75 mg/kg), oxfendazole (3 × 15 mg/kg), fenbendazole (1 × 30–60 mg/kg) and oxibendazole (10 mg/kg, po.). Ivermectin (200 µg/kg, po.) is highly effective. Two intramuscular dosages of ivermectin (200 µg/kg) cured summer sores within 5 weeks.

Prophylaxis: Fly prevention by stacking manure and using insecticides where horses are kept during the day. Skin breaks should be avoided during the fly season and existing wounds should be treated with fly repellents or a combination of an antiseptic and insecticide.

(Figures 409, 410, 411)

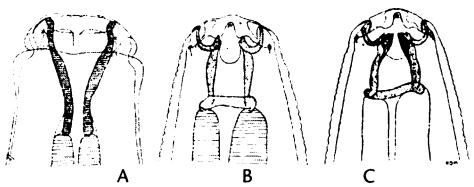

Fig. 409 Lateral views of anterior ends of equine species of *Habronema* spp. and *Draschia* spp. (schematic): (A) *D. megastoma* (males are 7–10 mm, females are: 10–13 mm), (B) *H. muscae* (males are 8–14 mm, females are 12–22 mm) and (C) *H. majus* (males are 9–16 mm, females are 15–25 mm) [3]

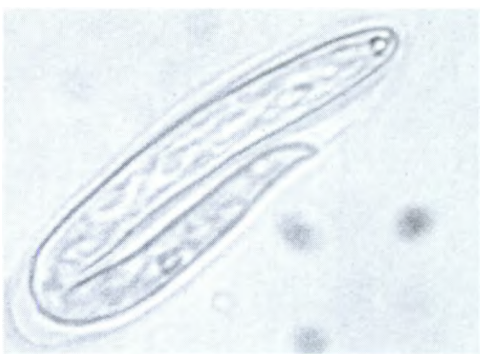

Fig. 410 Egg of *Habronema* spp. (40–50 × 10–16 µm) [11]

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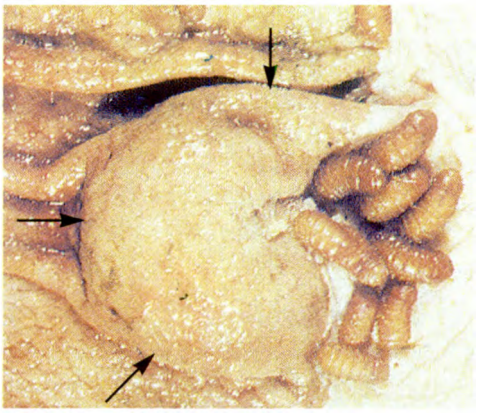

Fig. 411 Large fibrous granuloma in the stomach mucosa caused by *Draschia meagastoma*. The bot-fly larvae which are present in the illustration do not contribute to the pathological process [37]

Trichostrongylus axei Stomach hair worm

Location: Lumen of the stomach glands and in the small intestine

Hosts: Horse, donkey and ruminants (☞ CATTLE, 1)

Species description: Direct life cycle, typical for nematodes. *T. axei* has an exceptionally wide host-range and is common in many ruminant and equine species. The adults are slender and up to 8 mm long.

Geographic distribution: World-wide

Symptoms: Oedema, dark, foul-smelling diarrhoea and rapid loss of condition

Significance: *T. axei* is usually part of a mixed infection and therefore additive to the overall "worm-damage". Leakage of plasma proteins into the gut and decreased digestibility occurs in heavy infections. Chronic catarrhal gastritis is caused by larvae penetrating the gastric mucosa.

Diagnosis: It is difficult to rely only on faecal examination because the eggs are similar to strongyle eggs. The species can only be identified via third-stage larvae after copro-culture or adult worms at necropsy.

Therapy: ☞ below, LARGE AND SMALL STRONGYLES

Prophylaxis: As *T. axei* is primarily a parasite of cattle and sheep, grazing management is important. Overcrowding must be avoided and brood mares should be treated as foals are very susceptible. Removal of faeces and regular stool check is a helpful aid in strongyle control.

- **Large strongyles**

Strongylus vulgaris, Strongylus equinus and *Strongylus edentatus* Large strongyles

Location: Adult worms are found in the large intestine.

Hosts: Horse and donkey

Species description: Life cycle: adults of all 3 species live in the large intestine. Eggs develop into infective third-stage larvae within 3 days under moist and warm conditions. Infection is acquired by ingestion of larvae which drop their protective sheath in the small intestine. After this stage the *Strongylus* species have different somatic migration patterns which are mentioned under the individual species descriptions.

Geographic distribution: World-wide

Significance: Large strongyles are the most important parasites of equines. Of the three species, *S. vulgaris* is the most pathogenic.

(Figures 412, 413)

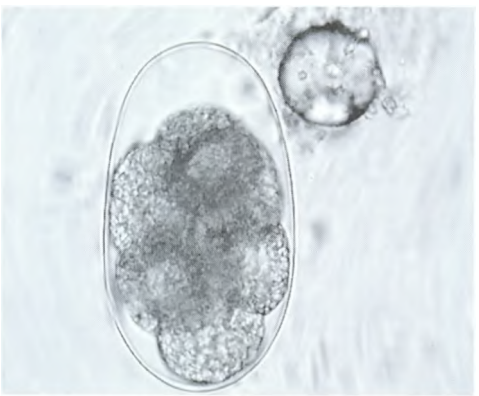

Fig. 412 Strongyle-type egg (72–93 × 40–54 µm) found in equines

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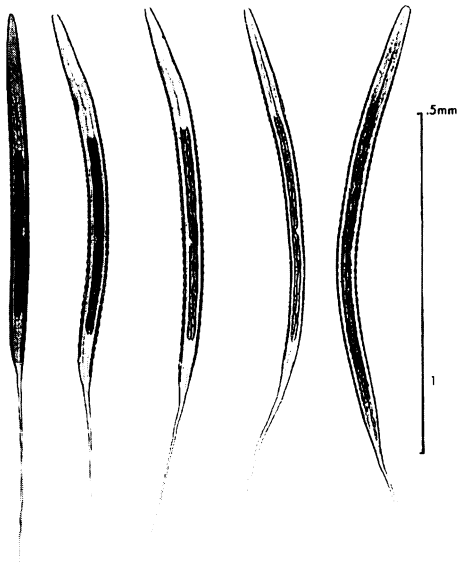

Fig. 413 Third-stage larvae of equine strongyles (850–1000) µm [38]

Strongylus vulgaris Bloodworm, Verminous endarteritis

Species description: *S. vulgaris* is the smallest of the large strongyles, reaching 2 cm in length and has typically ear-shaped teeth set in the deep of the buccal cavity. Third-stage larvae penetrate the intestinal mucosa, moult into fourth-stage larvae and penetrate further nearby blood vessels and wander through the arteries for 2 weeks before reaching the cranial mesenteric artery, where they remain for 4 months. After moulting to immature adults *S. vulgaris* returns via the arteries to the large intestine and burrows into the lumen. 6–8 months after the infection eggs are passed in the faeces. The colic associated with this parasite is not provoked by the adult worms in the gut but by the larvae during their migration through the arteries. As the larvae migrate they damage the endothelial lining of the vessels, thereby stimulating thrombus production. These clots are swept away to lodge in smaller vessels leading to thromboembolic colic.

Symptoms: *S. vulgaris* is especially important because of the damage the larvae do to the cranial mesenteric artery and its branches. Colic of various degrees, temporary lameness, gangrenous enteritis, intestinal stasis, possibly rupture and other effects depending on the location of the infarct may follow.

Diagnosis: Very difficult during the migratory, prepatent phase of the infection. Postmortem examination is indicated and larvae can be used for species identification. Adults may be found in the large intestine and eggs are present in the faeces.

(Figures 414, 415, 416, 417, 418)

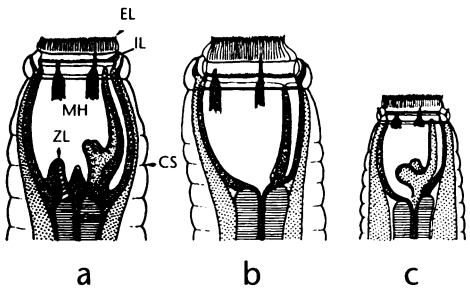

Fig. 414 Dorsolateral view of the anterior end of various *Strongylus* spp.: (a) *Strongylus equinus*, (b) *S. edentatus* and (c) *S. vulgaris*; EL = external leaf crown, IL = internal leaf crown, CS = cuticular striation and ZL = tooth [33]

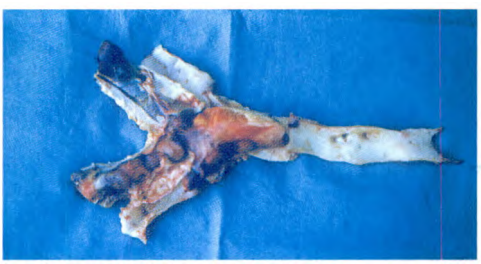

Fig. 415 Mesenteric artery with thrombosis due to migrating larvae of *Strongylus vulgaris*

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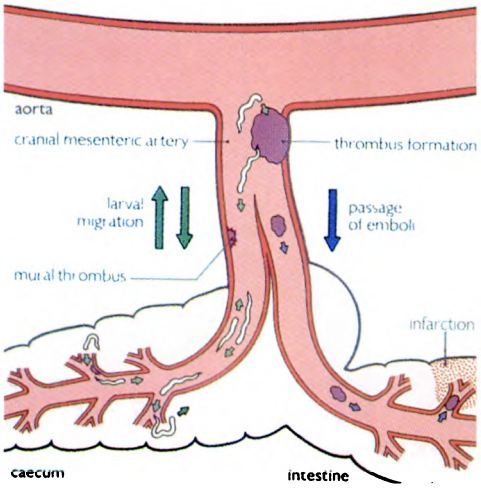

Fig. 416 Migratory route of *Strongylus vulgaris*. Note that the third-stage larvae migrate within the intima and not in the lumen of blood vessels [37]

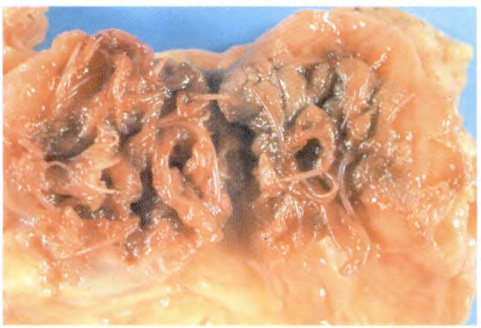

Fig. 417 *Strongylus vulgaris* larvae in a thrombus [4]

Strongylus edentatus

Species description: *S. edentatus* reaches 4 cm in length. Larvae penetrate the intestine and migrate via the portal vein to the liver, where moulting occurs and nodule formation takes place. After 9 weeks the fourth-stage larvae wander to the peritoneum, causing the formation of nodules. Migrating to the large intestine, larvae form caseous nodules in the gut wall, which they rupture to enter the colon lumen almost a full year after infection. Aberrant larvae or their migratory tracts can be found in unexpected sites such as the pleural cavity and testis.

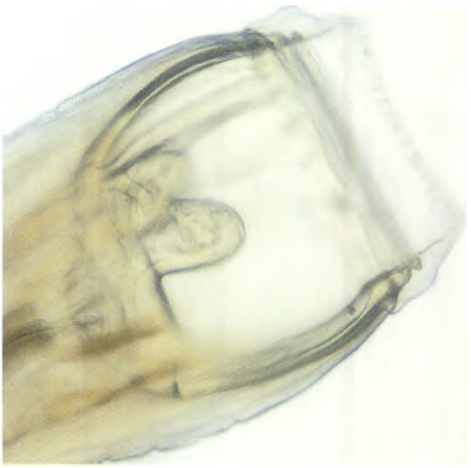

Fig. 418 *Strongylus vulgaris*; anterior end with buccal capsule

Symptoms: Adult large strongyles (*S. vulgaris*, *S. equinus* and *S. edentatus*) are "plug-feeders", ingesting plugs of mucosa as they move about in the intestine. Heavy feeding of this type produces intestinal damage, blood and protein loss into the intestine. Intestinal damage causes diarrhoea, fever, oedema, anorexia, depression, weight loss and dehydration.

The attendant blood loss may lead to anaemia, especially in *S. equinus* infections. *S. equinus* and *S. edentatus* larvae rarely cause damage of clinical consequence during their migratory phase which is in contrast to *S. vulgaris* larvae which cause great problems during migration within the arteries and hardly any damage as adult worms in the gut.

Diagnosis: Diagnosis of mixed strongyle infections is based on demonstration of eggs in the faeces (*Strongylus* spp.). Differential diagnosis can be made by identifying the infective larvae after copro-culture. Colic due to verminous arteritis can be associated with a palpable, painful enlargement at the root of the mesentery.

(Figures 419, 420, 421)

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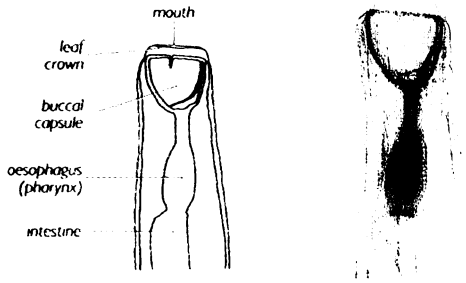

Fig. 419 *Strongylus edentatus*; anterior end [37]

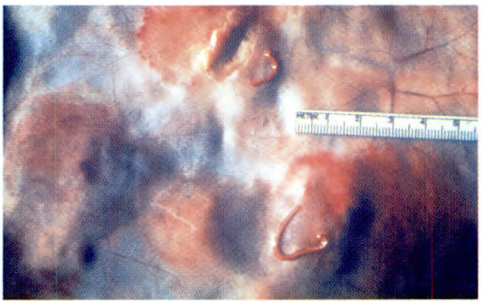

Fig. 421 *Strongylus edentatus* larvae from the parietal peritoneum of the flank [15]

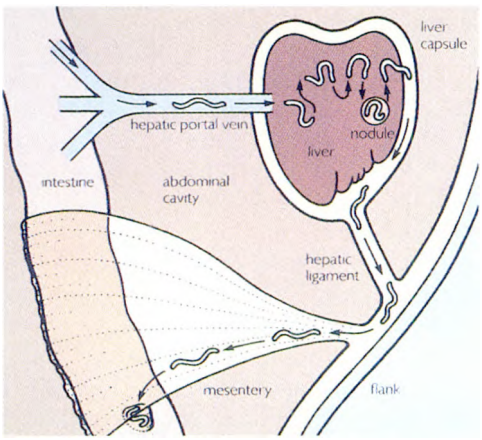

Fig. 420 Migratory pathway of *Strongylus edentatus* [37]

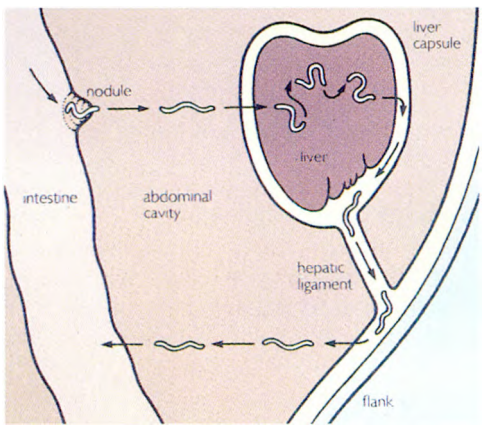

Fig. 422 Migratory pathway of *Strongylus equinus* [37]

Strongylus equinus

Species description: *S. equinus* reaches 5 cm in length. After burrowing into the submucosa of the small intestine and moulting to the fourth-stage larvae *S. equinus* forms a nodule on the intestine. After that larvae wander to the liver where they meander through the liver parenchyma for 6–7 weeks without nodule formation. Emerging from the liver they moult to immature adults and migrate in various abdominal organs (incl. pancreas) then return to the large intestine. Passage to and from the liver occurs directly across the peritoneal cavity. Prepatent period is about 9 months. *S. equinus* rarely causes damage of clinical consequence during its migratory phase. *S. equinus* suck up to 10 ml of blood per day. Heavy infections are associated with anaemia.

Symptoms and Diagnosis: ☞ above Symptoms and diagnosis of *S. edentatus* (Figures 422, 423)

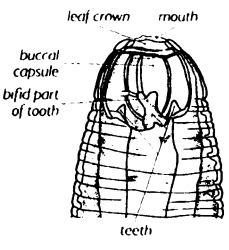

Fig. 423 *Strongylus equinus*; anterior end [37]

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- **Control of large strongyles**

Therapy: Ivermectin (0.2 mg/kg) is effective against both adult and larval stages of *S. vulgaris* and *S. edentatus* and only against adult *S. equinus* worms. Fenbendazole (10 mg/kg, po. during 5 days) and oxfendazole (15 mg/kg, po.) at dosages higher than for the adult worms are also effective against larval infections. Most of the modern broad-spectrum anthelmintics such as cambendazole, mebendazole, oxfendazole, oxibendazole are effective against adult large and small strongyles. Pyrantel (19 mg/kg, po.) is efficacious against adult small and large strongyles and larvae of most of the small strongyles. Febantel (6 mg/kg, po.) is effective against adult large and small strongyles. Dichlorvos (31–41 mg/kg, po.) is highly effective against *S. vulgaris* and *S. equinus* and against small strongyles. Haloxon (60–75 mg/kg,po.) is only effective against *S. vulgaris*. Trichlorfon (40 mg/kg, po.) combined with mebendazole, fenbendazole, oxfendazole or pyrantel (each at its regular therapeutic dosage) is highly effective against ascarids, pinworms, small and all the three large strongyles.

Prophylaxis: Grazing horses are likely to be infected, therefore a control program attempts to minimize the level of pasture contamination and reduce the risks associated with the migrating larvae. All horses kept in the same feeding-community should be treated and preferably at the same time. Newly acquired horses should be de-wormed before admittance to the herd. Whatever program is used, faecal samples should be examined periodically to maintain surveillance of the effectiveness. Horses kept in arid areas should be treated at least twice during the rains (midway and end of rains) with a compound which is effective against adults and larvae in order to increase the utilization of feed during that time. Regular dosing (generally every 4–8 weeks) is indicated in humid areas. Colic may be associated with the use of anthelmintics, especially when organophosphorous products (trichlorfon or dichlorvos) are used.

- **Small strongyles**

***Triodontophorus* spp., *Trichonema* spp., *Cyathostomum* spp., *Cylicocyclus* spp., *Cylicostephanus* spp., *Cylicodontophorus* spp., *Gyalocephalus* spp., *Oesophagodontus* spp. and *Craterostomum* spp.**

Location: Large intestine

Hosts: Horse and donkey

Species description: More than 25 species of small strongyles are found in the caecum and colon. Apart from *Triodontophorus* spp. which are almost as large as *S. vulgaris*, most of them are smaller than the large strongyles. *Triodontophorus* spp. are also known as the "non-migratory" large strongyles. One species, *Triodontophorus tenuicollis*, causes severe ulcers in the wall of the colon. There is no extra-intestinal migration of the larvae within the host. Small strongyles undergo a typical direct nematode life cycle and larvae develop within the wall of the large intestine. Larvae may undergo hypobiosis and remain dormant in the intestinal wall during periods of adverse environmental conditions (dry season, winter). Small strongyles usually appear as mixed infections. Damage is mainly caused by the adult worms which are plug feeders, ingesting plugs of intestinal mucosa and blood vessels. Larvae within the gut wall may also suck blood. Animals under three years of age are more susceptible particularly if kept on overcrowded pastures. Several species of small strongyles are resistant to many of the benzimidazoles which is a heritable trait and not reversible.

Geographic distribution: World-wide

Symptoms: Loss of condition, anaemia, weakness, acute or chronic diarrhoea

Significance: Small strongyles are extremely prevalent and cause considerable economic loss and severe disease wherever horses are raised.

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Diagnosis: Species identification is impossible solely based on the egg morphology. Larval identification following faecal culture is indicated. Strongyle eggs passed by foals of 6 months or less are those of the small strongyles as the prepatent period of *Strongylus* spp. is longer.

Therapy: ☞ above LARGE STRONGYLES. Benzimidazole-resistant strains are susceptible to piperazine (88 mg/kg, po.), pyrantel-pamoat (19 mg/kg, po.), dichlorvos (31–41 mg/kg, po.), ivermectin (0.2 mg/kg, sc.) haloxon (60–75 mg/kg, po.) and oxibendazole (10 mg/kg, po.).

Prophylaxis: ☞ above LARGE STRONGYLES

Remarks: The "horse strongyles" consist of more than 40 species many of which are distributed world-wide. They are responsible for a variety of clinical syndromes, including the well-known loss of condition, often associated with anaemia, that characterizes the "wormy-horse" and acute or chronic diarrhoea.

(Figures 424, 425, 426, 427, 428, 429, 430)

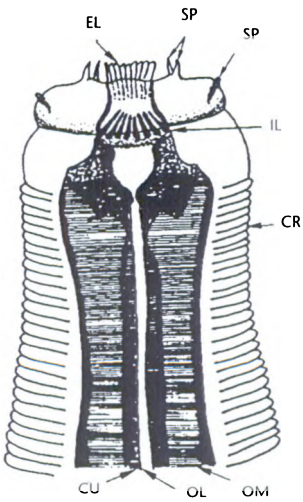

Fig. 424
Dorsolateral view of the anterior end of a small strongylid (*Cylicostephanus* sp.); EL = external leaf crown, IL = internal leaf crown, CR = cuticular striation, SP = papillae, CU = cuticula, OL = oesophagus and OM = oesophageal muscle [33]

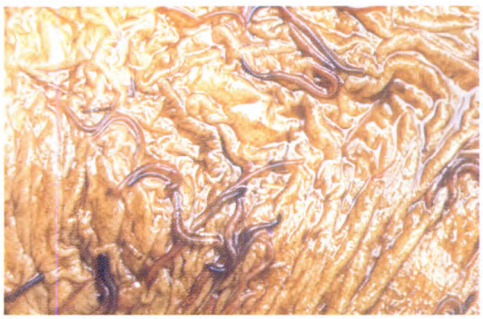

Fig. 425 Small strongyles (females up to 25 mm; males up to 15 mm) in the colon of a horse [10]

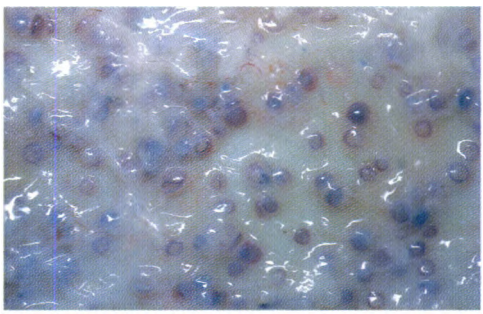

Fig. 426 Developing small strongyle larvae in the mucosa of the caecum

Fig. 427 Small strongyle with a well-developed leaf crown and a pronounced buccal capsule

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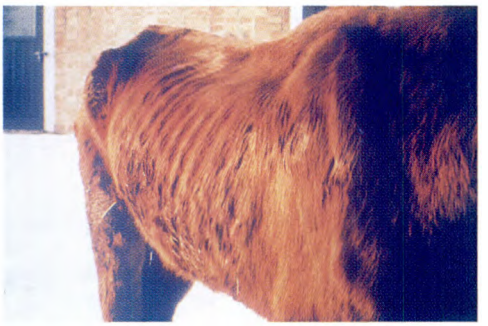

Fig. 428 A wormy horse due to winter cyathostominosis [15]

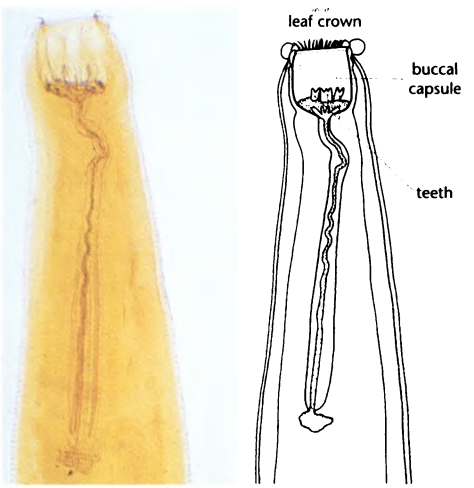

Fig. 429 *Triodontophorus* sp.; anterior end [37]

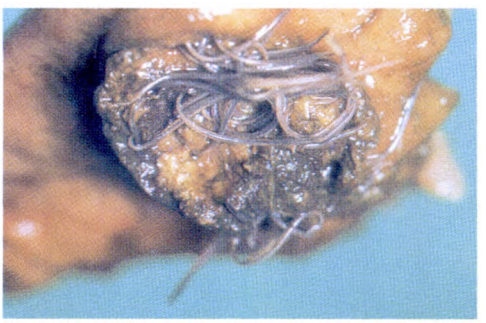

Fig. 430 *Triodontophorus* sp. group of adults forming a large ulcer in the large intestine (17–26 mm long) [37]

Dictyocaulus arnfieldi Equine lungworm

Location: Adult worms in the bronchi and bronchioles of donkeys and horses, embryonated eggs and larvae in fresh faeces (☞ HORSES AND DONKEYS, ■ 4.3).
(Figures 431, 432)

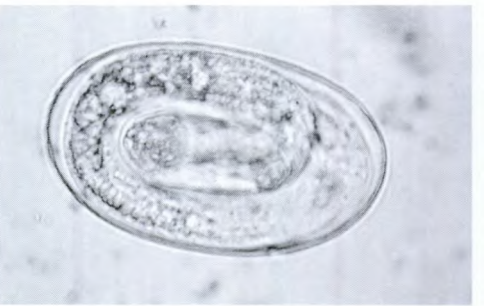

Fig. 431 *Dictyocaulus arnfieldi*; egg containing a larva (74–96 × 46–58 µm) [11]

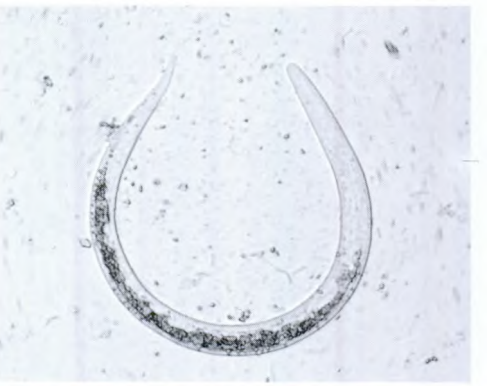

Fig. 432 *Dictyocaulus arnfieldi*; first-stage larva (420–480 µm)

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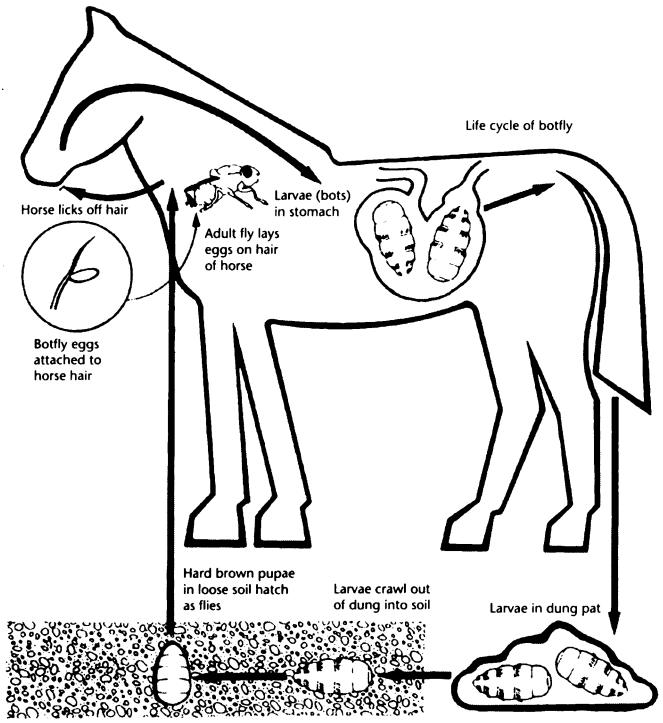

Fig. 433 Life cycle of horse botflies [36]

Fig. 434 *Gasterophilus* sp.; the horse bot fly (8–18 mm) [19]

- Insecta found in the gut

***Gasterophilus* spp.** Horse bot flies

Gasterophilus intestinalis (syn. *G. equi*) Common horse bot fly

Location: Eggs are deposited mainly on forelegs, between the knee and the hoof but also in the scapular region where they are licked by the host which stimulates hatching. Larvae are found in the cardiac portion of the stomach.

Gasterophilus nasalis The throat bot fly

Location: Eggs are laid in the intermandibulary space in numbers of up to 500 and usually 1 per hair. Larvae are found in the pylorus region of the stomach and duodenum.

Gasterophilus haemorrhoidalis The nose or lip bot fly or rectal bot fly

Location: Eggs are laid on hairs of the lips and around the mouth.
Third-stage larvae attach to the stomach but also to the hosts rectum, sometimes in great numbers.

Gasterophilus pecorum The plant bot fly

Location: Eggs are deposited on the leaves of plants and its larvae are ingested by grazing animals.
Second-stage larvae and sometimes third-stage larvae are found in the pharynx and the upper part of the oesophagus but third-stage larvae is normally attached to the fundus of the stomach.
Geographic distribution: Africa, Old World

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- **General features of the horse bot flies**

Hosts: *G. intestinalis* occurs in horses and donkeys whereas the other three species also parasitize other equids (e.g. zebras).

Species description: The larvae of these flies are gastrointestinal parasites of equines. After hatching the first larval instars burrow into dermal layers of the mouth cavity, from which they move to either the pharynx, stomach or rectum, where the second and third-stages attach. The larvae feed on tissue exudates and remain in the host up to 1 year before they become mature and are excreted with the faeces to pupate in the soil. The adult flies are brown in colour and hairy and resemble bees.

Geographic distribution: Global

Symptoms: Bots cause a mild gastritis, but large numbers can be found in the stomach without clinical signs. The first-stage instars migrating to the mouth can cause stomatitis and may produce pain on eating. Adult flies cause annoyance when laying eggs.

Significance: 1) The adult flies distress their equine host markedly up to the point of causing stampeding. 2) Irritation by the larvae during migration, e.g. dermatitis, inflammation of the pharynx, oesophagus, stomach or rectum (rectum prolapse). 3) Large numbers in the pyloric region may interfere with the sphincter function.

Diagnosis: Specific diagnosis of *Gasterophilus* infection is difficult and can be made only by demonstrating larvae in the faeces. Observation of the cream-white bot eggs (1–2 mm) on the animal's hair may help.

Therapy: In temperate areas it is assumed that most animals are infected by the end of the summer. A single treatment at a time when the adult flies have disappeared is usually adequate. Oral application (gel or pellet via feed, solutions via stomach tube) of trichlorfon (10 mg/kg, po. for bots only; 40 mg/kg, po. for bots and nematodes), dichlorvos (adults: 31–41 mg/kg, foals: 20 mg/kg for treatment of bots and ascarids; 10/mg/kg at 3–4 weeks intervals during fly season for bot control) and ivermectin (200 µg/kg, po.) is effective against all stages of bots and removes a high percentage of bots from the digestive tract. In temperate climates bot medication is usually administered 1 month after the onset of the winter and repeated in midwinter. A similar schedule can be applied to subhumid areas with treatments at the beginning and in the middle of the dry season.

Prophylaxis: Protection of exposed horses from attacks of the adult flies with repellents (dimethyl phtalate) is not satisfactory but if applied on a regional basis it can markedly reduce the fly numbers. Insecticides which can be used against adult flies (☞ HORSES AND DONKEYS, ■ 5.1, Table 18). Frequent grooming of horses is an important bot preventive measure. Visible eggs cemented to the hair are not easily removed with brushing; however, eggs can be induced to hatch by applying warm water (45°C) to affected areas. The hatched larvae can then be washed away easily.

(Figures 433, 434, 435, 436, 437, 438, 439)

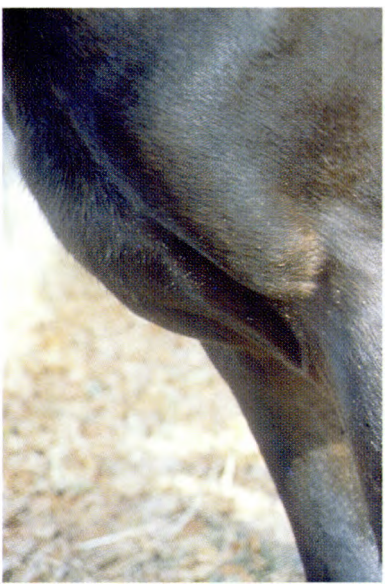

Fig. 435 *Gasterophilus* eggs deposited on the skin of the chest

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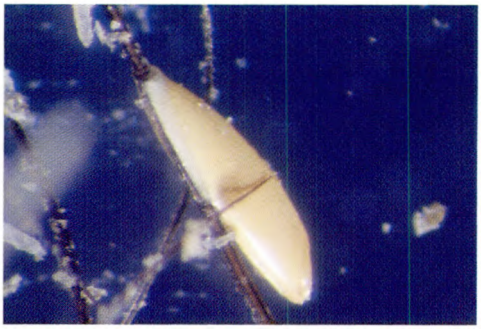

Fig. 436 *Gasterophilus* egg attached to a hair (up to 2 mm long)

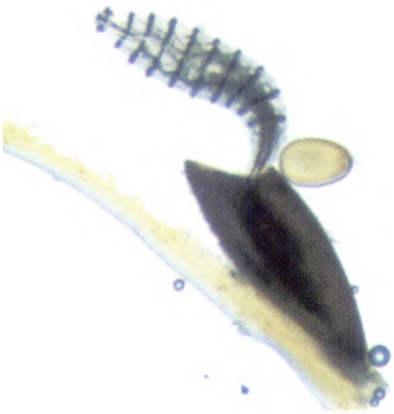

Fig. 438 *Gasterophilus* egg with a hatching larva

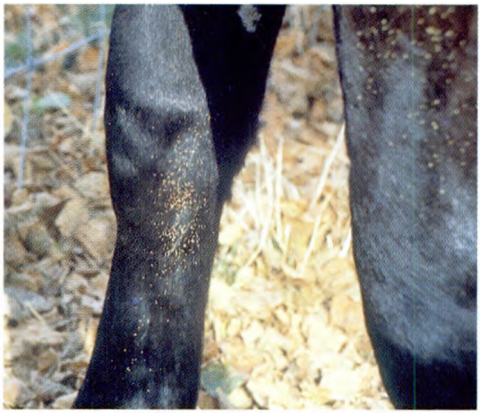

Fig. 437 *Gasterophilus* eggs deposited on the skin of the forelegs

Fig. 439 *Gasterophilus* larvae (up to 2 cm long) attached to the gastric mucosa

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2 Stages in the blood and circulatory system

PROTOZOA

Trypanosoma spp. (*Trypanosoma brucei*, *Trypanosoma congolense* and *Trypanosoma vivax*) "Nagana", "Sannare", sleeping sickness

Hosts: Horse, donkey and many other domestic and wild animals (☞ CATTLE, ■ 2)

Vector: Various tsetse flies of the genus *Glossina* (☞ CATTLE, ■ 5.1)

Species description: Tsetse-transmitted trypanosomosis is one of the major constraints for horse breeding in Africa south of the Sahara and within the tsetse belt. Donkeys are less susceptible and suffer significantly less from trypanosomosis than horses. *T. brucei* is very virulent in horses and can cause an acute infection with death within 15 days after disease onset. A subacute form may last for up to 3 months after the infection. *T. congolense* and *T. vivax* are usually chronic infections with progressive anaemia and weakness.

Geographic distribution: Africa, south of the Sahara

Symptoms: Acute *T. brucei* infections cause hyperthermia, rapid emaciation, oedema of the lower parts of the thorax and abdomen, joints and genitalia, keratitis and nasal discharge. Locomotory ataxia may develop to general paralysis or paresis. *T. congolense* and *T. vivax* infections are usually more protracted with progressive anaemia and emaciation, irregular fever attacks, corneal opacity and nasal discharge. Oedema is rare at the beginning of infection and "self-cure" may occur, especially if the animal is kept under good conditions. However, when animals are overworked or suffer from other conditions, the disease can become more acute or can render the animal more susceptible to other diseases.

Significance: Tsetse-transmitted trypanosomosis (mainly *T. brucei* but also *T. congolense* and *T. vivax*) is a major constraint for equines, especially horses in tsetse-infested regions of Africa. Both the direct noxious effect of trypanosomes on the host and their immunosuppressive effect are of great significance.

Diagnosis: A presumptive diagnosis is based on finding an anaemic animal in poor condition in an endemic area. Confirmation by demonstrating trypanosomes in stained bloodsmears or wet mounts. The most sensitive rapid method is a wet mount of the buffy coat area of a PCV tube after centrifugation. Other infections that cause anaemia and weight loss are babesiosis, anaplasmosis and should be excluded by examining a stained bloodsmear. African Horse Sickness and Equine Infectious Anaemia should be excluded as underlying viral infections.

Therapy: The most effective but often poorly tolerated compound against *T. brucei* and *T. evansi* (☞ below) is quinapyramine (5 mg/kg, sc.). The dosage should be split in three portions and administered at 6-hour intervals. Suramin sodium (7–10 mg/kg, im. or iv.) was highly effective but it is no longer commercially available. Isometamidium is effective against *T. vivax* (0.5 mg/kg, im.) and *T. congolense* and against *T. brucei* (0.5–1 mg/kg, im.), provided that the dosage is divided over several injection sites. Diminazene aceturate (3.5 mg/kg, im.) is highly effective against *T. brucei*, *T. vivax* and *T. congolense*, half of the dosage is administered immediately and the other half 6 hours later.

Prophylaxis: Isometamidium (0.5–1 mg/kg) protects horses for 2–4 moths against *T. vivax* and *T. congolense*. The injection must be given deeply intramuscularly and

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the dosage should be split over several injection sites. Quinapyramine prosalt (7.4 mg/kg, sc.) may be used to prevent *T. brucei* but also *T. evansi*. The prophylactic effect lasts for 3–4 months.

Insecticides, used as toxicants or as repellents may reduce the attacks by tsetse flies. At the time of high insect activity the use of insecticides may also reduce the general fly-annoyance in the environment of horses. Insecticides may be used as whole-body sprays or as sponge-on, smear-on, etc. (Figures 440, 441, 442, Table 17)

Fig. 440 Horse infected with *Trypanosoma congolense* showing oedema on the lower thorax

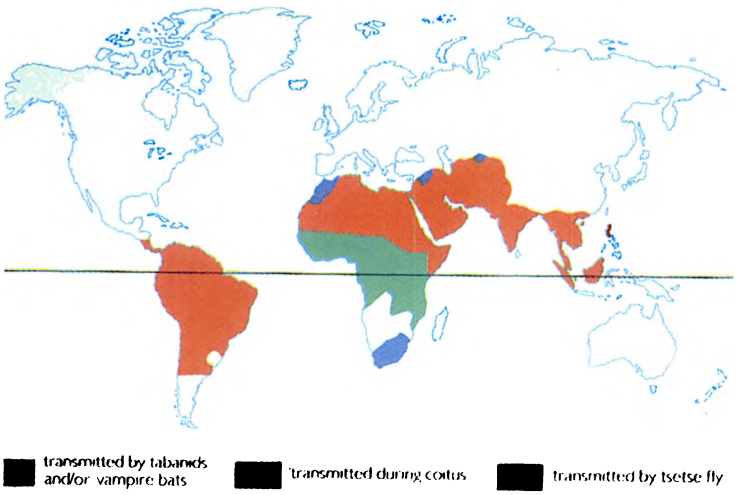

Fig. 441 Geographic distribution of equine trypanosomosis [37]

Table 17 Trypanocidal drugs to be used in equines

Generic name	Dose (mg/kg) and Route	Suscetible Trypanosomes
Quinapyramine dimethyl sulfate or chloride	5, subcut.	*T. brucei, T. congolense, T. equiperdum T. evansi, T. simiae , T. vivax*
Homidium chloride or bromide	1, IM	*T. brucei, T. congolense, T. vivax*
diminazene aceturate	3.5–7, IM	*T. brucei, T. congolense, T. evansi, T. vivax*
Isometamidium chloride	0.5–2, IM	*T. congolense, T. vivax*
Pyrithidium bromide	2, IM	*T. congolense, T. vivax*
Suramin	7–10. IV	*T. brucei, T. equiperdum, T. evansi*

Diminazene aceturate should not be administered to equines if possible. If there are no other compounds available, the daily dosage should be split into 2–3 aliquots and administered 4 h apart. Suramin is no longer commercially available [39]

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Fig. 442 Donkey with heavy *Trypanosoma brucei* infection

Trypanosoma evansi "Surra", "El Debab", "Dioufar", "Thaga", "Doukane"

Hosts: Horse and dromedary

Vector: Transmission by biting flies such as *Tabanus* spp., *Stomoxys* spp., *Chrysozona* spp. and *Lyperosia* spp.

Species description: *T. evansi* is difficult to differentiate from *T. brucei*. All domestic animals are susceptible but the disease is fatal only in horses, dromedaries and dogs. Other animal species may act as reservoirs of infection. Surra is usually fatal to horses if treatment is not applied, death occurs in a few days or months depending on the virulence of the strain. The disease progress is slower in Africa than in Asia.

Geographic distribution: Northern and Sahelian Africa, Asia, Central and South America

Symptoms: Progressive weakness, emaciation and oedema are the most common clinical signs. Oedema varying from urticarial plaques on the neck and the flanks to extended oedema of the legs and the lower parts of the body. Fever attacks, difficulties in walking, lacrimation, conjunctivitis, hypertrophy of the superficial lymph nodes and ascites may occur. Nervous signs only occur in the terminal stage.

Significance: *T. evansi* is a dangerous disease for horses and dromedaries in Africa north of latitude 15° and many other parts of the world.

Diagnosis: Emaciation and oedema. Trypanosomes are found in bloodsmears but the typical form is morphologically indistinguishable from the slender and intermediate forms of *T. brucei*.

Therapy: The most effective but often poorly tolerated compound against *T. evansi* is quinapyramine (5 mg/kg, sc.). The dosage should be split in three portions and administered at 6-hour intervals. Suramin sodium (7–10 mg/kg, im. or iv.) has also be used successfully but this compound is no longer commercially available.

Prophylaxis: Transmission by flies (but also fly annoyance in general) can be reduced by using insecticides (coumaphos, malathion, carbaryl, dioxathion, pyrethrins, permethrin, dichlorphos ☞ HORSES AND DONKEYS, ■ 5.1, Table 18). Horses may be treated with insecticides in dips, whole-body sprays, or fine mist sprays; as toxicants or repellents in aerosols or as sponge-on, smear-on, or wipe-on applications for control of biting flies. Chemoprophylaxis can be attempted with quinapyramine prosalt (7.4 mg/kg, sc.) which causes serious local reactions. The prophylactic effect lasts for 3–4 months. Suramin-quinapyramine complex (quinapyramine sulphate 10 g and anhydrous Suramin 8.4 g and water ad 200 ml of solution; administered at 10 mg/kg, sc.) protects horses against *T. evansi* for 6 months.

(Figure 443)

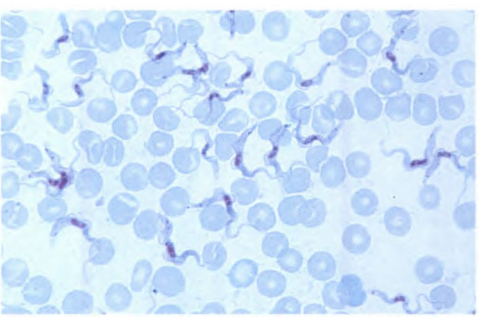

Fig. 443 *Trypanosoma evansi* (15–34 µm, mean: 24 µm long); stained bloodsmear

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Trypanosoma equiperdum "Dourine"

Species description: Dourine is venereal disease of horses and donkeys, transmitted mechanically by coitus and rarely by biting flies. Demonstration of the trypanosomes from the urethral or vaginal discharges, the skin plaques or the peripheral blood is difficult and centrifugation of these fluids may help to find the pathogens (☞ HORSES AND DONKEYS, ■ 3).

(Figure 444)

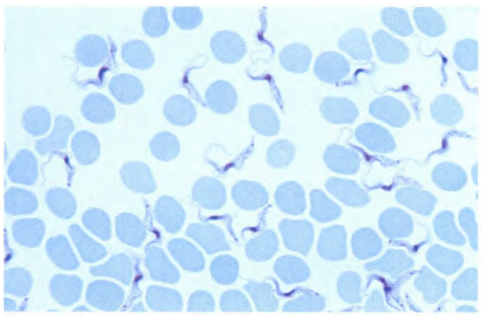

Fig. 444 *Trypanosoma equiperdum* (25 µm); stained bloodsmear (mouse)

Babesia equi (syn. *Nuttallia equi*)
Equine babesiosis

Hosts: Horse, mule, donkey and zebra

Vector: Vectors are *Rhipicephalus bursa* in the Mediterranean basin and *Rhipicephalus evertsi* in tropicoequatorial Africa. *Dermacentor* spp. and *Hyalomma* spp. may also play a role in other parts of Africa.

Species description: *B. equi* is a small *Babesia* species (2 µm in length) in horses and generally more pathogenic than *B. caballi*. It characteristically divides into four daughter organisms which form a Maltese cross. In endemic areas the course may be chronic. In the peracute course of the disease death may occur within 2 days after the onset of the disease and in the acute course the disease process lasts for 8–10 days after which the animal recovers and becomes a carrier. Special attention should be paid to previously non-exposed horses entering enzootic areas. Zebras may act as reservoirs. There is generally no transovarian transmission.

Geographic distribution: Tropical and subtropical areas

Symptoms: Around 10 days after the tick bite fever may occur (up to 41°C). *B. equi* organisms start to occur at this time in the circulating blood. Anaemia and haemoglobinuria may occur and there is listlessness, depression and inappetence. Oedema of the head, limbs and ascites occur and petechial haemorrhage and icterus of the mucous membranes of the eyes are seen. The symptoms are similar to Equine Infectious Anaemia, African Horse Sickness, leptospirosis and trypanosomosis. Intercurrent infections occur and *Babesia* spp. superimposed on a chronic horse sickness or trypanosomosis are often seen.

Significance: *B. equi* infections are of great importance as a primary cause of disease but also as secondary infections in many of the warmer parts of the world and all over the African continent.

Diagnosis: This is made by the demonstration of the parasites (Maltese cross forms) in stained bloodsmears during the period of fever. Petechiae of the mucous membranes of the eyes are characteristic of babesiosis but not specific.

(Figures 445, 446, 447, 448)

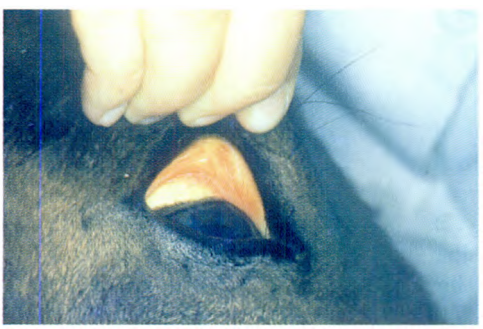

Fig. 445 Icterus due to *Babesia equi* infection [4]

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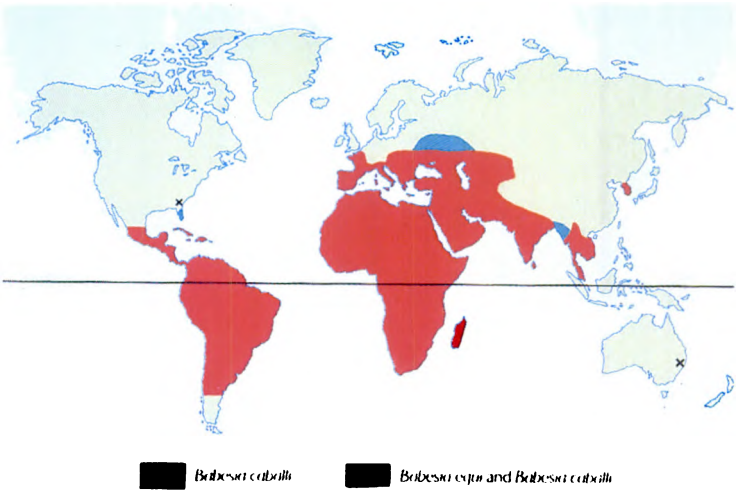

Fig. 446 Geographic distribution of equine babesiosis. The crosses mark localities where isolated outbreaks occur [37]

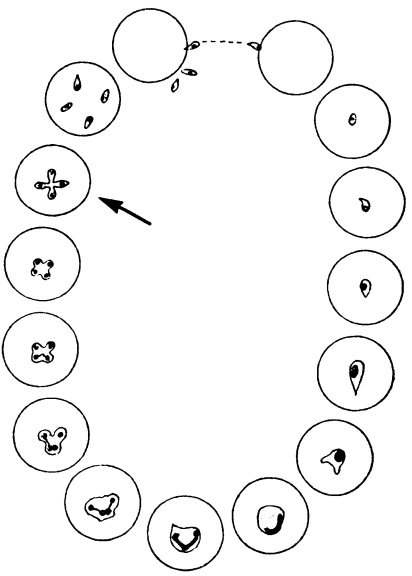

Fig. 447 Various erythrocytic forms of *Babesia equi* found in the red cells (maximum length of 2.5 µm). The *Maltese cross* (arrow) is typical of the species but not always seen at the examination of a bloodsmear [3]

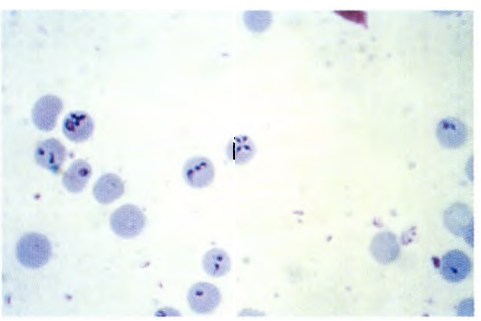

Fig. 448 *Babesia equi*; stained bloodsmear [36]

Babesia caballi (syn. *Piroplasama caballi*) Equine piroplasmosis

Hosts: Horse, donkey and mule

Vector: *Hyalomma* spp. in North Africa and the Mediterranean basin, *Dermacentor marginatus* (Mediterranean macchia) and *Rhipicephalus* spp. Transovarian transmission occurs regularly.

Species description: *B. caballi* is a large species, resembling *B. bigemina*. Parasites commonly occur as pairs, are pyriform and

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measure 2.5–4 µm. The course of *B. caballi* infections is characterized by a great variation of the clinical manifestations. It may be acute or chronic, mild or severe and end in death.

Geographic distribution: The Americas, Asia, Europe

Symptoms: Although anaemia and icterus occur, haemoglobinuria is rare. Paralysis as a result of central nervous disorders is common. Incoordination, restlessness and walking in circles may be found during infection.

Significance: *B. caballi* is a widespread infection of horses and may cause significant losses in horse populations where it is prevalent.

Diagnosis: Demonstration of the parasites, preferably in peripheral blood (skin vessels of the ear). The clinical signs and the presence of tick vectors may be of additional help. Serodiagnostic tests (IFAT, CFT) are available for both equine *Babesia* species. However, the indirect fluorescent antibody test (IFAT, ☞ METHODS, 5.2) is more sensitive than the complement fixation test (-CFT, ☞ METHODS, 5.5).

(Figures 449, 450)

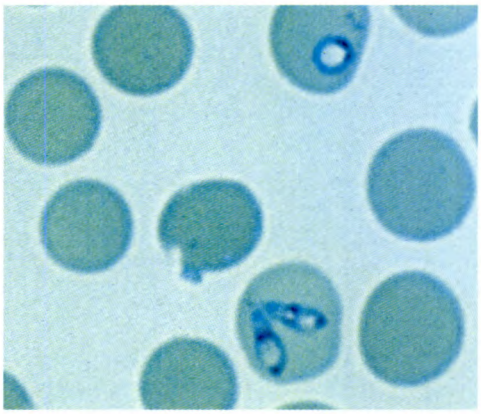

Fig. 449 *Babesia caballi*; stained bloodsmear

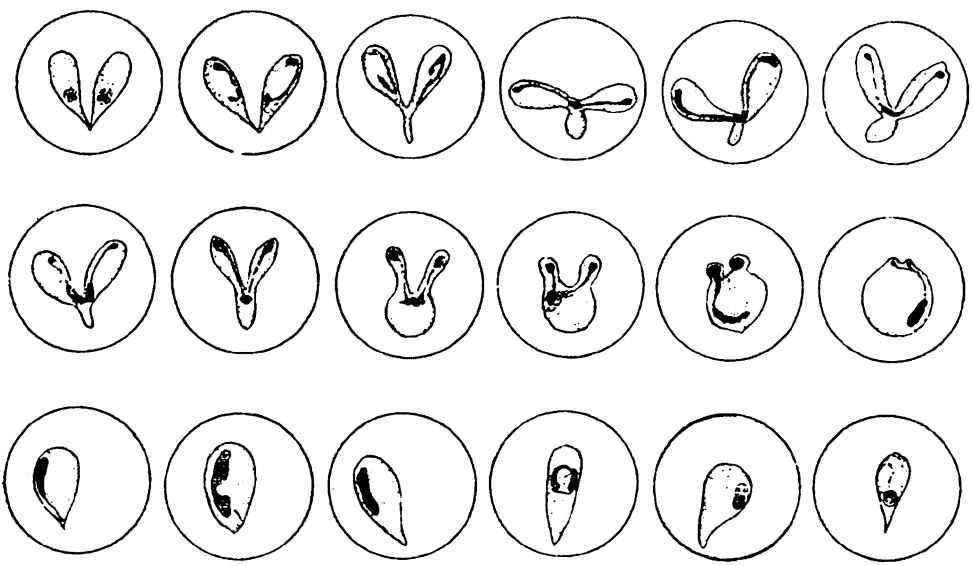

Fig. 450 Various erythrocytic forms of *Babesia caballi* found in the red cells (length: 2.5–4 µm) [3]

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- **Therapy and prophylaxis of *B. equi* and *B. caballi* infections**

Therapy: Most of the babesiacidal drugs used in other animal species are effective against *B. caballi* but only a few are effective against *B. equi* and often in markedly increased dosage rates (*Cave:* Side effects!). Imidocarb dipropionate (and dihydrochloride) administered once is used successfully against *B. caballi* (1 × 4.8 mg/kg, im. or sc.) and *B. equi* (2 × 4.8 mg/kg, sc. or im.). Side effects (extreme restlessness, sweating and abdominal pain), especially at 4.8 mg/kg, can be avoided by pretreatment with atropin sulfate (1%, 1 ml/100 kg) and dividing the dosage in two injections 3 hours apart.

B. caballi is susceptible to diminazene aceturate at 5 mg/kg, given twice at 24-hour intervals, but 6–12 mg/kg is required for *B. equi*.

The following compounds are effective against *B. caballi* but not against *B. equi*: trypan blue (2–3 mg/kg, iv.), quinoronium sulfate (2 × 0.3 mg/kg, sc., at 6-hour intervals), phenamidine (8–10 mg/kg, im.) and amicarbalide diisethoanate (5–10 mg/kg, im.).

Prophylaxis: Immunity in horses after infection lasts for more than one year and horses are therefore protected in enzootic areas even with the seasonal fluctuation of the tick population. Tick control is essential. Special attention should be paid to the ears, to the region under the tail and between the hind legs. Horses introduced into endemic areas are very susceptible and should therefore receive special attention.

Haemobartonella sp.

Hosts: Horse

Vector: Blood sucking ectoparasites (lice, ticks; species not known)

Species description: An unspecified *Haemobartonella* occurs in horses in tropical Africa (reported from Niger)

Geographic distribution: Tropical Africa

Symptoms: Anaemia, epistaxis, progressive weakening, locomotory disorders with painful joints and wasted muscles

Significance: As resurgent parasites in immunosuppressed animals *Haemobartonella* sp. may be of some importance.

Diagnosis: Coccoid (0.1–1 µm) or bacilliform (1–5 µm × 0.7–1.5 µm) parasites appear on the surface of erythrocytes.

Therapy: Oxytetracycline (5–15 mg/kg, iv.) and diminazene (3.5 mg/kg, im.)

Prophylaxis: ☞ THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 257

Ehrlichia equi

Remarks: An infectious, non-contagious seasonal disease seen in California and other parts of the USA. This rickettsial organism is found in cytoplasmatic vacuoles of neutrophils, and occasionally eosinophils during the acute stage. The organisms may be demonstrated in Giemsa-stained bloodsmears as morulae or inclusion bodies of 1.5–5 µm in diameter. The symptoms of the disease are fever, depression, ataxia, limb oedema, petechiation and icterus. The diagnosis is based on the demonstration of the organism in standard bloodsmears and an indirect fluorescent antibody test is available. Tetracycline (7 mg/kg, iv. once daily for 8 days) is highly effective. Recovered horses are immune for at least 2 years and are no carriers.

HELMINTHS

- **Trematoda found in the blood and circulatory system**

Schistosoma bovis, Schistosoma mattheei, Schistosoma indicum, Schistosoma japonicum and *Schistosoma spindale* Blood flukes

Remarks: These parasites occur in the mesenteric veins of cattle and sheep, horses and donkeys. *S. bovis* (Africa, Middle East, western Asia), *S. mattheei* (Central and Southern Africa), *S. indicum* (India sub-

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continent), *S. japonicum* (East Asia), *S. spindale* (India, Southeast Asia, East Asia). The major clinical signs associated with the intestinal and hepatic forms develop after the onset of the egg excretion and consist of enteritis and intestinal atony, anaemia and progressive emaciation in heavily infected animals.

Significance: Schistosomosis is primarily an infection of ruminants and only causes problems in horses when heavy burdens are acquired.

Diagnosis: Spindle shaped eggs occur in the faeces (☞ CATTLE, 1)

Therapy: ☞ CATTLE, ■ 2

Prophylaxis: ☞ CATTLE, ■ 1

Remarks: *Schistosoma nasale* parasitizes in the veins of the nasal mucosa and occurs in India and Southeast Asia. The eggs are found in the nasal discharge.

- **Nematoda larvae and microfilariae found in the blood**

Strongylus vulgaris

Location: Larvae congregate during their migration in the cranial mesenteric artery, causing thrombosis, embolism and intestinal infarction (☞ HORSES AND DONKEYS, 1).

Remarks: The problems (colic, intestinal infarcts) associated with *S. vulgaris* are not caused by the adult parasites in the caecum but almost exclusively by the migratory activities of the larvae.

Setaria equina

Remarks: The sheathed microfilariae can be demonstrated in peripheral blood samples. (Figures 451, 452)

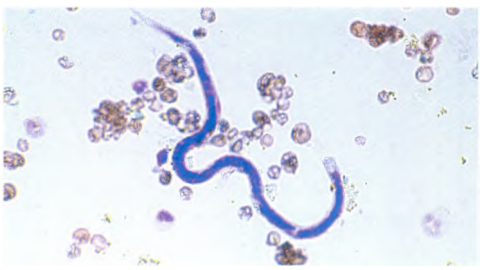

Fig. 451 *Setaria* sp. microfilaria (190–256 µm); stained bloodsmear

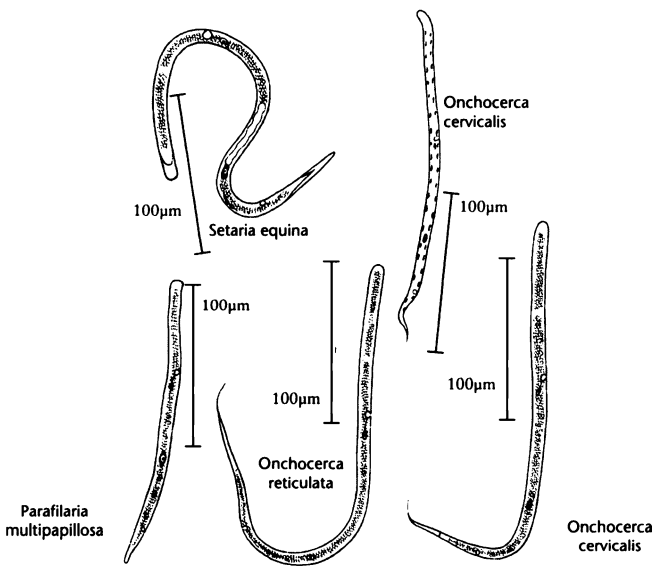

Fig. 452 Microfilariae of filariid parasites of horses [38]

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Parafilaria multipapillosa
"summer bleeding"

Remarks: Microfilariae can be demonstrated in blood discharged from "summer bleeding"nodules, caused by adult female parasites. They are less than 200 µm long and have a rounded posterior extremity.

Onchocerca cervicalis and *Onchocerca reticulata*

Remarks: Microfilariae of these two nematodes may be demonstrated by excising a small piece of skin from the linea alba and placing it in physiological saline solution. Microfilariae usually migrate out of the dermis into the solution (overnight). *O. cervicalis* microfilariae are slender and 207–240 µm long. *O. reticulata* microfilariae are 330–370 µm long and have a long, whip-like tail ending in a fine point. The distance from the genital cell to the tip of the tail is greater than 140 µm for *O. reticulata*.

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PROTOZOA

Neospora caninum

Remarks: *N. caninum* can be transmitted transplacentally in equines. Abortion and weakness with paralysis in newborn foals may occur. Tachyzoites of *N. caninum* were found in sections of lung of an equine fetus aborted 2 month before term. Individual tachyzoites were approximately 3–5 × 2–3 µm, divided by endodyogeny, and stained positively with anti-*N. caninum* serum but not with anti-*Toxoplasma gondii* serum. *T. gondii* antibody was not found in the mare's serum (☞ CATTLE, ■ 4.6).

Trypanosoma equiperdum Dourine

Hosts: Horse and donkey

Vector: Generally transmitted by coitus and very rarely by biting flies

Species description: Dourine is a venereal disease of horses, transmitted mechanically by coitus and rarely by biting flies. *T. equiperdum* occurs in donkeys but the disease is asymptomatic. Oedema on the genitals are not obvious and skin plaques only occur in less than 10% of infected donkeys. The sperm and the vaginal discharge are very virulent and donkeys therefore are a continuous reservoir of the pathogen.

Geographic distribution: North (Mediterranean coast) and South Africa, Middle East and South America.

Symptoms: Clinical signs develop over weeks or months. Early signs include mucopurulent discharge from the urethra in stallions and from the vagina in mares, followed by oedema of the genitalia. Later characteristic circular, raised, itching plaques 2–10 cm in diameter appear on the skin and infected horses become progressively emaciated. Paralysis occurs in the terminal phase, accompanied by intense emaciation and oedema of the lower parts of the body. Mortality in untreated cases is 50–70%.

Significance: *T. equiperdum* causes the most important venereal disease in horses and is responsible for great losses wherever it occurs.

Diagnosis: Demonstration of the trypanosomes from the urethral or vaginal discharges, the skin plaques or the peripheral blood is generally not possible, although centrifugation of these fluids may help to find the pathogens. The clinical disease is typical in endemic areas to allow diagnosis. Infected animals can be detected with the complement fixation test but cross reaction with *T. evansi* and *T. brucei* are common. In endemic areas infected horses should be treated.

Therapy: Quinapyramine sulfate (3–5 mg/kg, sc.) is one of the few compounds effective against *T. equiperdum*. In many countries chemotherapy is prohibited and strict border controls are required before introducing equines.

Prophylaxis: In endemic areas effective prophylaxis is not feasible. Infected horses transmit the disease during coitus. Donkeys act as reservoir hosts without clinical signs. Eradication is only possible if a strict control of all imported horses and breeding can be carried out and stray horses can be eliminated.

(Figures 103, 444, 453, 454, Table 6)

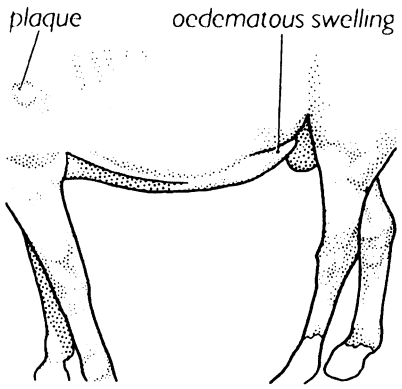

Fig. 453 Oedema of the genitalia and skin plaques associated with Dourine (*Trypanosoma equiperdum* infection) [37]

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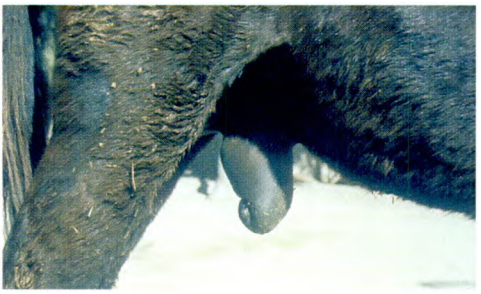

Fig. 454 Oedematous swellings of the praeputium due to *Trypanosoma equiperdum* infection (donkey) [4]

Klossiella equi

Remarks: This species occurs world-wide in the kidneys of horses, donkeys and zebras. Thin-shelled oocysts (50–100µm in diameter) may incidentally be found in the epithelial cells of the loop of Henle. These oocysts contain 40–60 sporocysts (4–6 × 6–10 µm) with 10–15 sporozoites each. Sporocysts are passed in the urine and may be discovered in the sediment. *K. equi* are generally regarded as nonpathogenic.
(Figure 455)

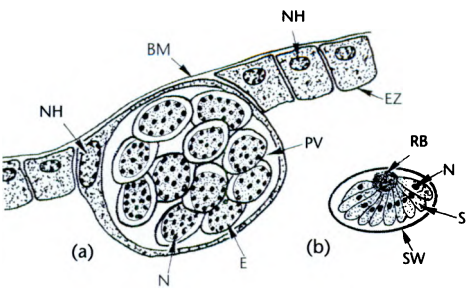

Fig. 455 *Klossiella equi*: (a) infected kidney cell (50–100 µm in diameter) containing sporocysts and (b) sporocyst (4–6 × 6–10 µm) excreted with the urine, NH = nucleus of host cell, N = nucleus of parasite, E = developing sporocyst, EZ = endothelial cell, SW = wall of the sporocyst, RB = residual bod, BM = basal membrane, PV = parasitophorous vacuole and S = sorozoite [7]

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4.1.1 Muscles

PROTOZOA

Sarcocystis bertrami, S. equicanis and *S. fayeri*

Location: Sarcocysts are found in the heart, diaphragm and other muscles

Hosts: Horse and donkey

Species description: *S. bertrami*, *S. equicanis* and *S. fayeri* are described as equine species and it seems that the dog acts as final host. The origin and general life cycle of *Sarcocystis* spp. is imperfectly understood. *S. neurona* is a recently described new species the life cycle of which is still unknown.

Geographic distribution: World-wide

Symptoms: *Sarcocystis* spp. of horses are only mildly pathogenic. Slight fever, anaemia may occur in the acute phase of infection while chronic infection may cause a multifocal granulomatous myositis.

Significance: *Sarcocystis* spp. in horses is very common and compared to those species found in cattle and small ruminants only mildly pathogenic (☞ CATTLE, ■ 4.1.1).

Diagnosis: Muscle cysts (up to 10 mm in length) may be found. These whitish streaks are embedded in the muscles and divided into compartments, packed with banana-shaped parasitic cells.

Therapy: Unknown

Prophylaxis: Uncooked horse meat fed to dogs and probably to other carnivores closes the life cycle of equine *Sarcocystis* spp.

Toxoplasma gondii

Remarks: *T. gondii* can infect virtually all warm-blooded animals. Only Felidae can act as definite hosts. Infection takes place by ingestion of sporulated oocysts. Horses can become infected by eating herbage contaminated with cat faeces or by accidentally ingesting tissues of infected rodents. The prevalence of infection is usually high (> 30%) but clinical disease attributable to

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this parasite is rare. However, in the muscle cells of horse tissue cysts of *T. gondii* are found (☞ SWINE, ■ 4.1).

4.1.2 Tendons

HELMINTHS

- **Nematoda larvae and adult nematodes found in the tendons**

Onchocerca cervicalis
Nuchal ligament worm

Location: Ligamentum nuchae
Hosts: Horse and donkey
Species description: The male is 6–7 cm long and the female up to 30 cm. The microfilariae are 200–240 µm in length and 4–5 µm in diameter, unsheathed and with a short tail. Microfilariae are found in the skin of the ventral midline. Intermediate hosts are biting midges (*Culicoides nubeculosus* and other *Culicoides* spp.).
Geographic distribution: World-wide
Significance: Adult *O. cervicalis* parasites are non-pathogenic and cause little reaction. Microfilariae may cause dermatitis.
(Figures 452, 456, 457)

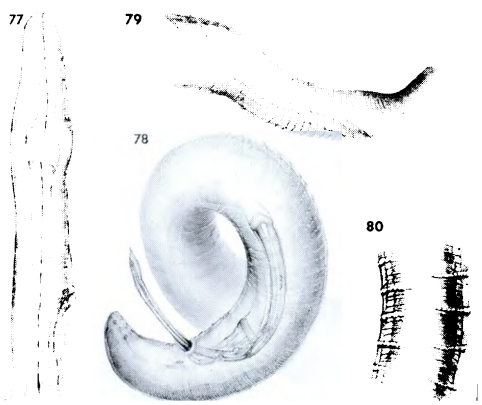

Fig. 456 *Onchocerca cervicalis*; anterior end (77), tail end of a male (78), posterior end of a female (79), cuticular striation (80); males are 6–7 cm long, and females are up to 50 cm long [40]

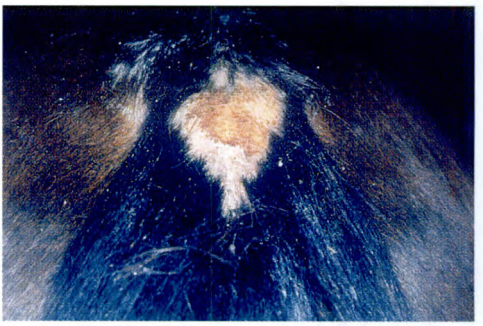

Fig. 457 Moist dermatitis and loss of tail hair due to *Onchocerca cervicalis*

Onchocerca reticulata Ligament worm

Location: Connective tissue of the flexor tendons and the suspensory ligament of the fetlock, mainly the foreleg.
Hosts: Horse and donkey
Species description: Microfilariae occur in the skin on either side of the linea alba and cause a dermatitis during the activity period of the intermediate host (*Culicoides nubeculosus* and other *Culicoides* spp.). They measure 330–370 µm and possess a long whiplash-like tail.
Geographic distribution: World-wide
Significance: Chronic inflammations and calcifications may cause lameness. Microfilariae may cause dermatitis.
(Figures 452, 458, 459)

Fig. 458 *Onchocerca reticulata*; adult worms in the tissue of the flexor tendons and associated tissue [15]

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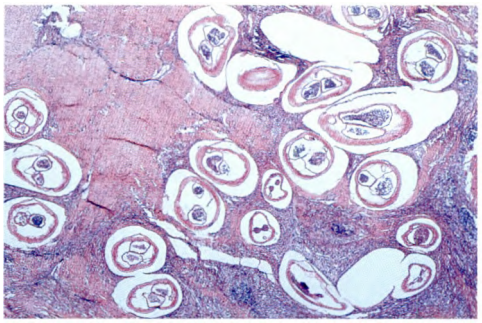

Fig. 459 *Onchocerca reticulata*; tendon of the musculus interosseus with multiple sections of the adult worms (histological section)

Onchocerca raillieti

Location: Adults occur in the cervical ligament, subcutaneous cysts on the penis and in the perimuscular connective tissue.

Hosts: Donkey

Species description: Very little is known about this parasite.

Geographic distribution: Africa

Significance: Adult parasites are non-pathogenic.

- **General features of *O. cervicalis*, *O. reticulata* and *O. raillieti***

Significance: Adult parasites of *O. cervicalis* and *O. raillieti* are non-pathogenic and cause few problems. However, *O. reticulata* infections in the flexor tendons and suspensory ligament may be associated with lameness. A seasonal, sporadic dermatitis is known to occur in many parts of the world due to the presence of microfilariae. The reaction may be induced by both the microfilariae (dead and dying ones) in the dermis and the bites of the insects (hypersensitivity to midge bites). Microfilariae also accumulate in the eyes of horses but there is still some debate about their role in the pathogenesis of equine uveitis. *Onchocerca* infections are often accompanied by depigmentation of the bulbar conjunctiva. Other lesions of onchocercosis involve the cornea and include oedema and punctate or streaking types of opacities of the stroma, superficial erosions and sclerosing keratitis.

Symptoms: Adult parasites cause little symptoms. Calcifications in the connective tissue (caused by dead parasites) may be found without clinical symptoms. Lameness due to *O. reticulata* infections may occur.

Papular or exudative dermatitis with alopecia and pruritus due to microfilariae ("summer mange", allergic "dermatitis") is a well-known entity associated with *Onchocerca* infections.

Diagnosis: The diagnosis of *Onchocerca*-associated dermatitis may be based on the good responsiveness to microfilaricidal treatment. Microfilariae can be found by a full-thickness skin biopsy > 6 mm. The tissue is macerated in isotonic saline for several hours. Microfilariae are concentrated and stained with methylene blue after removal of skin pieces. Microfilariae can be differentiated microscopically from *Setaria* spp. which occur in the blood and possess a sheath around the larvae.

Therapy: No treatment is effective against the adults. Ivermectin (200 µg/kg, sc.) is efficacious (> 99%) against microfilariae and produces marked clinical improvement in horses with onchocercal dermatitis. A single oral dosage of ivermectin showed marked clinical improvement within 1–2 weeks after treatment and the *Onchocerca* spp. microfilariae disappeared within this time. The presence of *O. cervicalis* microfilariae in the skin of horses is not always associated with dermatitis, but where dermatitis occurs, it can be treated successfully with ivermectin. Diethylcarbamazine (5–8 mg/kg, sc.) for 21 days is effective against microfilariae.

Prophylaxis: Generally not indicated. Insecticidal sprays and repellents may reduce the attacks by biting midges.

(Figures 452, 460, 461, 462, 463)

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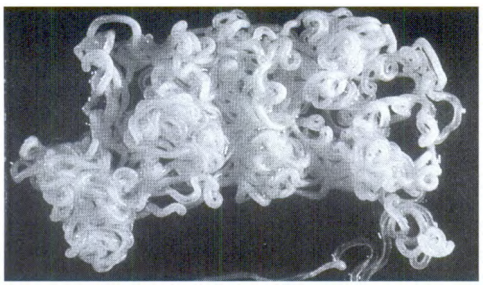

Fig. 460 *Onchocerca* sp.; adult convoluted parasite. The worm is up to 30 cm long and found interwined through the connective tissues and can be isolated by artificial digestion [37]

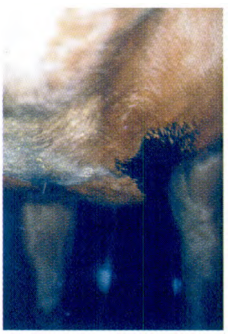

Fig. 461 *Onchocerca* sp. associated moist dermatitis (horse)

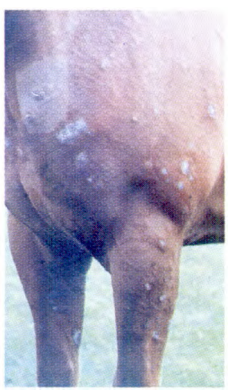

Fig. 462 *Onchocerca* sp. associated dermatitis [41]

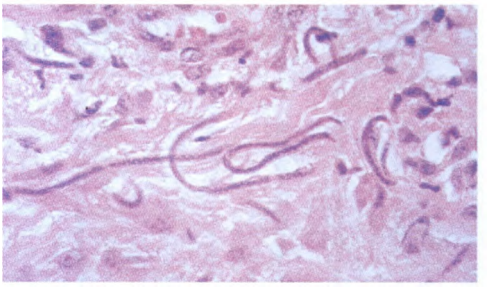

Fig. 463 Microfilariae of *Onchocerca* sp. found in the subcutaneous tissue of a horse (histological section) [41]

4.2 Liver

HELMINTHS

- **Trematoda found in the liver**

Fasciola gigantica ☞ CATTLE, ■ 4.2
Fasciola hepatica ☞ CATTLE, ■ 4.2
Dicrocoelium dendriticum ☞ CATTLE, ■ 4.2

- **Cestoda found in the liver**

Remarks: Larvae of the canine tapeworm *Taenia hydatigena* occasionally also occurs in equines (☞ CATTLE, ■ 4.2 and 4.4 and SHEEP AND GOATS, ■ 4.2 and 4.4).
Hydatid cysts (larvae) of the canine tapeworm *Echinococcus granulosus* are often found in the liver of horses (☞ CATTLE, ■ 4.2).
(Figure 464)

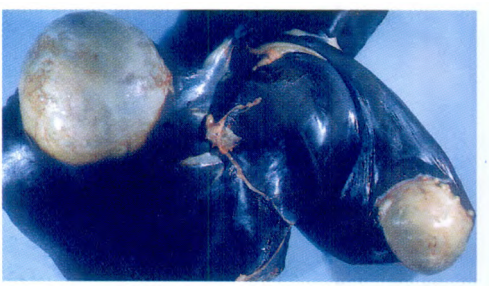

Fig. 464 Hydatid cysts of *Echinococcus granulosus* found in the liver of a horse

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- Nematoda found in the liver

Capillaria hepatica

Remarks: Occasionally the parasite is found in the liver of horses. It deposits its eggs in the liver parenchyma where they produce granuloma and fibrosis. *C. hepatica* is common in rodents but may occur from time to time in a variety of other hosts including horse and man. *C. hepatica* very rarely causes disease in horses.

Parascaris equorum

Remarks: Larvae are transient visitors in the liver during their migration from the intestine to the heart. They may cause "white spots" which are seen at necropsy (☞ HORSES AND DONKEYS, 1)

Strongylus edentatus and *Strongylus equinus*

Remarks: Fourth-stage larvae of these nematodes may cause nodules and tracts during their migratory period in the liver (☞ HORSES AND DONKEYS, 1)

4.3 Respiratory system

HELMINTHS

- Cestoda found in the respiratory system

Echinococcus granulosus

Remarks: Larvae (hydatid cysts) of the canine tapeworm *E. granulosus* may also be found in the lungs of equines (☞ CATTLE, ■ 4.2 and ☞ HORSES AND DONKEYS, ■ 4.2)

- Nematoda found in the respiratory system

Dictyocaulus arnfieldi Equine lungworm

Location: Bronchi and bronchioles
Hosts: Donkey and horse
Species description: *D. arnfieldi* has a direct life cycle. The ingested larvae penetrate the intestine and reach the lungs via the circulatory system. In the lungs they break through the blood vessels into the bronchi and develop to adults. The prepatent period is 5–6 weeks. *D. arnfieldi* is very prevalent in donkeys and other equines and relatively rare in horses. Donkeys are often carriers and thus a risk for horses kept in the same environment.
Geographic distribution: World-wide, particularly in areas with heavy rainfall
Symptoms: Severe coughing, loss of appetite, chronic pneumonia, pulmonary oedema, secondary bacterial infections. Heavy infections may cause death, particularly in foals.
Significance: Donkeys are common hosts and symptomless carriers who constitute a reservoir of infection for horses. *D. arnfieldi* does no damage if only a few are present, but large numbers can cause death.
Problems arise when horses and donkeys are kept together, especially in warm, humid areas and where hygiene is poor.
Diagnosis: Identification of embryonated eggs in fresh or larvae in older faeces and tracheal mucus; clinical signs in the infected horses.
Therapy: Levamisole (1 × 5 mg/kg, im.) twice, 3 weeks apart, mebendazole (20 mg/kg, po.) on five consecutive days, fenbendazole (50 mg/kg, po.) are effective against *D. arnfieldi*. Ivermectin oral paste (0.2 mg/kg) is highly effective against *D. arnfieldi* adults and fourth-stage larvae.
Prophylaxis: Regular treatment of both horses and donkeys if they are kept together. The treatment scheme against the other "horse strongyles" is also effective against *D. arnfieldi*. If possible, horses and donkeys should be kept on different pastures. Removal of faeces at the feeding place is important.
(Figure 432, 465)

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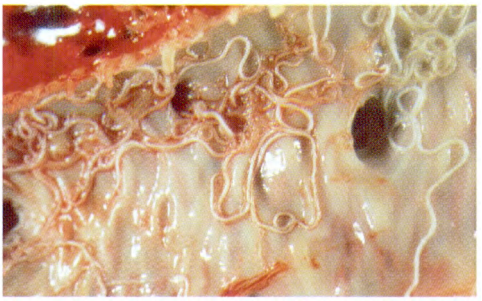

Fig. 465 *Dictyocaulus arnfieldi*; adult parasites in the trachea female: 3.8–8.5 cm, male: 2.6–4.3 cm [4]

Habronema spp.

Remarks: Larvae may occasionally occur in the lungs (☞ HORSES AND DONKEYS, ▦ 1).

ARTHROPODS

- **Insecta larvae found in the respiratory system**

Rhinoestrus purpureus

Remarks: Life cycle similar to those of the genus *Oestrus*. Larval stages are obligatory parasites of horses and mules in tropical Africa. Larvae are deposited by the adult fly in batches of 8–40 into the nostrils or eyes. The larvae crawl into the nasal cavity where they remain for several months before moving to the sinuses where they mature. They cause obstruction, irritation and sneezing (☞ SHEEP AND GOATS, ▦ 4.3 p. 183, Figure 489).

4.4 Abdominal cavity

HELMINTHS

- **Cestoda found in the abdominal cavity**

Cysticercus tenuicollis

Remarks: The larval stage of the canine tapeworm *Taenia hydatigena* may sometimes be found as long-necked cysts attached to the omentum, intestinal mesentery and serosal surface of abdominal organs (☞ SHEEP AND GOATS, ■ 4.4).

- **Nematoda found in the abdominal cavity**

Setaria equina Equine abdominal worm

Location: Adult worms in the abdominal cavity and very rarely as aberrant worms in other parts of the body (lungs, eyes). Microfilariae occur in the blood.

Hosts: Horse and donkey

Species description: Microfilariae produced by adult worms are ingested by mosquitoes (*Aedes* spp. and *Culex* spp.) where they develop within two weeks. When mosquitoes feed again on horses the infective larvae are re-injected. Adult males reach 8 cm in length and females 13 cm. The head end shows typical peribuccal prominences. Microfilariae are sheathed and 190–256 µm in length (Figure 452).

Geographic distribution: World-wide

Symptoms: Generally no symptoms are seen except when worms (mainly microfilariae) are present in the eyes causing blindness.

Significance: Adult *Setaria equina* in the abdominal cavity are non-pathogenic. Very rarely larvae may migrate to the anterior chamber of the eyes or to the brain and spinal cord where they cause damage.

Diagnosis: Detection of sheathed microfilariae in the bloodsmear.

Therapy: Is generally not indicated. There is no therapy yet against adult *Setaria*. Diethylcarbamazine (40 mg/kg, po.) given for 1–3 days or ivermectin (0.2 mg/kg, sc.) are partially effective against microfilariae.

Prophylaxis: *Setaria* control is closely associated to mosquito control. Topical insecticides may be useful to reduce mosquito bites.

(Figures 466, 467, 468)

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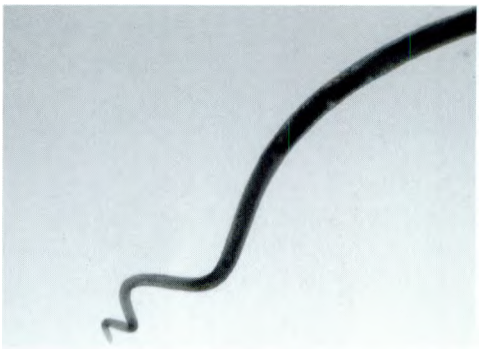

Fig. 466 *Setaria* sp.; coiled tail end (males reach 8 cm in length and females 13 cm)

Fig. 467 *Setaria equina*; adult; anterior end with typical peribuccal prominences

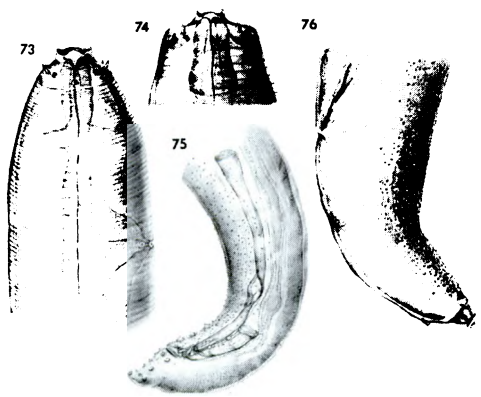

Fig. 468 *Setaria equina*; anterior end (73 and 74) and posterior end of a male (75), posterior end of a female (76) [40]

Strongylus edentatus and *Strongylus equinus*

Remarks: Fifth-stage larvae may occasionally be found during their migration subserous or within the abdominal cavity (☞ HORSES AND DONKEYS, 1).

4.5 Central nervous system

PROTOZOA

Sarcocystis neurona

Remarks: This is an apicomplexan parasite which is associated with myeloencephalitis in horses. Only asexual stages of this parasite presently are known, and they are found within neuronal cells and leukocytes of the brain and spinal cord. The parasite is located in the host cell cytoplasm, does not have a parasitophorous vacuole, and divides by endopolygeny. Schizonts are 5–35 µm × 5–20 µm and contain 4–40 merozoites arranged in a rosette around a prominent residual body. Merozoites are approximately 4 × 1 µm, have a central nucleus, and lack rhoptries. Schizonts and merozoites react with *Sarcocystis cruzi* antiserum but not with *Caryospora bigenetica*, *Toxoplasma gondii*, *Hammondia hammondi* or *Neospora caninum* antisera in an immunohistochemical test.

Toxoplasma gondii

Remarks: *T. gondii* tissue cysts may also be found in the brain and spinal cord of horses. Infection is acquired by ingesting sporulated oocysts (☞ HORSES AND DONKEYS, ■ 4.1.1 and ☞ SWINE, ■ 4.1)

Neospora caninum

Remarks: *N. caninum* tissue cysts were also found in the central nervous system of equines (☞ HORSES AND DONKEYS, ■ 3 and ☞ CATTLE, ■ 4.6)

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HELMINTHS

- **Cestoda cysts found in the central nervous system**

Echinococcus granulosus

Remarks: Larvae (hydatid cysts) of the canine tapeworm *E. granulosus* may also be found in the brain of equines (☞ CATTLE, ■ 4.2 and ☞ HORSES AND DONKEYS, ■ 4.2).

Coenurus cerebralis

Larvae of the canine tapeworm *Taenia multiceps* may also be found in the brain of equines (☞ SHEEP AND GOATS, ■ 4.6).

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5 Stages on the body surface

5.1. Skin and coat

5.2 Eyes

5.1 Skin and coat

PROTOZOA

Besnoitia bennetti

Location: The cysts of this parasite occur in subdermal connective tissue of horses.

Hosts: Horse and donkey

Species description: The morphology and life-cycle is essentially the same as for *B. besnoiti* of cattle, although the definitive host is unknown.

Geographic distribution: Africa, southern Europe, South America

Symptoms: General thickening of the skin, loss of hair and scurfy appearance. The disease has a chronic course (7–8 weeks) and progressive weakness in infected animals may occur.

Significance: *B. bennetti* may account for a chronic, depleting skin disease of horses in some parts of Africa.

Diagnosis, Therapy and Prophylaxis: (☞ CATTLE, ■ 5.1 *B. besnoiti)*

(Figure 469)

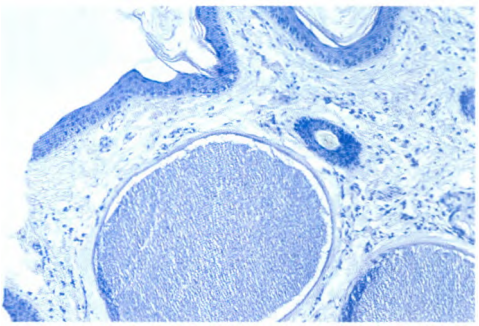

Fig. 469 *Besnoitia bennetti*; subcutaneous cyst (up to 600 µm in diameter) with several thousands of cystozoites (histological section)

HELMINTHS

- Nematoda found in the skin

Third-stage larvae of *Draschia megastoma* (syn. *Habronema megastoma)* and *Habronema* spp. Larvae of equine stomach worms; summer sores, cutaneous or ocular habronemosis

Location: Third-stage larvae of *Habronema* spp. are found in non-healing skin wounds. Adult worms are found in the stomach (☞ HORSES AND DONKEYS, 1).

Species description: Larvae of the spiruroid worms *Habronema* spp. which are deposited in existing wounds by infected flies (*Musca domestica* and *Stomoxys calcitrans*) cause a granulomatous dermatits which delays healing. Larvae deposited in the wounds migrate and feed, extending the wound and preventing healing. If licked by the host the larvae mature in the stomach.

Geographic distribution: World-wide

Symptoms: Typical skin wounds (ocular and cutaneous habronemosis) which are not healing and which contain larvae. These

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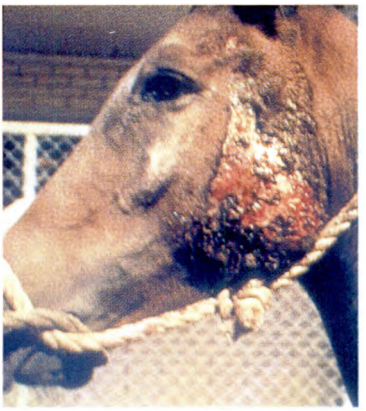

Fig. 470 Cutaneous habronemosis (summer sores) [36]

wounds tend to heal spontaneously during the period when flies are inactive (winter, dry season) but often recur in subsequent seasons when fly activity starts again (summer, wet season).

Diagnosis: Adult worms may be found during postmortem examinations (☞ HORSES AND DONKEYS, 1). Ova or larvae in the faeces are diagnostic but are easily overlooked. Non-healing, reddish brown, greasy skin granuloma that contain rice-grain-sized, calcified material is indicative for cutaneous habronemosis. Larvae are recognized by their spiny knobs on their tails, in lesion scrapings are indicative of ocular or cutaneous habronemosis.

Therapy: Two injections of ivermectin (200 µg/kg, im.) cured the cutaneous lesions caused by third-stage larvae of *Habronema* spp.. A insecticide (lindane) ointment should afterwards be applied to the wound to prevent reinfection.

Trichlorfon (25 mg/kg, iv.) administered in 1 l isotonic saline solution cured summer sores within 30 days. Premedication with atropin was necessary. Organophosphates (coumaphos, dichlorvos, crotoxyphos + dichlorvos, lindane) applied topically to the abraded surface may kill the larvae. Insect repellents may reduce transmission by flies.

Prophylaxis: Fly prevention by stacking manure and using insecticides. Skin wounds should be treated with fly repellents (citronella oil, diethyltoluamide and diethyl phtalate). Cypermethrin (0.1% solution, using 150–250 ml per animal) and pyrethrins (0.01%) are highly effective to control flies on horses.

(Figures 470, 471)

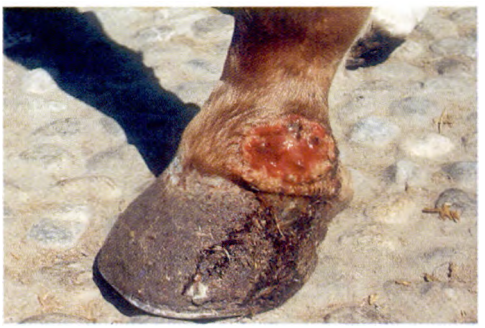

Fig. 471 Cutaneous habronemosis; typical lesions on the coronary border

Parafilaria multipapillosa
"summer bleeding"

Location: Adult worms are found in subcutaneous nodules. The haemorrhagic exudates of these nodules contain eggs and larvae.

Species description: Subcutaneous nodules, containing 3–7 cm long filarial worms, which break open and ooze blood. Blood-sucking flies (*Haematobia atripalpis* and other *Haematobia* spp.) act as intermediate hosts.

Geographic distribution: North Africa, eastern and southern Europe, Asia and South America

Symptoms: Subcutaneous nodules, particularly on the head and upper forequarters, which appear suddenly, break open, bleed and then heal up during the winter or dry season to re-appear the following season.

Significance: The nodules may interfere with the harness of working horses but are generally of little consequence.

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Diagnosis: Nodules in the skin, transient haemorrhage with eggs and unsheathed microfilariae in the exudate. The microfilariae are less than 200 µm long and have a rounded posterior extremity.

Therapy: No satisfactory treatment is available. Metrifonate (4–6 × 35 mg/kg, daily) and diethylcarbamazine (10–20 × 6–8 mg/kg, daily) but also fenbendazole (5 × 50 mg/kg, po.) are described to be used for treatment but with a moderate success.

Prophylaxis: Fly control may reduce the incidence.

(Figures 452, 472, 473, 474, 475)

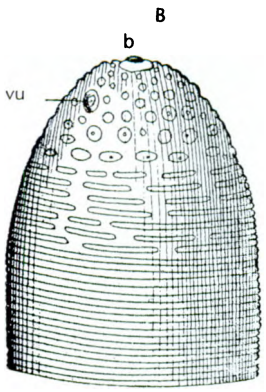

Fig. 474 *Parafilaria multipapillosa*; anterior end with vulva (vu) and buccal opening (b) [9]

Fig. 472 "Summer bleeding" due to *Parafilaria multipapillosa*; bleeding wound on the withers

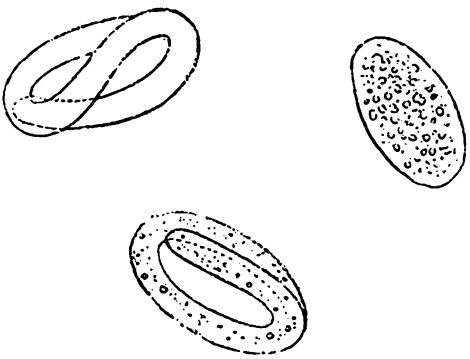

Fig. 475 *Parafilaria multipapillosa*; eggs (52–58 × 24–33 µm) containing a microfilaria [9]

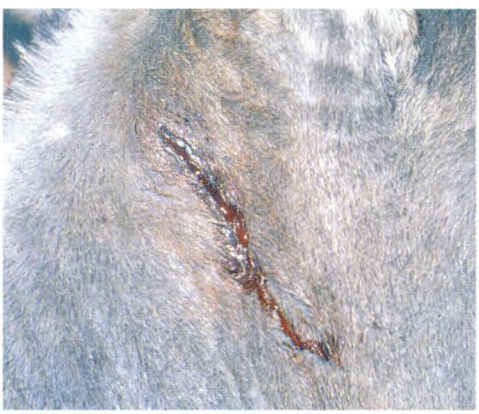

Fig. 473 "Summer bleeding" due to *Parafilaria multipapillosa*; bleeding wound

Onchocerca cervicalis Nuchal ligamen worm
Onchocerca reticulata Ligament worm

Remarks: The adult worms in ligaments cause swelling, calcification and pain, and the microfilariae may affect the cornea of the eye. Microfilariae concentrate in skin of the ventral midline, along the linea alba. Microfilariae can be found in horses with dermatitis of the face, neck, chest, withers, forelegs and abdomen. These lesions often

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include areas of scale, crusts, ulceration, alopecia and depigmentations and may be pruritic. An immunological reaction to dead or dying microfilariae may be involved in the pathogenesis of this type of dermatitis. Treatment with microfilaricidal drugs usually dramatically improves the condition, although the pathogenesis is not fully understood. Allergic reactions to the bites of small flies may produce similar forms of dermatitis and exacerbate microfilaria-associated dermatitis. The diagnosis of *Onchocerca*-associated dermatitis may therefore be based on responsiveness to microfilaricidal treatment. Large numbers of microfilariae can also be found in horses without dermatitis.

O. cervicalis has been associated with poll evil, fistulous withers and equine uveitis, but because of the presence of large numbers of the parasite in horses without these conditions, there is some debate about its role in the pathogenesis of these diseases (☞ HORSES AND DONKEYS, ■ 4.1.2).

Fig. 476 Moist dermatitis associated with the lacrimal duct. Masses of microfilariae were found in the wound [58]

Fig. 477 Fistulous withers may be caused by *Onchocerca cervicalis* [37]

Diagnosis, Therapy and Prophylaxis: ☞ HORSES AND DONKEYS, ■ 4.1.2
(Figures 452, 476, 477)

ARTHROPODS

Arthropods are divided into two main groups:

Arachnida (arachnids) including:
- *Ticks*
- *Mites*

Insecta including:
- *Lice*
- *Fleas*
- *Dipterida*

- **Arachnida found in/on the skin**

– Ticks

(☞ CATTLE, ■ 5.1)

Ticks may provoke disease in three different ways:

- **Direct noxious effects**

Heavy tick infestations cause irritation of the skin with subsequent ulceration and secondary infection. The wounds are attracted by screwworms and other flies and myiasis may develop. The presence of large numbers of ticks is associated with anaemia and annoyance and animals become anxious and restless.

In horses ticks of the following genera have a direct noxious effect:

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IXODIDAE ("hard ticks")
Amblyomma spp. (*A. variegatum* and *A. cohaerens*)
Boophilus spp.
Dermacentor spp.
Hyalomma spp.
Ixodes spp.
Rhipicephalus spp. (*R. appendiculatus*, *R. evertsi*, *R. bursa*, *R. parvus* and *R. pulchellus*)

ARGASIDAE ("soft ticks")
Otobius megnini Spinose ear tick

- **Transmission of diseases**

One of the most damaging effects of ticks is their ability to transmit diseases to their hosts. Some of these diseases are serious with fatal consequences. Transovarian transmission from one tick generation to another via the eggs is possible and aids to spread out the disease.

Babesia equi Equine biliary fever

Remarks: *B. equi* is transmitted by *Rhipicephalus evertsi* (South Africa), *R. sanguineus* (North Africa) and *R. pulchellus* (East Africa), *Hyalomma dromedarii* and *H. detritum* (North Africa). *Otobius megnini* is suspected to transmit *B. equi* in Madagascar (☞ HORSE AND DONKEYS, ■ 2).
(Figure 478)

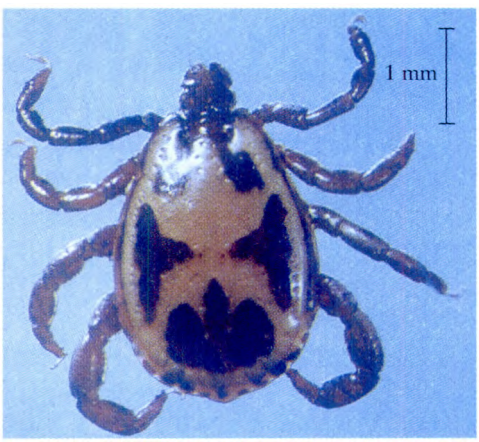

Fig. 478 *Rhipicephalus pulchellus*; male [8]

Babesia caballi

Remarks: *B. caballi* is transmitted by *Rhipicephalus evertsi* and *R. bursa* (South Africa), *R. sanguineus*, *H. dromedarii* and *H. excavatum* (North Africa) and *H. marginatum* rufipes (Africa south of the Sahara).

Borrelia theileri Spirochaetosis

Remarks: *B. theileri* is transmitted in South Africa by *Boophilus decoloratus* (the common blue tick) and *Rhipicephalus evertsi* (the red-legged tick) and *Hyalomma dromedarii* (North Africa).

- **Tick Toxicoses**

Tick paralysis
43 tick species belonging to 10 genera are known to induce tick paralysis in man and a variety of mammals. These ticks are capable of releasing a toxin into the host which causes a condition associated with progressive, ascending, afebrile, symmetrical paralysis, with hind legs being affected first followed by the forelegs. Animals may die. Paralysis is relieved if ticks are removed in time. Most of domestic animals appear to be susceptible to tick paralysis.

Amblyomma spp., *Ixodes* spp. and *Dermacentor* spp. in particular are capable of causing tick paralysis in horses.

Sweating sickness
An acute, febrile tick-borne toxicosis characterized by a profuse and moist eczema hyperaemia of the skin and visible mucous membranes. Watering of the eyes and nose, salivation and an extremely sensitive skin with a sour odour are other typical signs of sweating sickness. Eventually the skin becomes cracked and predisposed to secondary infections (incl. screwworm and myiasis). Often the course is acute and death may occur within a few days. In less acute cases recovery may occur.
Sweating sickness is mainly a disease of young calves but also sheep in eastern, central and

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southern Africa. The causative agent are certain strains of *Hyalomma truncatum* which produce an epitheliotropic toxin. Calf mortality may reach 70%.

Symptoms: Generalized hyperaemia with subsequent desquamation of the superficial layers of the mucous membranes of the upper respiratory, gastrointestinal and external genital tracts and profuse moist dermatitis. For diagnosis it is essential to determine the presence of the vector. Clinical signs appear 4–11 days after the tick bite.

Prophylaxis: Control of tick infestations is the only effective measure. Removal of ticks, symptomatic treatment and good nursing are indicated.

General toxicosis

Tick toxicosis is a general aggravation of the toxic effect of the parasite's saliva. Certain toxins have a suppressive effect on the immune system of the host and occasionally this may reactivate chronic infections (e.g. *Babesia* spp. and *Anaplasma* spp. during *Rhipicephalus appendiculatus* (brown ear tick) infections).
Toxicosis, associated with general disorders occurs with *Ornithodorus savignyi* (sand tampan) in young calves and lambs, especially when there are many bites. Animals show cutaneous oedema, haemorrhage, rapidly progressing weakness and prostration. Death can occur within 6 hours. Toxicosis may occur in recumbent animals, during rest.

- **Description of ticks affecting equines**

IXODIDAE ("hard ticks")

Amblyomma spp.
Boophilus spp.
Dermacentor spp.
Hyalomma spp.
Ixodes spp.
Rhipicephalus spp.

A general description of each genus is made in CATTLE, ■ 5.1

- **General features of horse tick infestations**

Significance: Members of these genera are important for horses. Ticks are of high economic significance for horses in tropical and subtropical regions. Heavy infestations cause great debilitation. Some species transmit diseases one of which is equine babesiosis which is accompanied by high mortality. Wounds left from tick bites are susceptible to additional attacks by various biting and calliphorine flies (myiasis).

Symptoms: Infested horses itch, bite and scratch, causing other skin wounds (self-trauma) which become infected. Anaemia, loss of appetite and weight loss are observed in heavy infestations. Tick paralysis caused by certain tick species (*see* above) can be fatal within several days if the parasites are not removed.

Diagnosis: Identification of adult ticks found on the host animal.

Therapy and Prophylaxis: ☞ below THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 257.

ARGASIDAE ("soft ticks")

Otobius megnini Spinose ear tick

Location: Larvae and nymphs feed deep in the external ear canal. Adults are non-parasitic and live on the ground.

Hosts: Horse, donkey, mule, cattle, sheep and goat

Species description: One-host tick. Free-living adult females lay eggs on the ground which hatch within 3 weeks. Larvae and nymphs live and feed on one host for up to 7 months. Infestations of this species usually build up in kraals and stables, where the host densities are high. *O. megnini* can persist in empty kraals and stables for more than 2 years.

Geographic distribution: Arid and semi-arid areas of South Africa, and South West Africa

Symptoms: Infested horses continuously shake their heads. Loss of appetite, debilitation

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and anaemia may be present. Irritation in the ear associated with secondary infections (incl. myiasis) may dominate the clinical picture. Rarely the middle and inner ear are also affected after perforation of the tympanic membrane.

Significance: Heavy infestations cause otitis externa, great annoyance and blood loss. *O. megnini* is an important tick of horses in some parts of Africa.

Diagnosis: Ticks may be found by swabbing the ear or by direct inspection of heavily infected ears.

Therapy and Prophylaxis: ☞ below THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 257 and ☞ CATTLE, ■ 5.1.

– Mites

Chorioptes equi Itchy leg mite

Location: Lower legs and feet, especially on the hind legs; horses with long hairs on the fetlocks are particularly susceptible.

Hosts: Horse, donkey and mule

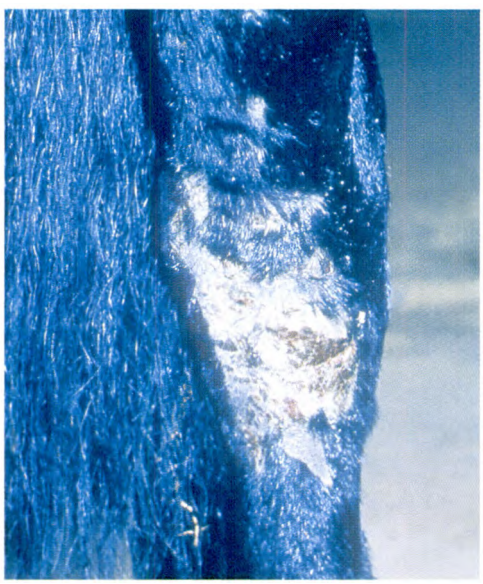

Fig. 479 *Chorioptes equi*; affected leg (posterior view) [15]

Species description: *Chorioptes* mites puncture the skin and suck lymph fluid. The mites can survive off the host for about 3 weeks.

Geographic distribution: World-wide

Symptoms: Chorioptic mange mites irritate the skin. The serum oozes and dries to crusts and scabs. These lesions are itching and infested horses bite, scratch and stamp.

Significance: Infestations are not usually severe but may persist for a long time.

Diagnosis: Demonstration of mites in skin scrapings taken from the edge of the lesions.

Therapy and Prophylaxis: ☞ CATTLE, ■ 5.1 and below THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 257

(Figure 479)

Psoroptes equi Scab mite, equine body mite

Location: Areas of thick hair such as neck and base of the tail of horses are preferred by *Psoroptes equi*. Although infections may spread over the entire body surface the disease is not as serious as in sheep.

Hosts: Horse, donkey and mule

Species description: *Psoroptes* mites puncture the skin and suck lymph fluid and can survive off the host for about 2–3 weeks.

Geographic distribution: World-wide

Symptoms: Intense itching, and scratching, followed by hair loss and skin trauma which may be secondarily infected. Severe infestations cause anorexia, emaciation and anaemia.

Significance: Psoroptic mange is rarely observed. Heavy infestations cause severe debilitation.

Diagnosis: Demonstration of mites in skin scrapings taken from the edge of the lesions.

Therapy and Prophylaxis: ☞ below THERAPY AND PROPHYLAXIES OF ECTOPARASITES, p. 257

Psoroptes hippopotis

Remarks: *Psoroptes hippopotis* occurs in ears of horses and mules. Its own significance is still under discussion.

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Sarcoptes equi Common mange mite, sarcoptic mange

Location: *Sarcoptes* mites prefer the head, the neck and the shoulders. Untreated infestations may spread over the entire body surface.
Hosts: Horse, donkey and mule
Species description: These mites can only survive a few days off the host, as they are highly susceptible to dryness. All stages occur in skin tunnels which may be extended over the entire skin of the host. The feeding and burrowing of *Sarcoptes* mites cause great irritation and itching, producing lesions with exudate which may become crusts.
Geographic distribution: World-wide
Symptoms: Infected horses bite and scratch. Chronic infestations cause thickened, wrinkled skin as a result of progressive proliferation of subcutaneous connective tissue.
Significance: Sarcoptic mange in horses is the most severe type but not very common, but it can cause much damage if allowed to spread out within a population.
Diagnosis: Demonstration of mites in skin scrapings collected from the damaged areas until the moist layers of skin are reached.
Therapy and Prophylaxis: ☞ below THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 257
(Figure 480)

Fig. 480 *Sarcoptes equi* infestation [4]

Demodex equi Demodectic mange

Location: The mites live in the hair follicles and the sebaceous glands
Hosts: Horse
Species description: *Demodex* mites are cigar-shaped organisms which are known to be able to live on their host in harmony. Only if the host's immune system is affected they may cause severe skin lesions.
Geographic distribution: World-wide
Symptoms: *D. equi* infestations produce papules and ulcers, particularly around the eyes and on the forehead. Subsequently the lesions spread to the shoulders and finally over the entire body. The affected body is covered with scales. There is no pruritus.
Significance: *D. equi* is seldom diagnosed in horses.
Diagnosis: Demonstration of the mites in the hair follicles and the sebaceous glands.
Therapy and Prophylaxis: There is no satisfactory treatment. Prophylaxis is unknown.

- **Control of mite infestations in horses**

Most organophosphates are effective against mange mite infections in horses. Acaricides are applied by spraying, rubbing or dipping (for application mode, *see* below). For groups of animals dipping is the most convenient and effective method. Permethrin spray (0.05%) but also other pyrethroids are effective. Flumethrin (0.005%) is effective against chorioptic mange in horses. Ivermectin (0.2 mg/kg, sc.) is highly effective against *Sarcoptes* mange and to a lesser extent against *Psoroptes* mange. All the tools (e.g. curry-combs, etc.) in infested stables should also be washed with an acaricide to avoid transmission.

- Insecta found on the skin

– Lice

MALLOPHAGA Chewing Lice

Werneckiella equi equi (syn. *Damalinia equi)* Biting louse of horses

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Werneckiella equi asini Biting louse of donkeys

Location: *Werneckiella* spp. are usually found on the neck and the base of the tail. In heavy infestations the parasite covers the whole body.

Hosts: Horse, donkey and mule

Species description: Adults are 1–2 mm long. The head is broad and flat. The body is wingless and flattened dorsoventrally. *Werneckiella* spp. feed on skin and hair. Development occurs exclusively on the host, within 3–4 weeks. Eggs are attached to the hair, hatch to nymphs which moults 3 times before reaching adulthood. *Werneckiella* spp. are specific for equines.

Geographic distribution: World-wide

Symptoms: Infested horses rub, scratch and bite themselves. This is accompanied by rough coat, skin infections, myiasis and loss of hair. Unthriftiness and weight loss are commonly seen in heavy infestations.

Significance: *Werneckiella* spp. bites cause mange-like skin irritations and itching. *Werneckiella* spp. may act as vectors of the Equine Infectious Anaemia.

Diagnosis: Identification of lice found on the coat.

Therapy and Prophylaxis: ☞ below *Haematopinus asini*

(Figures 481, 482)

Fig. 481 *Werneckiella equi*; equine chewing lice (up to 1.8 mm long)

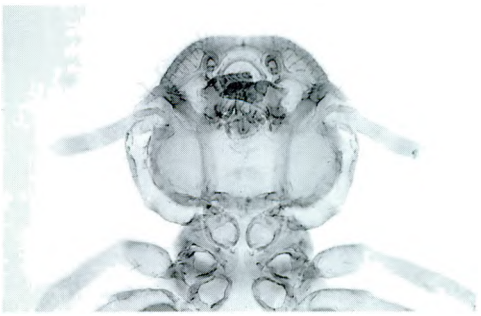

Fig. 482 *Werneckiella* sp.; head [15]

ANOPLURA Blood sucking lice

Haematopinus asini Sucking louse of horses

Location: *H. asini* is predominantly found around the head, neck, on the back and brisket and between the legs.

Hosts: Horse, donkey and mule

Species description: *H. asini* is 3–3.5 mm long, with 3 pairs of legs and a long narrow head with piercing mouth parts to suck blood and tissue fluids. Development occurs exclusively on the host, within 3–4 weeks. Eggs are attached to the hair and hatch to nymphs which moult 3 times before reaching adulthood. *H. equi* is specific for equines.

Geographic distribution: World-wide

Symptoms: Anaemia due to heavy blood loss, lowered condition and weight loss and increased susceptibility to other diseases may occur. Heavy infestations cause loss of appetite and vitality, particularly in foals.

Significance: *H. equi* is more dangerous than *D. equi* because of its blood sucking activity.

Diagnosis: Identification of lice found on the coat.

Therapy: Because lice do not leave the host, treatment of the horse with an insecticide is necessary. Organophosphates, carbamates (carbaryl) but also pyrethrins and pyrethroids are effective against lice.

Prophylaxis: Frequent grooming helps to reduce the parasite numbers.

(Figures 483, 484, 485)

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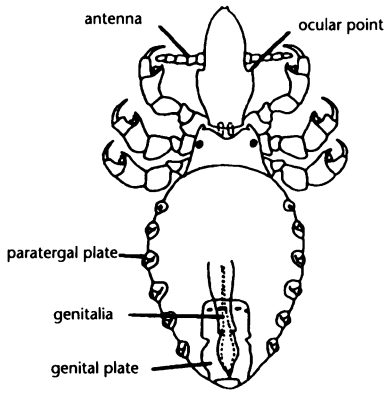

Fig. 483 *Haematopinus asini*; dorsal view (schematic) [29]

Fig. 484 *Haematopinus asini* (male is up to 2.4 mm long; female is up to 3.5 mm long)

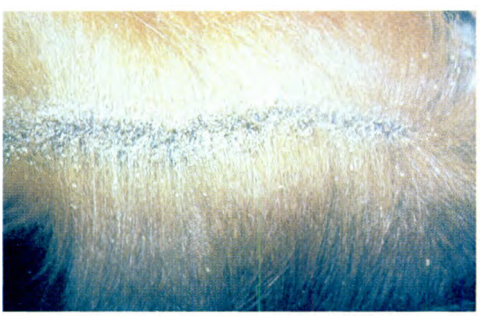

Fig. 485 *Haematopinus asini* infestation

– Fleas

Fleas are not found on horses.

– Dipterida

CULICIDAE Mosquitoes

They belong, like flies, to the order Dipterida, with a single pair of wings. The main genera are *Anopheles, Culex* and *Aedes*.
Females are capable of sucking blood which appears to be necessary for the laying of eggs. Eggs are laid either on water or on vegetation floating on the surface of water, therefore mosquitoes are found near stagnant pools (☞ CATTLE, ■ 5.1).

Significance: Mosquitoes can cause considerable distress to livestock but their main importance is their ability to act as intermediate hosts or vectors of equine encephalomyelitis, African Horse Sickness (not the main vector) and *Setaria equina*.

SIMULIIDAE

Simulium spp. Black flies, midges

Remarks: Small size (1–5 mm), found in swarms near free-running well-aerated streams. Simulids cause severe irritation to horses and other livestock when they occur in large numbers. Herds and flock will stampede, often with disastrous results. Bites are inflicted on all parts of the body, giving rise to vesicles which burst exposing the underlying flesh. Skin wounds caused by simulids heal very slowly. Certain areas of the tropics are rendered uninhabitable by simulids (☞ CATTLE, ■ 5.1).

Significance: Irritation of horses and other livestock. Skin wounds with secondary myiasis. Transmission of *Onchocerca volvulus* in man.

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CERATOPOGONIDAE

Culicoides spp. Biting midges, seasonal dermatitis, sweet itch

Remarks: Very small size (1–3 mm long). Adult female midges attack horse, man and other species (☞ CATTLE, ■ 5.1), causing marked irritation by penetration of the skin with their proboscis. The bites cause intense itching. Only the females suck blood mainly during the twilight periods and at night. *Culicoides* occur often in large swarms a few hundred metres around the breeding sites. These are moist areas such as fresh or brackish water or seepages from decaying vegetable or dung heaps.

Significance: 1) Massive irritation of livestock, itching, allergic skin reactions in horses (sweet-itch, seasonal dermatitis). 2) Transmission of *Onchocerca gibsoni* and other *Onchocerca* spp. in cattle and horses; African Horse Sickness, blue tongue of sheep.

Therapy and Prophylaxis: It has been reported that horses, stabled from one hour before sunset until the dew has disappeared the following morning, are protected from attacks by biting midges and thus from transmission of African Horse Sickness. Insecticides to be used ☞ THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 257. Permethrin (0.05%), is especially used to control *Culicoides* spp. on horses.

(Figure 486)

TABANIDAE

Tabanus spp., *Haematopota* spp. and *Chrysops* spp. Horse flies

Significance: Female tabanids attack mammals to suck blood. They cause deep, painful, irritating bites. They generally bite several times for one blood meal because they are disturbed by the host's defense. A number of diseases (besnoitiosis, anaplasmosis, trypanosomosis and anthrax, equine infectious anaemia) are mechanically transmitted by tabanids. This is especially important when horseflies are numerous among a crowded livestock population.

Therapy: ☞ THERAPY AND PROPHYLAXIS OF ECTOPARASITES, p. 257 and CATTLE, ■ 5.1).

Prophylaxis: Insecticidal spraying also against the larval stages on the ground (e.g. wet soil, vegetation, etc.).

MUSCIDAE

Musca spp., *Lyperosia* spp. and *Haematobia* spp.

Significance: Muscid flies annoy horses, especially during the summer or rainy season. They swarm around horses and other farm livestock. The resulting irritation is incessant and much of the energy of the animals is lost to defend against fly attacks. In addition to the nuisance the flies often carry pathogens on their feet and bodies. Some

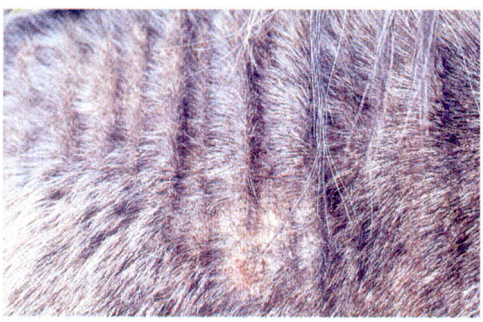

Fig. 486 Sweet-itch lesions ("seasonal dermatitis") due to *Culicoides* spp. [37]

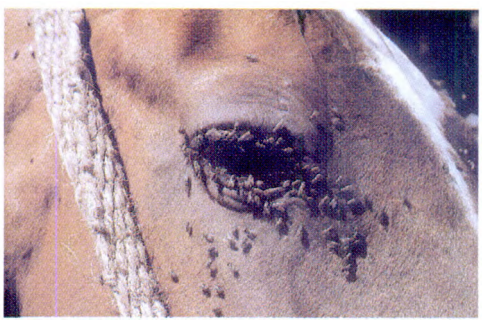

Fig. 487 "Fly worry" due to muscid flies (e.g. *Musca autumnalis*) [37]

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also act as intermediate host for other parasites. *Musca domestica* (non-biting muscid) is attracted to wounds and moist parts of the body, especially the eyes where they may provoke an ulcerative dermatitis. *M. domestica* also act as vector of *Habronema* spp. (*H. muscae* and *H. megastoma*), *Thelazia* spp. and *Haematobia* spp. The horn flies are biting muscids and vectors of the filarial worm *Parafilaria multipapillosa* (☞ CATTLE, ■ 5.1).
(Figure 487)

Stomoxys calcitrans The stable fly

Significance: *S. calcitrans* also belongs to the muscids. It attacks a wide variety of mammals including horses. It is a biting muscid and occurs world-wide. Both sexes of this fly are bloodsuckers and can become extremely irritating pests of man and domestic animals. *S. calcitrans* acts as intermediate host of *Habronema microstoma* which lives in the stomach of horses and causes intense worry and irritation to animals, toxic reactions and immunosuppression (☞ CATTLE, ■ 5.1).

GLOSSINIDAE

Glossina spp. Tsetse flies

Significance: Both sexes suck blood and are equally capable or transmitting trypanosomes. Tsetse flies, especially in great numbers can significantly contribute to the fly-worry in horse populations (☞ CATTLE, ■ 5.1).

CALLIPHORIDAE
The blow flies and their allies

They are highly important in many domestic animal species and man. The adults are free-

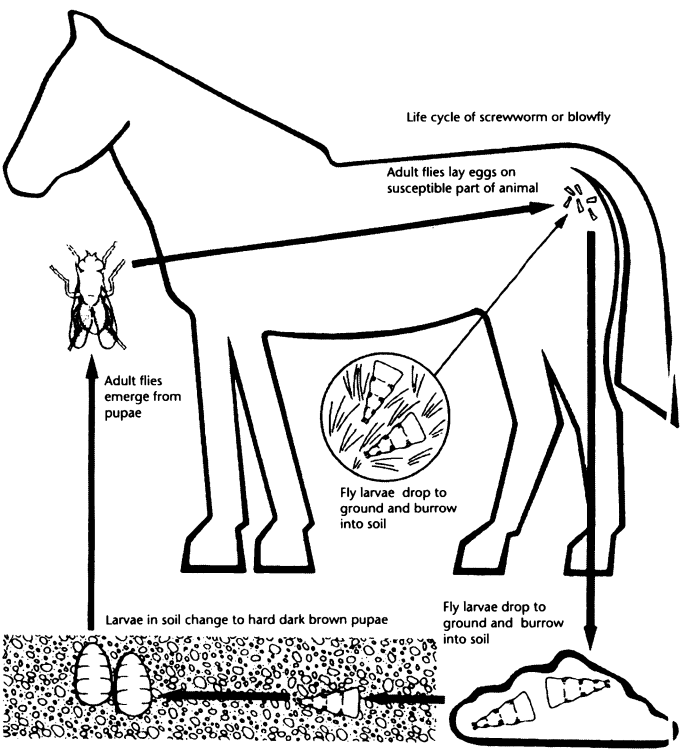

Fig. 488 Life cycle of screwworm or blowfly [36]

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living and the larvae are parasitic maggots which develop in the tissue of their host, causing a condition called myiasis. The larvae may be laid into pre-existing wounds. Myiasis is often a secondary skin problem.

The family of Calliphoridae can be divided into 2 groups: ***metallic- and non-metallic flies***

Metallic flies are green, blue or purplish coloured and belong to the genera *Chrysomya, Lucilia* and *Calliphora*.

Lucilia and *Chrysomya* are the most important genera responsible for sheep myiasis (☞ SHEEP AND GOATS, 5.1).

Chrysomya bezziana The cattle screwworm

Remarks: *C. bezziana* infests cattle but also horses, sheep, dogs and sometimes man. It occurs in tropical and southern Africa. *C. bezziana* causes severe myiasis and toxins produced by the larvae result in retarded healing of wounds. Death may occur in severe cases (☞ CATTLE, ■ 5.1).
(Figure 488)

Callitroga (syn. *Cochliomyia*) ***hominivorax***
The "American screwworm"

Remarks: *C. hominivorax* occurs since recently also in North Africa. Cattle, pigs and equines suffer most frequently, but other animals, including fowls and even man may also be affected. Pathology is essentially the same as in *C. bezziana* (☞ CATTLE, ■ 5.1).

Non-metallic flies are dull grey, yellow-brown or black and belong to the genera *Wohlfartia* and *Sarcophaga*.

Sarcophaga haemorrhoidalis
The red-tailed flesh-fly

Remarks: *S. haemorrhoidalis* sometimes causes myiasis in man and animals (☞ CATTLE, ■ 5.1).

Wohlfartia magnifica
The Old World flesh fly

Remarks: *W. magnifica* may cause myiasis (☞ CATTLE, ■ 5.1).

OESTRIDAE

Rhinoestrus purpureus

Remarks: Life cycle similar to those of the genus *Oestrus*. Larval stages are obligatory parasites of horses and mules in tropical Africa. Larvae are deposited by the adult fly in batches of 8–40 into the nostrils or eyes. The larvae crawl into the nasal cavity where they remain for several months before moving to the sinuses where they mature. They cause obstruction, irritation and sneezing (☞ HORSES AND DONKEYS, ■ 4.3).
(Figure 489)

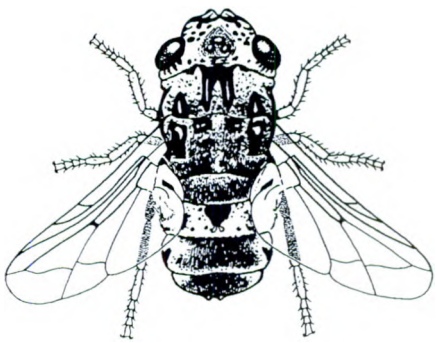

Fig. 489 *Rhinoestrus purpureus*; adult fly (8–11 mm long) [33]

Hypoderma bovis Cattle grub, warble fly

Remarks: *Hypoderma bovis* is of minor significance in horses, except where horses are kept within cattle populations in areas which are heavily infested with *H. bovis*. The adult flies constitute serious annoyance for horses. Subcutaneous larvae damage the skin and underlying tissue. Allergic reactions to these grubs may occur. The back of

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infested horses becomes irritated, thickened and disfigured and the breathing holes are visible in the skin (☞ CATTLE, ■ 5.1). (Figure 490)

Fig. 490 *Hypoderma bovis*; adult fly (15 mm long) is of minor significance in horses, except where horses are kept within cattle populations in areas with a high *H. bovis* density [19]

GASTROPHILIDAE Bot flies

Gasterophilus (syn. *Gastrophilus*) spp. Horse bot flies

Remarks: The larvae of these flies are gastrointestinal parasites of equines. After hatching, the first larval instars burrow into dermal layers of the mouth cavity, from which they move to either the pharynx, stomach or rectum, where the second- and third-stage larvae attach. The larvae feed on tissue exudates and remain in the host up to 1 year before they become mature and are excreted with the faeces to pupate in the soil. The adult flies are brown in colour and hairy and resemble bees which lay their eggs onto the skin of their host (☞ HORSES AND DONKEYS, 1).

Gasterophilus intestinalis (syn. *G. equi*) Common horse bot fly

Remarks: *G. intestinalis* occurs in horse and donkeys global. Female flies deposit their eggs mainly on forelegs, between the knee and the hoof but also in the scapular region where they are licked by the host which stimulates hatching (8–18 mm). (Figure 491)

Fig. 491 *Gasterophilus* sp.; adult fly

Gasterophilus nasalis Throat bot fly

Remarks: *G. nasalis* eggs are laid in the intermandibulary space up to 500 and usually 1 per hair.

Gastrophilus pecorum

Remarks: *G. pecorum* eggs are laid on the leaves of plants and its larvae are ingested by grazing animals.

Gasterophilus haemorrhoidalis The nose or lip bot fly

Remarks: *G. haemorrhoidalis* eggs are laid on hairs of the lips and around the mouth. The third-stage larvae attach in the hosts rectum, sometimes in great numbers.

- **General features of Gasterophilus spp.**

Significance: 1) The adult flies distress their equine host markedly up to the point of causing stampeding.
2) Irritation by the larvae during migration, e.g. dermatitis, inflammation of the pharynx, oesophagus, stomach or rectum (rectum prolapse).
3) Large numbers in the pyloric region may interfere with the sphincter function.

Therapy and Prophylaxis: ☞ HORSES AND DONKEYS, 1

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Fig. 492 *Hippobosca equi* (up to 8 mm long) [29]

HIPPOBOSCIDAE

***Hippobosca* spp.** Louse flies
(Figure 492)
Hippobosca equina The horse louse fly

Hosts: Horse and cattle
Geographic distribution: World-wide
(Figure 493)

Fig. 493 *Hippobosca equina* (up to 8 mm long); attached to the skin

Hippobosca variegata

Hosts: Horse and cattle
Geographic distribution: Afrotropical and Oriental regions

Hippobosca rufipes The cattle louse fly

Hosts: Wild and domestic Bovidae, less frequent in horses
Geographic distribution: Afrotropical regions (warm arid and semi arid areas of Africa)

Hippobosca maculata
Horse and cattle louse fly

Hosts: Horse and cattle
Geographic distribution: Tropical and subtropical areas of Africa

- **General features of *Hippobosca* spp.**

Location: The adult louse flies remain for long periods on their host and cluster in the perineal region, between the hind-legs to the pubic region, but may also bite on other parts of the body.
Species description: The flies live permanently on their host and feed on blood. Mainly cattle and horses are affected. They rarely fly and usually not more than a few metres. They spend their whole time on the host and are strongly attached to the hairs of their host and therefore difficult to dislodge. When disturbed they can quickly move sideways.
Symptoms: Infested animals scratch and rub and skin-trauma is often seen as a consequence of heavy infestations.
Significance: These flies are a source of great irritation.
Diagnosis: Identification of flies located under the tail and between the hindlegs.
Therapy: Infested animals should be treated topically with insecticides (☞ THERAPY AND PROPHYLAXIS OF ECTOPARASITES).
Prophylaxis: Regular grooming may be helpful to reduce louse fly infestations.

- **Therapy and prophylaxis of ectoparasites (arachnids and insects)**

A great number of acaricides and insecticides is used to control both arachnids and insects. Methods of application include whole-body sprays, dips, dusts and topical application to the dermis and ears. Dips are more effective for ticks. Thorough treatment of the infested area is required, especially when ticks infest the ears or underside of the tail or when mites infest localized patches on the skin. Elimination of parasites or stages in the environment is difficult because

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Table 18 Acaricides or insecticides approved for the control of ticks, flies and lice in horses (☞ Table 11, CATTLE, ■ 5.1)

Organophosphates	
	Bromocyclene
	Coumaphos
	Crotoxyphos + dichlorvos
	Diazinon
	Dichlorvos
	Malathion
	Phoxim
	Propoxur
	Stirofos
	Trichlorfon (metrifonat)
Carbamates	
	Carbaryl
Chlorinated Hydrocarbons	
	Lindane
	Methoxychlor
Pyrethrins and Pyrethroids	
	Flumethrin
	Cypermethrin
	Fenvalerate
	Permethrin
	Pyrethrin
Avermectins	
	Ivermectin

many ectoparasites are capable of surviving in the environment for prolonged periods (e.g. some ticks can live on the ground for more than 300 days without feeding). In areas where many ticks exist reinfection of the host occurs continuously and treatment therefore must be repeated regularly. Some ticks are not strictly species-specific and ticks normally adapted to horses can occasionally also affect cattle or other animals and they must be treated, too, if attempts are made to reduce overall tick infestations.

In subhumid areas the period of highest tick activities is the wet season and only few ticks are found on animals during the dry season. Consequently tick control is focussed on the wet season. In tropical areas most ticks are active throughout the year and must be controlled continuously.

5.2 Eyes

HELMINTHS

- **Nematoda found in the eyes**

Thelazia lacrymalis Eyeworm

Location: Tear ducts and conjunctival sacs

Hosts: Horse

Species description: Viviparous female sheds larvae into the tear secretions. Larvae are picked up by the intermediate hosts which are several species of muscid flies. Transmission occurs when flies feed on the eyes of horses.

Geographic distribution: World-wide

Symptoms: Chronic conjunctivitis, lacrimation, swollen eyes, covered with exudate and pus. Kertatitis, opacity of the cornea and in severe cases ulceration of the cornea.

Significance: *T. lacrymalis* is very common in some areas but only heavy infections cause clinical problems. Irritation of the eyes followed by bacterial secondary infections may occur.

Diagnosis: Detection of adult worms in the conjunctival sacs. Examination of the lacrimal secretion may reveal first-stage larvae.

Differential Diagnosis: Infective larvae of *Habronema* spp. may produce ulcerative granulomas near the medial canthus of the eyelid. *Onchocerca* spp. larvae invade the eye and may also cause conjunctivitis. Small (< 1 mm), raised, white nodules in the pigmented conjunctiva are pathognomonic for *Onchocerca* infections.

Therapy: Fenbendazole (10 mg/kg, po.) administered for 5 days is efficacious against *T. lacrymalis*. Ivermectin (200 µg/kg, sc.) or levamisole (5 mg/kg., po.) or directly into the conjunctival sac have limited effect against *T. lacrymalis*. Mechanical removal with forceps following instillation of a local

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Fig. 494 *Thelazia lacrymalis*; anterior end with typical cuticular striation (62 and 63), posterior end of a male (64), posterior end of a female (65) and tip of the tail (66); females are up to 18 mm long and males are up to 11 mm long [40]

anaesthetic may be helpful. Concurrent use of an antibiotic suspension against secondary invaders is recommended.

Prophylaxis: Fly control measures, directed especially against the face fly, may help to control thelaziosis.

(Figure 494)

Setaria equina

Remarks: Microfilariae may occasionally be found in the eyes (☞ HORSES AND DONKEYS, ■ 4.4).

Rhinoestrus purpureus

Remarks: Larvae are often deposited in batches of 8–40 at a time into and around the eyes and may occasionally cause "ophtalmomyiasis". Larvae may be found in the eye exudates (☞ HORSES AND DONKEYS, ■ 4.3).

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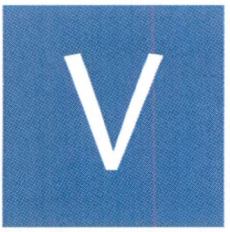

Parasites of Dromedaries*

CONTENTS

* In this chapter main emphasis is given to the description of the parasites of the one-humped camel (*Camelus dromedarius*). Some remarks will be made to parasites which are also important for the two-humped camel (*Camelus bactrianus*).

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Fig. 495 Oocyst of *Eimeria cameli* (80–100 × 62–94 µm) [4]

HELMINTHS

Eimeria spp. Coccidiosis of camels

Eimeria cameli

Location: Small intestine, rarely caecum
Hosts: Dromedary and Bactrian camel
Species description: This is the most frequently encountered *Eimeria* species in camels in North Africa. The oocysts are large and measure 80–100 × 62–94 µm. A large micropyle (10–27 µm wide) is characteristic. Developmental stages occur in the small intestine. Giant schizonts up to 350 µm in diameter may be seen. Inflammatory lesions of the small intestine have been associated with this species.
Geographic distribution: World-wide
(Figures 495, 496)

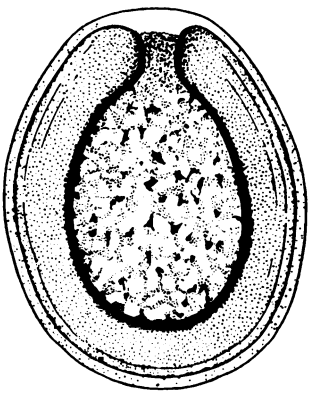

Fig. 496 Oocyst of *Eimeria cameli* (schematic) [42]

Eimeria dromedarii

Location: Small intestine
Hosts: Dromedary and Bactrian camel
Species description: This species is found frequently, often together with *E. cameli*. The oocysts are much smaller than those of *E. cameli*, measuring 23–33 × 20–25 µm.
Geographic distribution: World-wide, especially Iraq, Pakistan, India

- **General features of coccidiosis in camels**

Symptoms: Inappetence and severe enteritis due to *Eimeria* spp. have been observed. Progressive rapid weight loss and emaciation may result. Watery diarrhoea sometimes containing blood has been found in heavy infections. Dehydration and secondary

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infections may severely aggravate the disease and cause mortalities in young camels.

Significance: Coccidiosis in camels may cause high mortalities especially in young camels.

Diagnosis: This is based on the clinical signs and the demonstration of high numbers of oocysts in the diarrhoeic faeces. Cystic structures containing immature oocysts occur in the intestinal mucosa.

Therapy and Prophylaxis: ☞ CATTLE, 1; COCCIDIOSIS OF CATTLE, p. 24 ff.

Remarks: Young camels are much more susceptible to infections. Watering devices should be protected from faecal contamination.

Other *Eimeria* species harboured by camels (one- and two-humped camels) are: *Eimeria bactriani* (oocysts: 21–34 × 20–28 µm, CIS), *Eimeria rajasthani* (34–39 × 25–27 µm, India) and *Eimeria nölleri*. Two species of *Isospora* occur in camels: *Isospora orlovi* (27–35 × 15–20 µm) and *Isospora cameli*. In this genus oocysts contain 2 sporocysts, each of which contains 4 sporozoites. The pathogenic role of *Isospora* spp. in camels is unknown.

Cryptosporidium parvum (syn. *C. bovis*)

Remarks: Cryptosporidiosis may affect young camels. Severe diarrhoea, emaciation, dehydration and death may occur (☞ CATTLE, 1). This protozoan parasite is often superimposed on a primary immunosuppression. Diagnosis is based on the demonstration of oocysts in carbol-fuchsin stained smears of fresh faeces (☞ CATTLE, 1).

Balantidium coli

Remarks: Cases of clinical balantidiosis have been reported from camels. Diarrhoea and emaciation and faeces containing 300 cysts per gram were found. However, it is likely that *Balantidium* spp. play a secondary role, superimposed on a massive irritation of the intestinal mucosa by other pathogens or following immunosuppression. Therapy is rarely indicated. Oxytetracycline (5–10 mg/kg, im. or iv.) may support recovery (☞ Swine, 1 p. 295).

HELMINTHS

- **Trematoda eggs found in the faeces and adult trematodes living in the gastrointestinal tract**

Paramphistomatidae Rumen flukes

Remarks: Various species of *Paramphistomum* which also occur in other domestic and wild ruminants (☞ CATTLE, 1) have been found in camels. Rumen flukes normally do not cause clinical signs unless masses of immature worms attach to the intestinal mucosa.

- **Trematodes whose eggs may appear in the faeces**

Fasciola hepatica Liver flukes

Large liver fluke of temperate areas and high altitude regions in East Africa. This is one of the trematodes most frequently encountered in camels in Africa and Asia (☞ CATTLE, 1 and ■ 4.2 and DROMEDARIES, ■ 4.2).

Fasciola gigantica Giant liver fluke

Tropical large liver fluke; this species also occurs in camels in Asia and Africa (☞ CATTLE, 1 and ■ 4.2 and DROMEDARIES, ■ 4.2).

Dicrocoelium spp. Liver flukes

Location: Adult liver flukes in biliary ducts; eggs in the faeces

Remarks: This genus is very seldom found in camels.

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Eurytrema pancreaticum Pancreas fluke

Location: Adult pancreas flukes in pancreatic ducts; eggs in the faeces

Remarks: Camels are rarely affected by this trematode (☞ CATTLE, ■ 1 and 4.5; SHEEP AND GOATS, 4.5 and DROMEDARIES, ■ 4.5).

Schistosoma bovis and *Schistosoma mattheei* Blood flukes

Location: Adult blood flukes in mesenteric veins; eggs in the intestinal wall and faeces (☞ CATTLE ■ 1 and ■ 2 and DROMEDARIES, ■ 2).

- **Cestoda eggs found in the faeces and adult cestodes living in the gastrointestinal tract**

Seven species of cestodes, all belonging to the family Anoplocephalidae, have been identified in the small intestine of camels.

Moniezia expansa and *Moniezia benedeni* Common tapeworms

Remarks: *Moniezia expansa* is a cosmopolitan parasite which has been found in camels in Africa and Asia. *Moniezia benedeni* has been found in camels only in Africa (☞ CATTLE, ■ 1).

Stilesia globipunctata

Remarks: This is mainly a parasite of small ruminants. It also occurs in camels and is widespread in Africa. This parasite also has been reported from Pakistan and India. (☞ SHEEP AND GOATS, ■ 1)

Stilesia vittata

Remarks: This parasite is similar to *S. globipunctata*. It is 18–23 cm long and ≤ 1 mm wide. The scolex is 0.5–0.6 mm in size and the number of testicles is 10–14. Pathology and significance is the same as with *S. globipunctata*.
(Fig. 497, 498)

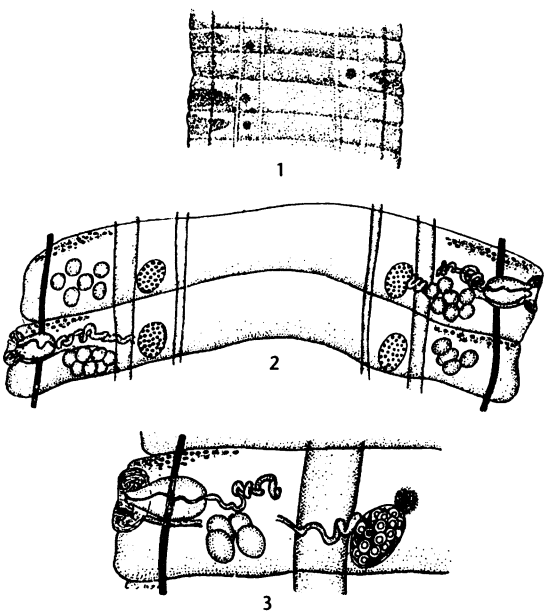

Fig. 497 *Stilesia vittata*; details of segments: (1) immature segment, (2) mature segments and (3) details of genitals [43]

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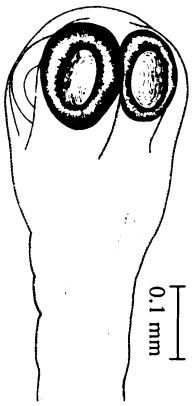

Fig. 498 *Stilesia vittata*; scolex [43]

Avitellina centripunctata and *Avitellina woodlandi*

Remarks: *A. centripunctata* is widespread in camels in Africa and Asia (☞ CATTLE, 1 and SHEEP AND GOATS, 1). *Avitellina woodlandi* has only been described in dromedaries from Africa.
(Figure 499)

Fig. 499 Tapeworms (*Avitellina* spp.) in the small intestine of a dromedary [13]

Thysaniezia ovilla (syn. *T. giardi*)

Remarks: This species has been found in dromedaries in Chad (☞ CATTLE, 1 and SHEEP AND GOATS, 1).

- **Therapy and prophylaxis of intestinal tapeworms of dromedaries**

Most of the drugs used against tape worm infections in cattle may be used for dromedaries (☞ CATTLE, 1).

- **Nematoda eggs found in the faeces, adult nematodes living in the gastrointestinal tract and first-stage larvae of *Dictyocaulus filaria***

Gongylonema pulchrum Gullet worm

Location: Oesophagus
Remarks: ☞ CATTLE, 1

Gongylonema verrucosum Rumen gullet worm

Location: Rumen
Remarks: ☞ CATTLE, 1

Parabronema skrjabini

Location: Abomasum
Remarks: This spirurid nematode occurs usually in small and large ruminants (☞ CATTLE, 1 and SHEEP AND GOATS, 1). In Africa it has been found in dromedaries in many countries.

Haemonchus longistipes

Location: Abomasum
Remarks: This species is specific to camels and occurs in Africa and Asia. In general, *H. longistipes* is very similar to *H. contortus* (☞ SHEEP AND GOATS, 1). The male is 18–28 mm long. The spicules are 534–664 µm long. The gubernaculum is 250–380 µm long. The female is 26–39 mm long, with a vulva which is not covered by a linguiform flap. The eggs are 60–80 × 69–50 µm. *H. longistipes* is an important parasite of camels, causing great losses, especially in young animals. It occurs mostly in mixed infections with other gastrointestinal nematodes. (Figure 500)

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Fig. 500 *Haemonchus longistipes* in the abomasum of a dromedary [13]

Haemonchus contortus Twisted wire worm

Location: Abomasum

Remarks: This nematode is frequently found in camels which have shared grazing with sheep (☞ CATTLE, 1 and SHEEP AND GOATS, 1).

Camelostrongylus mentulatus

Location: Abomasum

Remarks: This trichostrongylid is commonly found in camels in the north of the African continent and is rare elsewhere. This genus resembles *Ostertagia* except that the spicules have peculiar denticular structures extending in a longitudinal row throughout the entire length. The male is 6.5–7.5 mm long, with spicules 600–730 µm long. The gubernaculum is 68–82 µm long. The male bursa is composed of 2 large lateral lobes and a small dorsal lobe. There is an accessory bursal membrane. The female is 8–10 mm long. The eggs are of typical strongyle-type and measure 75–85 × 40–50 µm. *C. mentulatus* may cause great problems in camels and often occurs in mixed infections.
(Figure 501)

Ostertagia circumcincta and *Ostertagia trifurcata*

Location: Abomasum

Remarks: These two species are cosmopolitan parasites and best adapted to sheep and goat (☞ SHEEP AND GOATS, 1). However, they are also found in camels.

Impalaia tuberculata and *Impalaia nudicollis*

Location: Abomasum and sometimes small intestine

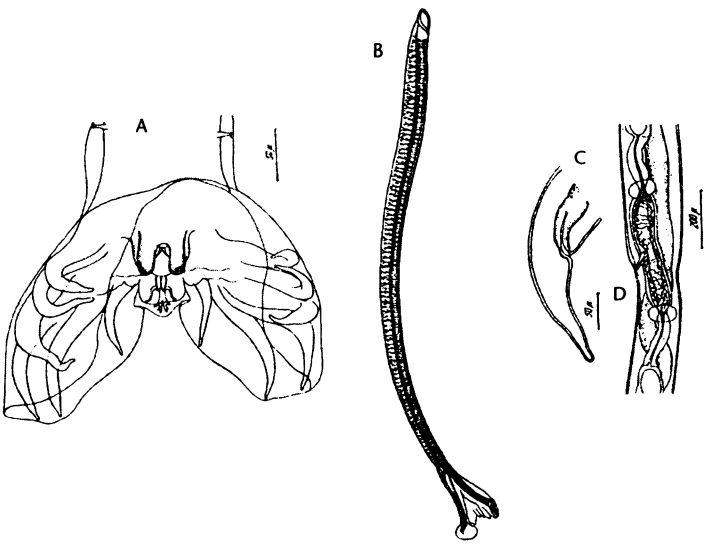

Fig. 501 *Camelostrongylus mentulatus*; bursa copulatrix (A), spicule (B), tail end of female (C) and vulva region (D) [9]

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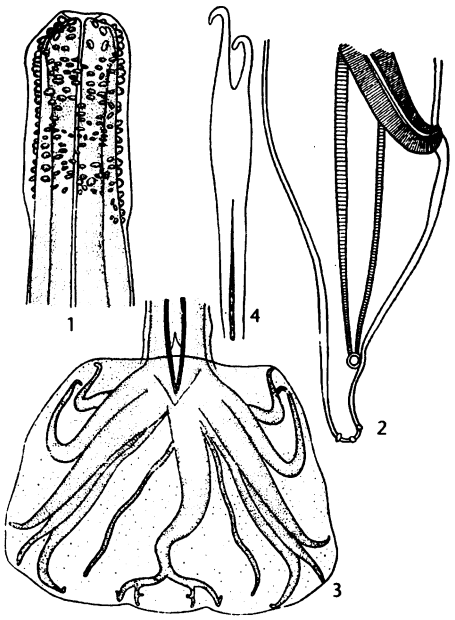

Fig. 502 *Impalaia tuberculata*; anterior end (1), posterior end of female (2), male bursa (3) and spicule (4) [44]

Remarks: These parasites belong to the Nematodirinae and have both been identified in dromedaries throughout Africa. *I. tuberculata* males are 7–9 mm long, with spicules about 600 µm long and a gubernaculum 9 µm long. The cervical cuticle is studded with small papillae. The female is 14–18 mm long. The eggs measure 60 × 32 µm. The males of *I. nudicollis* are 7.5–8.2 mm long, with spicules 910–980 µm long and a gubernaculum which is 94–100 µm long. The female is 14.8–16.7 mm long. The eggs are 63–74 × 39–46 µm. *I. aegyptiaca* has been found only in Egypt.
(Figure 502)

***Nematodirus* spp. (*N. dromedarii, N. spathiger, N. mauritanicus, N. abnormalis* and *N. helvetianus*)**

Location: Small intestine
Remarks: *N. spathiger* is the most prevalent species found in dromedaries (☞ CATTLE, 1 and SHEEP AND GOATS, 1). The males of *N. mauritanicus* are 13–15 mm long, with spicules 4.5–5.5 mm long, joined for a part of their length, with their tip enclosed in a thin, lanceolate membrane. The females are 21–24 mm long. The eggs measure 220–280 × 110 × 115 µm.
(Figure 503)

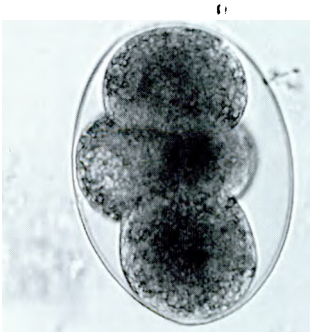

Fig. 503 Egg of *Nematodirus* sp. (220–280 × 110–115 µm)

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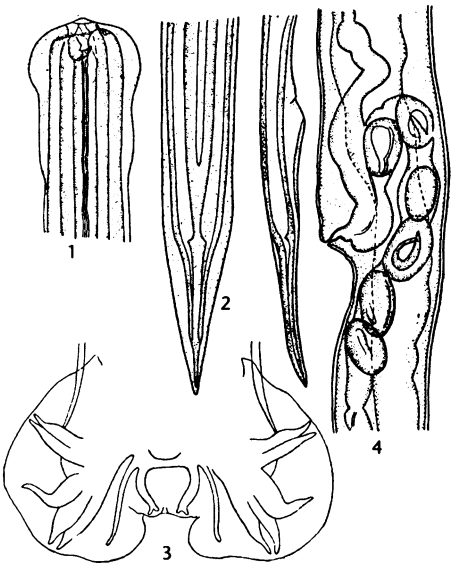

Fig. 504 *Nematodirella dromedarii*; head end with cephalic vesicle (1), tip of the spicule (2), male bursa (3) and vulva (4) [45]

Nematodirella dromedarii

Location: Small intestine
Remarks: This genus resembles *Nematodirus* but differs in that the vulva is in the anterior third of the body and the anterior branch of the female genital system is atrophied and sterile. The male is 10–15 mm long, with spicules 5.0–5.4 mm long. The female is 10–29 mm long. The eggs measure 230–260 × 100–120 µm.
(Figure 504)

Trichostrongylus axei

Location: Abomasum
Remarks: This species occurs in a wide range of hosts (☞ CATTLE, ▩ 1), including camels in Africa. High numbers cause massive irritation of the abomasal mucosa, reduced digestion and absorption of nutrients.

Trichostrongylus spp. *(T. colubriformis, T. probolurus, T. vitrinus* and other *Trichostrongylus* spp.)

Location: Small intestine
Remarks: *Trichostrongylus* spp. may occur in the small intestine of dromedaries in all parts of Africa. High numbers may cause massive inflammation and irritation of the duodenum, associated with diarrhoea which may be fatal in young camels. For therapy and control ☞ CATTLE, ■ 1 and SHEEP AND GOATS, ▩ 1.

Cooperia oncophora

Location: Small intestine
Remarks: *C. oncophora* may occasionally occur in dromedaries. Infections are often mixed with other trichostrongylids. *Cooperia* spp. have an extensive geographic distribution. For therapy and control ☞ CATTLE, ▩ 1 and SHEEP AND GOATS, ■ 1.

Lungworms whose first-stage larvae may be found in the faeces of the dromedary

DICTYOCAULIDAE

Dictyocaulus filaria Common lungworm

(☞ SHEEP AND GOATS, ■ 4.3 and DROMEDARIES, ■ 4.3)

Dictyocaulus cameli Camel lungworm

(☞ DROMEDARIES, ■ 4.3)

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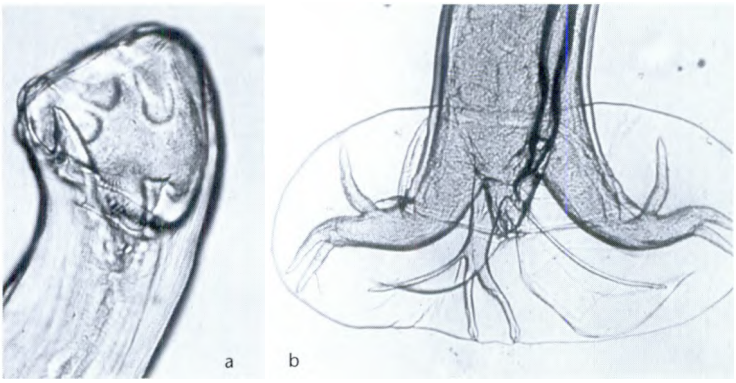

Fig. 505 *Bunostomum trigonocephalum*; anterior end (a) and male bursa (b) [4]

Bunostomum trigonocephalum Hookworm

Location: Small intestine
Remarks: This is essentially a parasite of small ruminants (☞ SHEEP AND GOATS, ■ 1), but it has been found in dromedaries in Africa and Asia. The male is 12–18 mm, and the female 22–25 mm long. It is a highly pathogenic species and may cause anaemia when present in high numbers.
(Figure 505)

Oesophagostomum columbianum Nodular worm

Location: Large intestine
Remarks: This is essentially a parasite of sheep and goats (☞ SHEEP AND GOATS, ■ 1) but it has also been found in dromedaries in Africa. Extensive nodule formation in the entire gastrointestinal tract is the predominant sign of this parasite.

Oesophagostomum venulosum Nodular worm

Location: Large intestine
Remarks: This is another nematode which occurs predominantly in small ruminants (☞ SHEEP AND GOATS, ■ 1). However, it may also be found in dromedaries in Africa and Asia.

Oesophagostomum vigintimembrum (syn. *O. venulosum*)

Location: Large intestine
Remarks: This species seems to be specific for dromedaries, but its frequency of occurrence is low.

Chabertia ovina "Large-mouthed bowel worm"

Location: Colon
Remarks: This is a parasite of ruminants, particularly sheep, encountered rarely in dromedaries (☞ SHEEP AND GOATS, ■ 1).

Trichuris ovis and *Trichuris globulosa* Whipworm

Location: Caecum
Remarks: *T. globulosa* is the commonest and most widespread trichurid of camels, reported from Africa and Asia. *T. ovis* is a cosmopolitan parasite of sheep, occasionally encountered in camels (☞ SHEEP AND GOATS, ■ 1). A number of other *Trichuris* spp. occur in camels elsewhere in the world: *T. cameli* and *T. raoi* (India), *T. skrjabini* (CIS), *T. affinis* (Asia). *Trichuris* spp. cause irritation and inflammation of the caecum and colon which often results in impaired water absorption and dehydration in heavy infections. For therapy and control ☞ below and SHEEP AND GOATS, ■ 1.

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Strongyloides papillosus

Location: Small intestine

Remarks: This nematode has been found frequently in dromedaries in Africa. Only the parthenogenetic females are parasitic and infection may be acquired by percutaneous penetration of infective larvae (☞ CATTLE, 1 and SHEEP AND GOATS, 1).

(Figure 506)

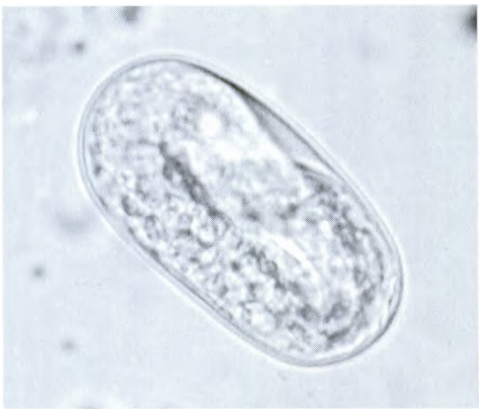

Fig. 506 Egg of *Strongyloides* sp. (47–65 × 25–26 µm)

- **General features of gastrointestinal nematode infections in dromedaries**

Although it seems that the environment in which camels live is conducive to the development and transmission of helminths, the helminth fauna is very rich and losses due to helminths play an important role in camel rearing. Under natural conditions camels are practically never infected with just a single species of gastrointestinal helminths; multiple parasitism is the rule. It is practically impossible to distinguish the diseases produced by different helminths with the exception of an acute haemonchosis which may cause a distinct disease entity. Haemonchosis caused by *H. longistipes* and/or *H. contortus* is the most severe gastrointestinal helminth infection of camels. Heavy infections occur more often in young camels and cause general weakness, anaemia, oedema of the hollow above the eyes, the sides of the sternal cushion (resulting in a characteristic swelling) and sometimes between the jaws. Progressive wasting and death may occur. Heavy infections are associated with a low PCV, albumin, calcium, phosphate, magnesium and copper level. Pathological findings include emaciation, ascites, hydrothorax and hydropericardium. The abomasum shows marked oedema, petechiae and sometimes erosions. However, clinical signs are often more pronounced in adult compared to adult camels (reverse age susceptibility), a phenomenon which was also described in context with ovine haemonchosis. Other helminth infections cause retarded growth, progressive wasting, alternation of constipation and diarrhoea. Anaemia only develops in a chronic stage of infection. *Oesophagostomum* spp. cause multiple nodule formation and *Trichuris* spp. cause a catarrhal enteritis.

Diagnosis: It is often possible to establish a diagnosis from the clinical signs of severe infections. By contrast, diagnosis can prove difficult in less severe cases. However, haemonchosis has to be distinguished from acute trypanosomosis which causes febrile episodes and a somnolent attitude, both normally absent from haemonchosis and infections with other gastrointestinal helminths. Faecal examination for worm eggs is usually helpful, although sometimes in peracute infections eggs are not present in the faeces. During the dry season some helminths undergo hypobiosis and cannot be detected by coprological methods during this period.

Therapy and Prophylaxis: Helminth infections are seasonal and usually concentrated to the rainy season when the preparasitic development may take place (☞ CATTLE, 1). Anthelmintic treatments during the rains may reduce the worm load and increase the growth rate which may be of vital importance for animals to survive the dry season. Most of the anthelmintics used in cattle and small ruminants for the treatment of gastrointestinal helminths may also be used in camels. Thiabendazole

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(100–150 mg/kg, po.), tetramisole (10 mg/kg, sc. or 15 mg/kg, po.), levamisole (7.5 mg/kg, sc.), morantel tartrate (3–5 mg/kg, po.) and pyrantel (25 mg/kg, po.) are effective against adult nematodes including *Dictyocaulus* spp. but they are not effective against hypobiotic larvae which occur during the dry season. For dry season treatments the modern benzimidazoles (albendazole, fenbendazole, mebendazole, netobimin, etc., ☞ CATTLE, 1) or ivermectin (200 µg/kg, sc.) may be used in order to eliminate also the hypobiotic larvae. A monthly treatment during the rainy season and an early dry season treatment with a larvicidal compound may markedly reduce the problems associated with helminth infections. Special attention should be paid to the weaned animals. Development of infective larvae on the ground may be reduced by keeping the ground around watering points as dry as possible and by using clean water for drinking purposes. (Figure 507)

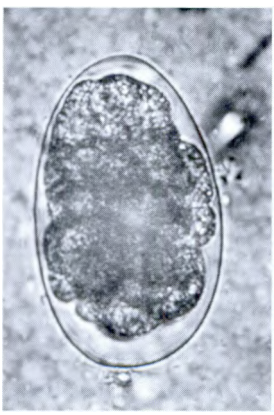

Fig. 507 Strongyle-type egg (70–100 × 30–45 µm) [13]

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2 Stages in the blood and circulatory system

PROTOZOA

TRYPANOSOMATIDAE

Trypanosoma evansi (syn. *T. brucei evansi*)
Surra, tahaga, djaffa, dbab

Hosts: Dromedary, equine, buffalo, carnivore and many other mammal species

Vector: Blood-sucking flies such as *Tabanus* spp., *Haematopota* spp., *Atylotus* spp., *Philoliche* spp. and occasionally *Stomoxys* spp., *Lyperosia* spp. and *Haematobia* spp.

Species description: *T. evansi* is not transmitted by tsetse flies. Several species of blood sucking flies act as vectors. No developmental stages have been demonstrated in the vectors which differentiate the parasite from *T. brucei*. *T. evansi* is 15–35 µm long and 1.5–2.5 µm wide. *T. evansi* is morphologically identical with *T. brucei* and other members of the subgenus *Trypanozoon*. It can be distinguished from *T. brucei* by isoenzyme electrophoresis and DNA probes. *T. evansi* is not restricted to the bloodstream and may enter the joint fluids and other tissue compartments. It may cross the blood-brain barrier. These extravascular trypanosomes are less accessible to chemotherapy. Surra can attack camels at any age. There is a hig incidence of infection in juvenile camels shortly after weaning.

Geographic distribution: Africa north of the Sahara desert, Asia, CIS

Symptoms: In a typical case the dromedary loses weight, develops drooping hump, is unable to walk long distances and may develop oedema of the feet, brisket, underbelly and eyelids. The coat becomes rough. During the initial attack of fever there may be lacrimation, shivering, reduced appetite and mild diarrhoea. The animal always shows progressive anaemia and fluctuating body temperature with initial peaks of fever up to 41°C. Later, in the chronic stage of infection the appetite is relatively unimpaired and the temperature may become normal or slightly elevated. The mucous membranes are pale and the PCV (particular cellular volume) drops below 25%, sometimes as low as 10%. The herders may notice a characteristic odour of the camel's urine and may identify infected animals by this sign alone. Abortion in all stages of pregnancy is common. Infected calves may be born alive but weak with parasitaemia. Death of the newborn calf is common. Reduced milk production, cases of blindness and central nervous lesions have been reported. The herd eventually reaches an endemic disease situation. Some animals may carry the trypanosomes for years, whereas others remain free of infection. Within such a group there are all forms and stages of surra, from new infections to subclinical and chronic conditions. Some animals live for up to 4 years with subclinical infections, some animals suddenly die due to other, superimposed infections and some eventually undergo self-cure. Numerous environmental and host factors influence the course of the disease, such as other infections, nutritional constraints, age, pregnancy, previous exposure or immunosuppression by other diseases and stress.

Significance: This trypanosome species causes the most widespread and most important disease in dromedaries. The overall productivity of a camel herd regarding calves, milk and weight gains is greatly impaired. Mortality may reach 20%.

Diagnosis: Trypanosomosis is diagnosed by demonstrating the parasite. However, dromedaries are usually far away from laboratory facilities. A tentative diagnosis

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can be reached by taking into account the owners observations such as weight loss, weakness, blindness, abortions, change of the odour of the urine and the clinical examinations of camels in the field. The predominant sign is anaemia, the mucous membranes being pale or white. Tachycardia may result. Postmortem examination reveals no absolutely typical signs. The carcass is pale and there are signs of dehydration, hypoproteinaemia, enlarged lymph nodes and splenomegaly. The direct methods of trypanosome detection utilize a wet blood film, a stained thick drop of blood and a thin bloodsmear. Concentration techniques such as the buffy coat technique under dark ground illumination provide both information about the level of anaemia (PCV) and the level of parasitaemia at the same time. For mass screening of dromedary herds there are numerous indirect tests. An immunofluorescent antibody test and ELISA tests have been successfully used for epidemiological surveys. A card agglutination test set has been introduced for the serodiagnosis of *T. evansi* infection in camels.

Therapy: Two drugs are recommended for the treatment of *T. evansi* infections in dromedaries: Quinapyramine as prosalt and Cymelarsan.

Cave: Most of the drugs for cattle trypanosomosis are either not curative (e.g. homidium bromide = Ethidium; pyrithidium bromide = Prothidium) or are too toxic for camels (e.g. diminazene aceturate = Berenil). Suramin and quinapyramine sulfate were successfully used but are no longer commercially available. Suramin (12 mg/kg or about 5 g/adult camel, given by slow intravenous injection) may cure infections and due to its slow elimination it has also a prophylactic effect for 6–12 weeks.

Cave: Suramin may cause phlebitis when administered paravenously. It is well tolerated up to 3-times the recommended dosage.

Quinapyramine methylsulfate (3–5 mg/kg, sc.) is used as a curative drug, whereas quinapyramine methylsulfate and quinapyramine chloride at a ratio of 3:2 (5–8.3 mg/kg, sc.) are applied for prophylactic purposes (duration 4–6 months). This compound is easier to dissolve and its subcutaneous application is easier.

Cave: Severe overdosing causes salivation and muscle tremor. Numerous *T. evansi* strains with a dual resistance to suramin and quinapyramine exist widely nowadays. In such cases isometamidium chloride (Samorin or Trypamidium, 0.5–0.7 mg/kg, iv., administered as a 2% solution) may be used. Unfortunately it has only a moderate effect against *T. evansi* and its use should be restricted to emergencies where dual resistance occurs. Isometamidium is only curative in acute cases when trypanosomes are still intravascular but it fails when they are extravascular. A recently developed arsenical (Cymelarsan®, 0.25 mg/kg, im.) is highly effective against *T. evansi* which are resistant to other drugs. It may be used for the treatment of acute and chronic *T. evansi* infections in camels.

Prophylaxis: There is no vaccine and vector control is difficult. Therefore, the control of *T. evansi* in camels relies entirely on drug therapy.

(Figures 508, 509, 510, Table 6)

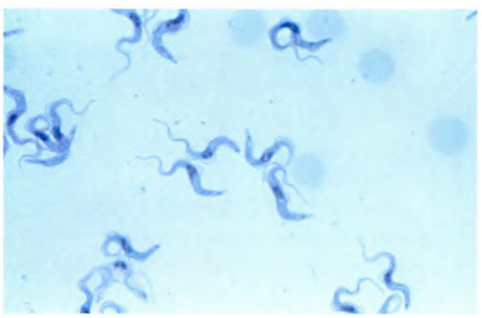

Fig. 508 *Trypanosoma evansi*; stained bloodsmear (15–35 × 1.5–2.5 µm) [4]

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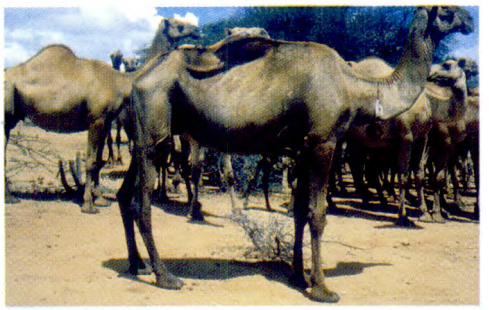

Fig. 509 Weight loss and emaciation due to a chronic *Trypanosoma evansi* infection [46]

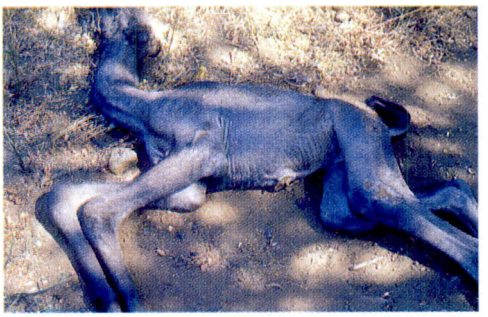

Fig. 510 Weakness of a newborn dromedary due to *Trypanosoma evansi* infection of the dam [46]

Trypanosoma congolense, Trypanosoma vivax and *Trypanosoma brucei*

Nagana, sleeping sickness

Remarks: Tsetse-transmitted trypanosomosis occurs in cattle, horse, camel, sheep and goat, and many other domestic and wild animal species (☞ CATTLE, ■ 2, SHEEP AND GOATS, ■ 2 and HORSES AND DONKEYS, ■ 2). Most camels live outside the tsetse belt and their trypanosomosis is caused by *T. evansi*. Camels are also affected to a lesser extent by tsetse-transmitted trypanosome species such as *T. brucei* and *T. congolense* which cause an acute disease with very high mortalities. Tsetse flies may have prevented the movement of camels into central Africa. Reports on the pathogenicity of *T. vivax* and *T. simiae* in camels are contradictory. For diagnosis ☞ CATTLE, ■ 2. For therapy and prophylaxis ☞ *T. evansi*.

THEILERIIDAE

Theileria camelensis

Remarks: This is a non-pathogenic *Theileria* species found in erythrocytes of camels in Egypt and Eritrea. The vector is *Hyalomma dromedarii* (☞ CATTLE, ■ 2)
(Figures 511, 512)

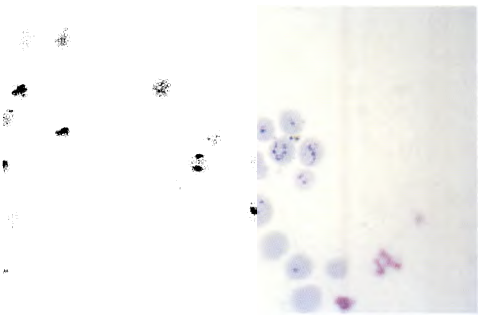

Fig. 511 *Theileria camelensis*; erythrocyte forms [15]

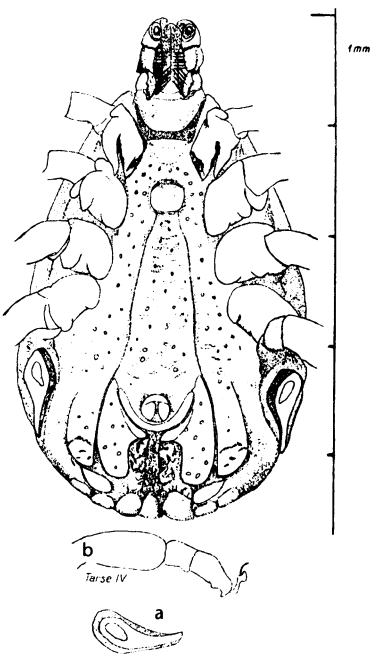

Fig. 512 *Hyalomma dromedarii*, the vector of *Theileria camelensis*; ventral view of a male, stigmata (a) and tarsus of leg IV (b) [9]

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RICKETTSIALES

ANAPLASMATACEAE

Anaplasma marginale

Remarks: *A. marginale* may also be found in camels. Little is known about the pathogenic role in camels. It may be important in immunosuppressed animals (☞ CATTLE, ■ 2).

Cowdria ruminantium Heartwater

Remarks: *Amblyomma lepidum* and *A. gemma* are the vectors of the disease in East Africa (☞ CATTLE, ■ 2 and DROMEDARIES, ■ 5.1).

HELMINTHS

- Trematoda found in the blood and circulatory system

Schistosoma bovis and *Schistosoma mattheei*
Blood flukes, bilharziosis

Remarks: S. bovis occurs in the portal and mesenteric veins of cattle, sheep and goats, equines and dromedaries in central, eastern and western Africa. The eggs are spindle-shaped and partially passed in the faeces. The size of the eggs is 132–247 by 38–60 µm. S. mattheei occurs in the portal and mesenteric veins and less frequently in cystic and lung veins of cattle, sheep, goats, wild ruminants, equines and dromedaries in northern and Central Africa. There are usually no specific clinical signs of infection (☞ Cattle, ■ 2 and Sheep and Goats, ■ 2).

- Nematoda found in the blood and circulatory system

Dipetalonema evansi
(syn. *Deraiophoronema evansi*)

Remarks: This filarial nematode is specific for camels. It develops in the heart and in hepatic, pulmonary and spermatic arteries as well as in lymph nodes and lymph vessels. It has been found in eastern and northern Africa. The vectors are mosquitos of the genus *Aedes*. The males are 8–11 cm long and the females 14.5–18.5 cm. The microfilariae are ensheathed and measure 200–315 µm and occur in the peripheral blood. Light infections are inapparent. However, massive infections cause emaciation, apathy and sometimes orchitis and nervous symptoms. Filarial nematodes within arteries cannot be detected clinically. Any clinical manifestation can be confused with trypanosomosis, although the latter is accompanied by febrile episodes which are absent from filarial infections. Fouadin (stibophen, 0.5 mg/kg, iv.) was effective for both therapeutic and prophylactic purposes as prevention during the period of activity of the mosquito's intermediate hosts. Ivermectin (200 µg/kg, sc.) may eliminate the microfilariae.
(Figure 513)

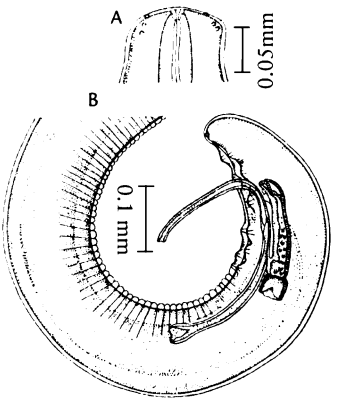

Fig. 513 *Dipetalonema evansi*; anterior end (A) and male posterior end (B) [9]

Onchocerca armillata Aortic filariosis

Remarks: This species develops within the aorta, particularly in cattle (☞ CATTLE, ■ 2). It has been found in this location in drom-

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edaries in Nigeria. This infections is difficult to diagnose and usually does not cause clinical signs. Therapy is usually not indicated.

3 Stages in the urogenital system

No pathogenic parasites have been found so far in the urogential system of dromedaries

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4.1 Locomotory system

4.1.1 Muscles

PROTOZOA

Sarcocystis cameli

Remarks: This species occurs commonly in North Africa (Egypt, Morocco, Sudan). The parasite is widespread, a high percentage of camels investigated at slaughter being infected. However, the pathogenic role of *S. cameli* is uncertain. Myocardial lesions have been attributed to *Sarcocystis* spp. in camels. In another study almost 50% of emaciated camels killed at Cairo abattoir showed sarcocysts. The definitive host is the dog. The prepatent period in dogs is 10–14 days, the patent period 69–73 days. The sarcocysts are up to 12 mm long and 2 mm in diameter. The bradyzoites are banana shaped, 15–20 × 4–6 µm. General life cycle of *Sarcocystis* spp. ☞ CATTLE, ■ 4.The antemortem diagnosis is difficult and therapy is usually not indicated. Infected, raw meat of camels is the only source of infection for dogs. If the overall incidence of sarcosporidiosis is to be reduced, the life cycle has to be interrupted at the stage where the definitive host (dog) has access to infected meat of the intermediate host (camel).

Toxoplasma gondii

Remarks: Tissue cysts of *T. gondii* may also occur in camels. A diagnosis based on clinical signs is not possible. Cysts may be seen in histopathological examinations. The chronic stage of infection is usually inapparent. The infection has recently been detected in camels by means of serological

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tests such as the Sabin-Feldman dye test, complement fixation, indirect immunofluorescence and indirect haemagglutination. The prevalence ranged from 3% to 67%. A heavy infection with a virulent strain may cause severe, fatal illness. Inappetence, lacrimation and polypnoea may be seen during the course of infection. For the general life cycle of *Toxoplasma gondii* ☞ SWINE, ■ 4.1. Felidae act as definitive hosts and many mammal species including man may be intermediate hosts. Raw, infected meat of camels may also be a source of infection for man and certainly for cats. Prevention is based on the interruption of this complex life cycle.

HELMINTHS

- **Cestoda larvae found in the muscles**

The following two cysticerci are found in the muscles of dromedaries.

Cysticercus dromedarii (syn. *C. cameli*)

Remarks: The larvae (*Cysticercus dromedarii*) of a hyena tapeworm (*Taenia hyaenae*) are frequently found in the muscles of the dromedary, cattle and goats. Antelopes are the usual intermediate hosts. They are rarely found in sheep. *C. dromedarii* cysts are twice as large as those of *C. bovis*, measuring

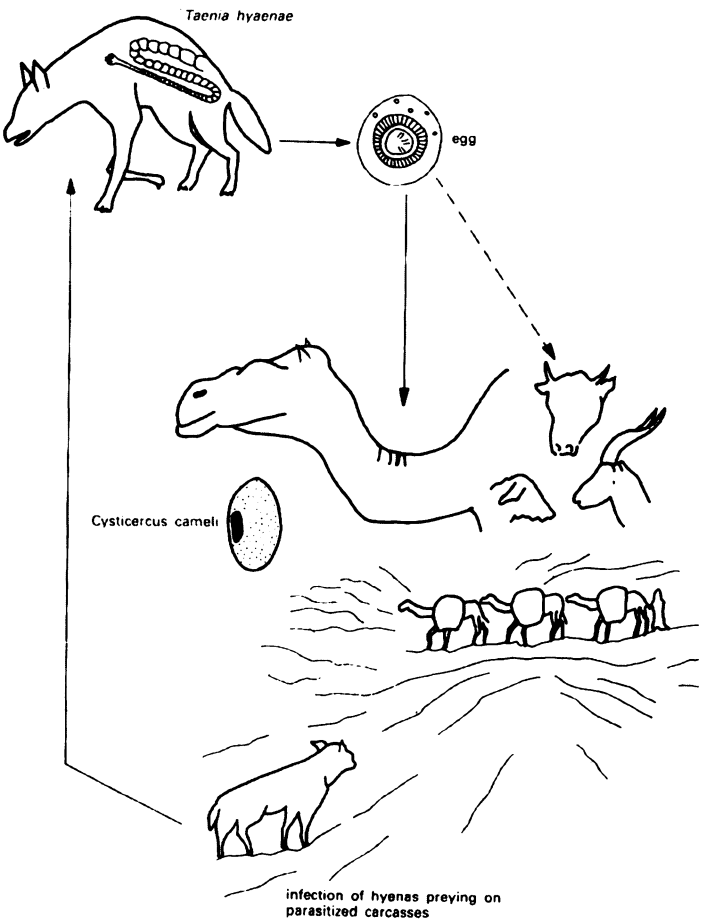

Fig. 514 Life cycle of *Cysticercus dromedarii*; hyena infected with the tapeworm *Taenia hyaenae* act as final host. Excreted eggs cause the development of cysticerci in the dromedary [16]

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12–18 mm in length. They are pearly white and the lateral invaginated protoscolex has a double row of hooks. Although not dangerous to man, their presence is repugnant, and parasitized meat has to be condemned. (Figure 514)

Cysticercus bovis

Remarks: This is the larval stage of *Taenia saginata*, a tapeworm of man. Bovines are the usual intermediate hosts (☞ CATTLE, ■ 4.1.1), and infections of camels are very rare.

- **Prophylaxis of *Cysticercus* spp. infections in camels**

Infections of muscles with cysticerci are usually not associated with any detectable clinical signs. Prevention is based on the interruption of the life cycle which is in case of *C. cameli* almost impossible. In case of *C. bovis* the prevention of infection in camels may be achieved by anthelmintic treatments of infected human beings. Human infection is acquired by eating infected camel meat as well as beef. This may be prevented by a rigourous meat inspection, directed to the heart, masseter, muscles and tongue. Carcasses infected with cysticerci must be cooked sufficiently to destroy the cysticerci. Abattoir waste should be destroyed and not be given to dogs or left for wild carnivores.

4.1.2 Tendons

HELMINTHS

- **Nematoda larvae and adult nematodes found in the tendons**

Two species of filarial nematodes of the genus *Onchocerca* have been identified in camels (☞ CATTLE, ■ 5.1).

Onchocerca fasciata

Remarks: This parasite is specific for camels and occurs in the subcutaneous connective tissue and the nuchal ligament. It has been reported from Sudan, Ethiopia, Kenya and Mauritania. There is only fragmentary knowledge of the structure of this worm. The microfilariae are 200–285 µm long and 35–50 µm wide (mean 255 × 40 µm).

Onchocerca gutturosa

Remarks: This nematode develops in subcutaneous connective tissue, the nuchal ligament and other tissues, such as the scapular cartilage. It is mainly a parasite of cattle (☞ CATTLE, ■ 4.1.2), but it has also been found in camels.

- **General features of filarial nematode infections**

Affected animals have skin nodules on various parts of the body, particularly on the head and neck. The nodules are firm, not sensitive to the touch, and range from 0.5–4 cm in diameter. Microfilariae may be found in the surrounding skin or within the nodules. They are rarely found in the peripheral blood. The nodules have a fibrous capsule. The filaria can be seen when a nodule is cut open, perhaps accompanied by pus. The animals seem to tolerate these infections without clinical signs. Allergic skin reactions due to microfilariae may occur. Therapy is usually not indicated. Allergic dermatitis may successfully be treated with ivermectin (0.2 mg/kg, sc.).

4.2 Liver

HELMINTHS

- **Trematoda found in the liver**

Four species of trematodes parasitize the liver of camels.

Fasciola hepatica Common liver fluke

Remarks: This trematode is frequently encountered in camels in Africa and Asia

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in those regions where *F. hepatica* is very prevalent in the cattle population (☞ CATTLE, ■ 4.2).

Fasciola gigantica Giant liver fluke

Remarks: This species is often found in camels in Africa and Asia. *F. gigantica* is common in cattle in many parts of Africa (☞ CATTLE, ■ 4.2).

***Dicrocoelium* spp.** (*D. dendriticum* and *D. hospes*) Lancet flukes

Remarks: Lancet flukes are found very seldom in camels. In many parts of the world this parasite is very prevalent among cattle and small ruminants (☞ CATTLE, ■ 4.2).

Eurytrema pancreaticum Pancreas fluke

Remarks: This trematode normally occurs in the pancreatic ducts and less often in the bile ducts of ruminants (☞ CATTLE, ■ 4.5). Camels may also become infected.

- **General features of trematode infections of the liver**

Hepatic infections are generally subclinical in camels, with the exception of heavy *Fasciola* spp. infections. Chronic fasciolosis is the most common form in camels and other hosts (☞ CATTLE, ■ 4.2). The symptoms of chronic fasciolosis are generally associated with hepatic fibrosis and hyperplastic cholangitis. Anaemia, oedema (bottle jaw), digestive disturbances (constipation, diarrhoea) and cachexia develop gradually. Acute fasciolosis is less common than the chronic disease and occurs mainly in sheep (☞ SHEEP AND GOATS, ■ 4.2). It is basically a hepatitis caused by the simultaneous migration of large numbers of immature flukes. Sudden death may occur in acute fasciolosis.

Diagnosis: The oval, operculated, golden-brown eggs (130–150 × 65–90 µm) appear in the sediment of the faeces (☞ CATTLE, 1). The detection of the adult flukes in the liver at necropsy or slaughter is the most reliable method to confirm fasciolosis.

Therapy: Treatment is indicated in those rare cases when fasciolosis is diagnosed or expected to cause problems in camel herds. Several fasciolicidal drugs are currently available (☞ CATTLE, ■ 4.2) and may be used, although few publications refer to their use in camels. Triclabendazole (12 mg/kg, po.) is highly effective in cattle. Nitroxynil (10 mg/kg, sc.) and rafoxanide (7.5 mg/kg, po.) are drugs recommended for camels. These two anthelmintics are also effective, in the same dosage, against myiasis of the nasal cavity, particularly *Cephalopina titillator*, and bloodsucking nematodes. Albendazole (10 mg/kg, po.) is also effective against *Fasciola* spp.

- **Cestoda found in the liver**

Stilesia hepatica

Remarks: This cestode occurs occasionally in the bile ducts of camels. It is common in small ruminants in many parts of Africa (☞ SHEEP AND GOATS, ■ 4.2). Infections are usually inapparent. Heavy burdens were associated with cholangitis and cirrhosis. Therapy is not indicated.

Echinococcus granulosus
(syn. *E. polymorphus, E. unilocularis*)
Hydatid tapeworm, hydatidosis

Remarks: Larvae (hydatid cysts) of *E. granulosus* are found in the liver and the lungs of dromedary, cattle, sheep, goat, swine, horse, and other domestic animals and man (= intermediate hosts). Adult tapeworms are found in the small intestine of the dog, hyena and other related carnivores (= final hosts). Hydatids are localized mainly in the liver and lungs, but also in other organs of camels (☞ CATTLE, ■ 4.2 and ■ 4.3, and DROMEDARIES, ■ 4.3). The incidence of hydatidosis in camels is highest in North and East Africa. It also occurs

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in Central and West Africa with a rather low incidence, although in Nigeria infection rates of 50% have been reported. Both liver and lung hydatidosis in camels are usually asymptomatic. The diagnosis is based on postmortem findings. Therapy is usually not indicated. Although infections with hydatids do not have serious consequences for camels, echinococcosis is a major helminthosis with regard to the public health. It is therefore necessary to limit the threat to human health by excluding dogs from places where camels are slaughtered and by seizure and destruction of any organ bearing hydatid cysts at slaughter.

Prophylaxis: ☞ CATTLE, ■ 4.2 and ■ 4.3

Cysticercus tenuicollis

Remarks: This is the larval stage of *Taenia hydatigena*, a tapeworm which occurs in the small intestine of domestic and wild canidae. In camels these larvae develop on the liver surface and serosal surface of the abdominal cavity as they do in the preferred hosts sheep and cattle (☞ CATTLE, ■ 4.4 and SHEEP AND GOATS, ■ 4.4). *C. tenuicollis* may be found occasionally in camels at the abattoir. The infection is asymptomatic unless a large number of larvae migrate through the liver parenchyma. Blood loss and death may then occur (☞ SHEEP AND GOATS, ■ 4.4).

4.3 Respiratory system

HELMINTHS

- **Cestoda found in the lungs and trachea**

Echinococcus granulosus
Hydatid tapeworm, hydatidosis

Remarks: Hydatid cysts of *E. granulosus* are found in the liver and the lungs of dromedary and other domestic animals and man (= intermediate hosts). Lungs harbouring hydatid cysts near the surface show protuberances. Within the lung tissue they can be felt as hard lumps, not very mobile. (☞ DROMEDARIES, ■ 4.2 and CATTLE, ■ 4.2).

- **Nematoda found in the lungs and trachea**

Dictyocaulus filaria Parasitic bronchitis

Remarks: This lungworm is encountered most frequently in the respiratory tract of camels in Africa. It is also prevalent in Asia and Europe. Severe infections with *Dictyocaulus* spp. are accompanied by general depression, cough, and polypnoea, dyspnoea and rapid loss of condition. Apathy and anorexia may occur. The adult worms occur in the bronchi and bronchioles. The life cycle is direct. First-stage larvae are passed in the faeces and their demonstration is essential, together with the respiratory signs for the diagnosis (☞ SHEEP AND GOATS, ■ 4.3). The following anthelmintics are recommended to treat *D. filaria* infections in camels: albendazole (2.5 mg/kg, po.), febantel (7.5 mg/kg, po.), fenbendazole (10–15 mg/kg, po.), oxfendazole (7 mg/kg, po.), ivermectin (0.2 mg/kg, sc.) tetramisole (10 mg/kg, sc. or 15 mg/kg, po.) and levamisole (7.5 mg/kg, sc.). All of these compounds are also effective against the most important gastrointestinal nematodes of camels.

Dictyocaulus viviparus and *Dictyocaulus cameli* (syn. *D. viviparus*)

These species may rarely occur in camels (☞ CATTLE, ■ 4.3).

ARTHROPODS

- **Insecta larvae found in the respiratory system**

Cephalopina (Cephalopsis) titillator
Camel nasal botfly

Remarks: The adult fly occurs in subsaharan

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Africa, Middle East and Asia. The larvae migrate into the nasal cavity, frontal sinus and pharynx of camels. The larvae are similar to those of *Hypoderma bovis* and measure 25–35 × 8–9 mm. The larvae irritate and damage the mucosa markedly. Infestations may be associated with extensive sneezing, restlessness and head shaking. Severe conditions must be differentiated from central nervous disorders. Ivermectin (0.2 mg/kg, sc.) has shown variable efficacy against the larvae of *C. titillator*. Rafoxanide (7.5–10 mg/kg, po.) as a drench or bolus, trichlorfon (75 mg/kg, po. or as a drench) and nitroxynil (10–15 mg/kg, sc.) have been reported to be effective.

(Figures 515, 516, 517)

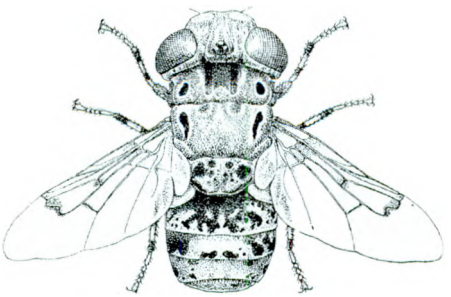

Fig. 515 *Cephalopina titillator* (camel nasal bot fly); adult [47]

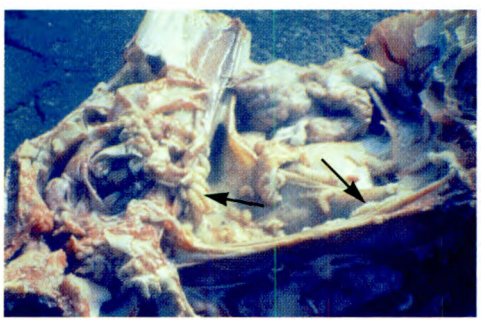

Fig. 516 Masses of nasal bot fly larvae (arrows) in the nasal cavity of a dromedary [13]

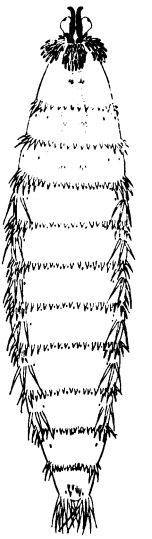

Fig. 517 *Cephalopina titillator*; larva [48]

Oestrus ovis

Remarks: *O. ovis* infestations in camels have been reported from Egypt (☞ SHEEP AND GOATS, ■ 4.3).

4.4 Abdominal cavity

HELMINTHS

- **Cestoda found in the abdominal cavity**

Cysticercus tenuicollis Larval stage of the canine tapeworm *Taenia hydatigena*

Remarks: Cysticerci are found attached to the omentum, the intestinal mesentery and to the serosal surface of abdominal organs, especially the liver. Adult tapeworms occur in the small intestine of dogs and other Canidae. Infections in camels and other intermediate hosts are usually inapparent (☞ DROMEDARIES, ■ 4.2 and SHEEP AND GOATS, ■ 4.4).

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4.5 Pancreas

HELMINTHS

- Trematoda found in the pancreas

Eurytrema pancreaticum Pancreas fluke

Remarks: This trematode normally occurs in the pancreatic ducts and less often in the bile ducts of ruminants. Camels may also become infected (☞ DROMEDARIES, ■ 4.2 and SHEEP AND GOATS, ■ 4.5).

4.6 Central nervous system

HELMINTHS

- Cestoda cysts found in the central nervous system

Coenurus cerebralis Larval stage of the canine tapeworm *Taenia multiceps* (syn. *Multiceps multiceps*); "gid", "stuggers", sturdy"

Remarks: The intermediate stage, a coenurus, develops in the brain and spinal cord of sheep and goat and sometimes camel. Adult *Taenia multiceps* occurs in the small intestine of the dog, fox, coyote, hyena and other Canidae (☞ SHEEP AND GOATS, ■ 4.6). *Coenurus* cysts cause increased intracranial pressure which is the cause of the neurological symptoms. Development of coenurus in the nervous system produces noticeable changes in behaviour, with diminution of appetite, even anorexia, depression, unwillingness to move or difficulty in walking. Animals may make circling in movements. Clinical diagnosis has to be distinguished from myiasis of the respiratory passages caused by *Cephalopina titillator* (☞ DROMEDARIES, ■ 4.3) and from rabies. The latter produces excitement, sneezing, snorting and sometimes abnormal behaviour. No drug therapy is available. Prophylaxis consists of destroying any coenurus cysts at slaughter, although this applies mainly to sheep, because the skull of camels is not usually opened at slaughter. The adult tapeworm in dogs can be controlled by using one of the cestodicidal anthelmintics such as praziquantel (5 mg/kg, po. or 5.7 mg/kg, sc. or im.), bunamidine hydrochloride (25–50 mg/kg, po.), nitroscanate (50 mg/kg, po.) or the combination of febantel/praziquantel/pyrantel (☞ SHEEP AND GOATS, ■ 4.6).

(Figure 518)

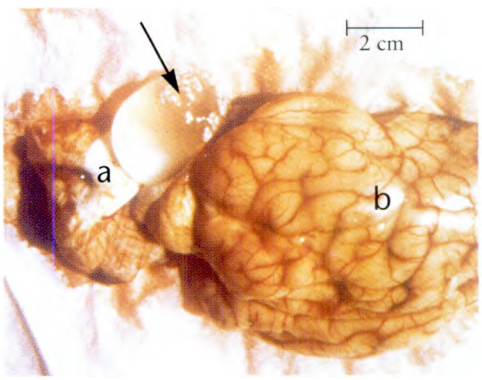

Fig. 518 Cyst of *Coenurus cerebralis* (arrow) located between the cerebellum (a) and cerebrum (b) [8]

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5 Stages on the body surface

5.1. Skin and coat

5.2 Eyes

5.1. Skin and coat

HELMINTHS

- Nematoda found in the skin and subcutis

Onchocerca fasciata

Remarks: This parasite is specific for camels and occurs in the subcutaneous connective tissue and the nuchal ligament. Skin nodules may appear but clinical signs are usually absent.

Onchocerca gibsoni

Remarks: The adult worms are found in subcutaneous nodules on the brisket, shoulders and external surfaces of the hindlimbs of cattle (☞ CATTLE, ■ 5.1) and occasionally of dromedaries. Microfilariae are found intradermally around the nodules with a predilection to the brisket region. Adult worms cause nodular swellings in the skin but infected animals are not clinically ill. The intermediate hosts are members of the genus *Culicoides* (e.g. *C. pungens* and other *Culicoides* spp.).

Stephanofilaria sp.

Remarks: *Stephanofilaria* spp. are small filarial parasites (family: Filariidae) which are responsible for circumscribed dermatitis (☞ CATTLE, ✎ 5.1). Several genera of the order Dipterida act as intermediate hosts. (Figure 519)

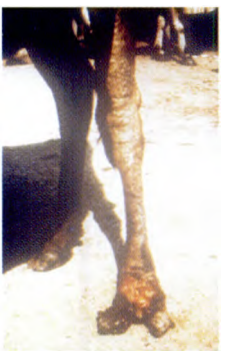

Fig. 519 *Stephanofilaria* sp. infection, causing a moist dermatitis on the leg of a camel [8]

ARTHROPODS

Arthropods are divided into two main groups:

Arachnida (arachnids) including:
- *Ticks*
- *Mites*

Insecta including:
- *Lice*
- *Fleas*
- *Dipterida*

- Arachnida found in/on the skin

– Ticks

The role of ticks as disease vectors (bunyaviruses, rickettsiae) in camels is minor. Routine prophylactic tick control is therefore not as widely practised in camel herds as it is done in cattle rearing systems. Nevertheless ticks suck blood and can cause severe anaemia and debil-

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ity in camels as well as paralysis (☞ CATTLE, ■ 5.1). Tick infestations are seasonal, occurring predominantly during the wet season. Heavy infestations should be treated with an acaricide. When animals bear a high number of ticks and, at the same time, show signs of paralysis or other non-specific clinical signs, acaricidal treatment should be applied. *Cowdria ruminantium* is transmitted by *Amblyomma lepidum* and *Amblyomma gemma* in East Africa. The non-pathogenic *Theileria camelensis* is transmitted by *Hyalomma dromedarii* (☞ DROMEDARIES, ■ 2).
(Figures 520, 521, 522, 523)

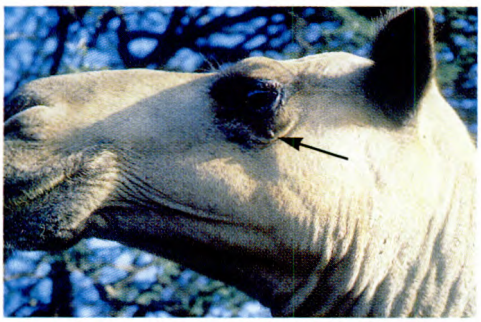

Fig. 520 Ticks affecting the eyelid of a dromedary [46]

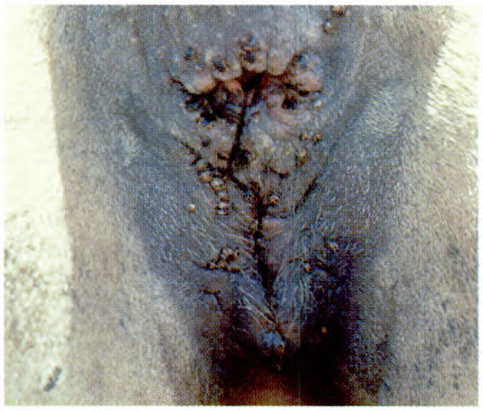

Fig. 521 Ticks affecting the vulva and perineal region of a dromedary [46]

The following ticks are commonly found on camels:

Fig. 522 *Amblyomma gemma*; female (2-5 mm × 4 mm) [13]

Fig. 523 *Amblyomma lepidum*; female (5 mm × 4 mm) [13]

IXODIDAE ("hard ticks")
Hyalomma dromedarii
Hyalomma rufipes and other *H.* spp.
(H. anatolicum, H. detritum, H. impressum and *H. plumbeum)*
Amblyomma lepidum, A. gemma, A. variegatum
Boophilus decoloratus
Rhipicephalus pulchellus, R. appendiculatus, R. sanguineus

A general description of each genus is made in CATTLE, ⁂ 5.1

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ARGASIDAE ("soft ticks")

Ornithodoros savignyi The sand tampan

Remarks: *O. savignyi* is a major pest of domestic stock in areas where it occurs. Its bites are painful and the saliva may cause tick toxicosis. The sand tampan may also cause allergic dermatitis in man. *O. savignyi* may severely irritate camels (☞ CATTLE, ■ 5.1 and SHEEP AND GOATS, ■ 5.1).

• **Tick control in camels**

Infested camels may be sprayed or doused with lindane (Gammatox) which has proved to be a highly effective and well-tolerated acaricide for camels. However, many of the modern ectoparasiticides recommended for cattle may also be used in camels. ☞ CATTLE ■ 5.1 THERAPY AND PROPHYLAXIS OF ECTOPARASITES (arachnids and insects) p. 141

– **Mites**

Sarcoptes scabiei var. *cameli*
Camel mange, "Jarab"

Location: Skin

Hosts: Dromedary and Bacterian camel

Species description: Mange has probably been the most common disease of camels. In the past when camels were important for civil and military transport, mange was regarded as a major disease. Animals were usually in poor condition and fatigued by long journeys and poor feeding. Unsatisfactory hygienic condition favoured the development and transmission of mange. Camel mange is caused by *S. scabiei* var. *cameli*, a mite almost exclusively confined to the genus *Camelus*. However, it has been reported that human beings may also be infected by this mite. The life cycle of the mite lasts for 4–5 weeks. Fertilized females burrow into the epidermis, causing inflammation and intense pruritus. Direct transmission occurs between animals. Infestations can also be transmitted indirectly via objects (e.g. harness, tents, luggage, etc.) which have been in contact with an infested camel or via soil (resting place). The mites survive off the host for a maximum of 2 weeks. The development and spread of camel mange may be favoured by factors such as poor nutrition, poor hygiene, stress (long journeys) and other diseases (trypanosomosis). Mange may be more acute during the cold season and during rainy periods. Both very young and very old camels are particularly susceptible.

Geographic distribution: Sarcoptic mange occurs wherever camels are kept.

Symptoms: Skin reactions start on the head, base of the neck, mammary gland, prepuce and flank. The head is almost always affected since camels use their teeth to scratch affected areas. First clinical signs such as erythema, vesicles and intense pruritus occur 2–3 weeks after infection. About 2 weeks after the first signs, the affected skin has lost its hair and becomes reddened and moist. The lesions may become generalized after 20–30 days. Later the skin becomes dry and hard, with folds (hyperkeratosis) forming in the neck region, around joints and on the thighs. Itchiness is less pronounced in this stage. Intense itching, scratching and rubbing distract the affected animal from eating, and progressive weight loss and emaciation result. Decubitus sores develop rapidly and secondary infections, particularly with pyogenic bacteria may aggravate the condition.

Significance: Sarcoptic mange in camels is one of the most important diseases of camels, causing weight loss, emaciation and severe secondary skin infections. Direct transmission from camel to man is common and causes a condition termed pseudoscabies.

Diagnosis: Mange is easy to diagnose clinically. Intense itching, pruritus, depilation and encrusted plaques are characteristic for the mange. These signs must be differentiated from tick-associated skin reactions, eczema (rare in camels), depilation due to malnutrition and poxvirus infections in young camels. The chronic stage is characterized

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by extensive hyperkeratosis, showing thickened skin and folds around joints, hind limbs and neck. Laboratory diagnosis is not easy. Deep skin scrapings (until the skin starts to bleed) may reveal the mite. Pseudoscabies in man is transmitted during milking and is therefore seen in the interdigital space of the hands, the flexor surface of the wrists, the forearms, the elbows and the axillary folds. Camel riders show lesions between the thighs.

Therapy: The most commonly used acaricide is lindane (0.05%). However, most of the acaricides recommended for cattle mange (organophosphates and pyrethroids) may also be used for camels, except malathion (0.75%) which does not seem to be very effective. The acaricides are applied by brushing the affected regions and/or as sprays. Spray treatment must be applied thoroughly to all parts of the body and treatment must be repeated after 8–15 days. Once a herd is infected, continuous reinfection of both man and camels occurs and it is therefore essential to treat both. The recommended treatment for man is hexachlorcyclohexane. Ivermectin (0.2 mg/kg, sc., twice at 2-week-intervals) proved to be highly effective against camel mange. It is also effective against the most important gastrointestinal nematodes (*Haemonchus* spp.).

Prophylaxis: The entire herd should be treated at the same time. Freshly introduced animals must be treated. Attention must be paid to the indirect transmission of mange via harness and other equipment. These should also be treated with an acaricide. Dietary supplements would help to improve the general condition and thus reduce the susceptibility to mange.

(Figures 220, 524, 525, 526)

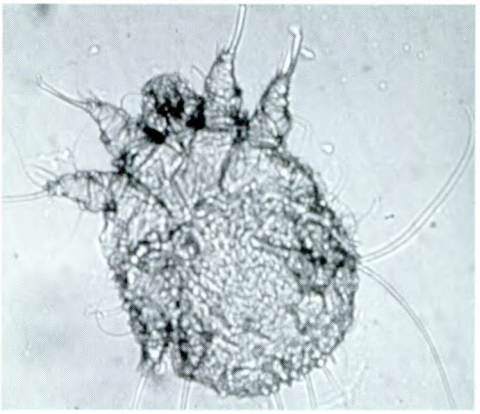

Fig. 524 *Sarcoptes scabiei* var. *cameli* (syn. *camelensis*); adult mite (female up to 0.6 mm long, male up to 0.3 mm long) with long unjointed pedicels [13]

Fig. 525 Dromedary with generalized sarcoptic mange [46]

Fig. 526 Hairless, rough skin due to sarcoptic mange [46]

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Chorioptes spp. Chorioptic mite, foot mange, leg mite

Remarks: Chorioptic mange occurs mainly on the legs, base of tail and upper rear surface of the udder. Chorioptic mange has been found repeatedly in dromedaries kept in zoos. For diagnosis, therapy and prophylaxis ☞ above *Sarcoptes scabies* and CATTLE, ■ 5.1.

(Figure 527)

Fig. 527 Hyperkeratosis, dry and hard skin due to Chorioptic mange [8]

- **Insecta found on the skin**

– **Lice**

ANOPLURA
Blood sucking lice of camels

Microthoracius cameli (syn. *Haematopinus cameli*)

Remarks: This species is closely related to members of the genus *Linognathus* (☞ CATTLE, ■ 5.1). It is the only blood-sucking louse of camels. Animals in poor conditions carry high numbers of lice. Louse worry is characterized by licking, scratching and rubbing. Heavy infestations may cause anaemia, particularly in young animals. The coat becomes rough and secondary bacterial infections may occur. Most of the insecticides recommended for cattle may be used for louse infestations in camels.

(Figure 528)

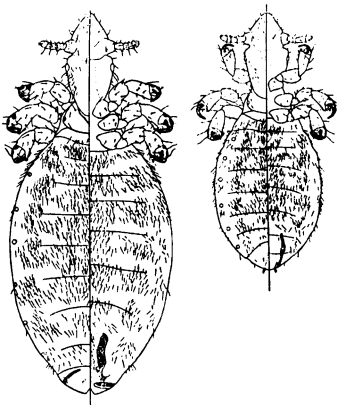

Fig. 528 *Microthoracius cameli*; female (4 mm long) dorsal and ventral view (left); male (2 mm long) dorsal and ventral view (right) [9]

– **Fleas**
No fleas have been found on the body surface of dromedaries

– **Dipterida**

Many of the dipteran flies which attack cattle may also affect camels, when they enter the habitat of these flies. Fly worry is particularly a problem around watering places, in oases and along well aerated streams. For symptoms, therapy and prophylaxis ☞ CATTLE, ■ 5.1.

CALLIPHORIDAE
The blowflies and their allies

They are highly important in many domestic animal species and man. The adults are free-living and the larvae are parasitic maggots which develop in the tissue of their host, causing a condition called myiasis. The larvae may be laid into pre-existing wounds. Myiasis is often a secondary skin problem. Many of the species found on cattle and small ruminants may occasionally occur in camels (☞ CATTLE, ■ 5.1 and SHEEP AND GOATS, ■ 5).

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Fig. 529 Saddle wounds are often invaded by blowflies [15]

In camels larvae of the following flesh flies often cause myiasis.
(Figure 529)

Wohlfahrtia magnifica
The Old World flesh fly
(☞ Cattle, ■ 5.1)

Wohlfahrtia nuba

Remarks: This larviparous flesh fly occurs in man and dromedaries in Sudan and Ethiopia, Senegal and probably in many other countries of North Africa and the Middle East. Primary skin wounds are often infested by larvae of this fly. This myiasis (mainly vaginal myiasis) is currently a severe problem in two-humped camels in Mongolia.

Sarcophaga dux

Remarks: This fly lays its larvae in decomposing flesh, wounds and ulcers. It has been found in skin lesions of camels in India.

HIPPOBOSCIDAE
The louse flies

Hippobosca camelina Camel louse fly

Remarks: This blood sucking louse fly occurs on camel and horse and occasionally cattle wherever camels are present, mainly in the northern part of eastern Africa and the Mediterranean region. These flies are a source of great irritation and occur in clusters in the perineal region, between the hindlegs to the pubic region, but may also bite on other parts of the body. Infested animals scratch and rub and skin-trauma is often seen as a consequence of heavy infestations. ☞ Cattle, ■ 5.1; Therapy and Prophylaxis of Ectoparasites, p. 141.

5.2 Eyes

***Thelazia* spp.** (*Thelazia rhodesi* and *Thelazia leesei*) Eye worms

Remarks: *T. rhodesi* (☞ Cattle, ■ 5.2) may occasionally occur in camel. *T. leesei* is specific for camels and transmitted by *Musca lucidala*. This spirurid nematode develops in the conjunctival sacs of camels in Africa and Asia. One or both eyes may be affected often without clinical signs. Heavy infections may cause irritation and keratitis with marked discharges. Eggs or first-stage larvae may be found in the lacrimal secretions. Therapy (☞ Cattle, ■ 5.2) is only indicated when intense lacrimal secretion and secondary infections occur. This condition is rather rare.

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Parasites of Swine

CONTENTS

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PROTOZOA

Eimeria spp. and *Isospora suis*
Coccidiosis in pigs

Location: Jejunum and ileum

Hosts: Pig

Species description: Coccidiosis in swine is caused by eight species of the genus *Eimeria* and one of *Isospora*. Coccidiosis is caused by intracellular parasites that inhabit the intestinal tract, particularly the small intestine. The life cycles of coccidia are complex and include both sexual (gametogony) and asexual (schizogony) cycles within the host. Some development (sporogony) also takes place outside the host. The host cells are destroyed by schizogony, gametogony and the release of oocysts. Each oocyst that is ingested has the potential of destroying thousands or even millions of intestinal epithelial cells since each schizont contains numerous merozoites and several generations of schizonts may be produced. Multiplication of coccidia depends on a continuous ingestion of oocysts. The severity of coccidiosis therefore depends on the number of oocysts ingested and the pathogenicity of the particular species. Some species of coccidia are more pathogenic (*I. suis, E. debliecki, E. scabra, E. polita, E. spinosa*) than others (*E. neodebliecki, E. perminuta, E. porci, E. suis*). Usually mixed coccidia infections occur. Unsporulated oocysts are passed in the faeces but infection is only transmitted if the oocysts are sporulated. Coccidiosis (especially caused by *I. suis*) as a disease entity occurs mainly in newborn piglets (3–21 days of age); however, older pigs may act as carriers. Morbidity is usually high (50–75%) but the mortality varies markedly due to differences in the pathogenicity of a particular species, or in the environment (poor hygiene, lack of milk supply, etc.) and the presence of other coexisting diseases (colibacillosis and rotavirus infections, transmissible gastroenteritis, etc.).

Geographic distribution: World-wide

Symptoms: Diarrhoea which persists for 4–6 days, in neonatal piglets, is the predominant clinical sign. The faeces are generally yellow-white in colour but rarely bloody. In cases where the diarrhoea stops itself, stunting and emaciation are the only clinical signs.

Significance: Several coccidia species (especially caused by *Isospora suis*) may be important among the pathogens causing diarrhoea in neonatal piglets.

Diagnosis: Oocysts may not be shed during the diarrhoeal phase, so faecal flotation examination is of little value for diagnosing swine coccidiosis during the acute phase. The presence of fibrino-necrotic membranes (macroscopic), shortened villi and necrosis of epithelial cells (microscopic) in the jejunum and ileum are found at necropsy. The endogenous forms of coccidia (merozoites, small schizonts) are revealed in the villous epithelial cells by histopathological techniques or stained impression smears of the jejunum and ileum. The characteristic oocysts are most easily detected in the faeces of recovered litter-mates but generally not during the diarrhoeic period. Postmortem examination of the intestine of previously ill pigs for endogenous forms is the preferred diagnostic method.

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Therapy: Coccidia infections are often self-limiting and end spontaneously within a few weeks unless reinfection occurs. Medication usually does not cure but slows or inhibits the development of stages resulting from reinfection and thus shortens the length of illness, reduces discharge of oocysts and lessens the incidence of secondary infections. Reduction of the oocyst excretion of clinically ill piglets can be achieved with sulfaguanidine (0.22 mg/kg, po.), sulfamethazine or sulfamerazine. Amprolium (25–65 mg/kg, po.) is beneficial for prevention but treatment of scouring piglets has minimal effect. Supportive treatment with antibiotics and rehydration may also be very important.

Prophylaxis: Good sanitation (removal of faeces, disinfection of farrowing facilities) is of utmost importance if the coccidia life cyle should be broken, since the previous litters may be the source of infection. Amprolium (8 days before until 8 days after birth) prevented cocciodiosis in piglets. Salinomycin sodium (60 ppm for body weight < 50 kg, 25 ppm for > 50 kg) and halofuginone hydrobromide (3 ppm) reduced *Eimeria* spp. and *I. suis* oocyst excretion.

(Figures 530, 531, 532, 533, Table 19)

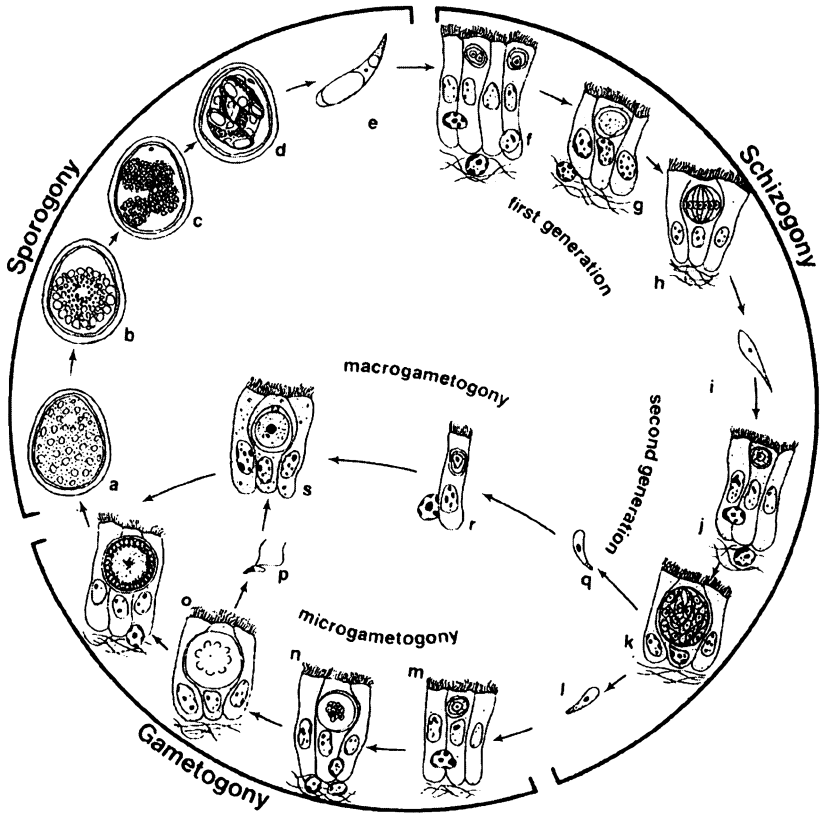

Fig. 530 Life cycle of *Eimeria debliecki*; oocyst sporulation (a–d), isolated sporozoite (e), schizogony with first and second generation of merozoites (f–k), second generation of merozoites (l and q), formation of microgametes (m–o), microgamete (p) and formation of macrogametes (r–s). The cycle ends with the formation of a zygote [14]

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Table 19 Coccidia of pigs [1]

Species	Average dimensions (microns)	Shape	Morphology
E. debliecki	25 × 17	Ellipsoidal	Wall smooth, colourless, asymmetrical sporocysts
E. neodebliecki	21 × 16	Ellipsoideal	Wall smooth, colourless
E. perminuta	13 × 12	Spherical, subsperical	Wall rough, yellow
E. polita	27 × 21	Ellipsoideal	Wall rough, yellow to colourless
E. porci	22 × 16	Ovoid	Wall smooth, colourless, broad sporocysts
E. scabra	32 × 23	Ovoid	Wall rough with radial striations, yellow to brown
E. spinosa	20 × 13	Ovoid	Wall rough, spined, brown
E. suis	17 × 13	Ellipsoidal, subsperical	Wall smooth, colourless
Isospora suis	20 × 17	Spherical, subspherical	Wall smooth, colourless, thin

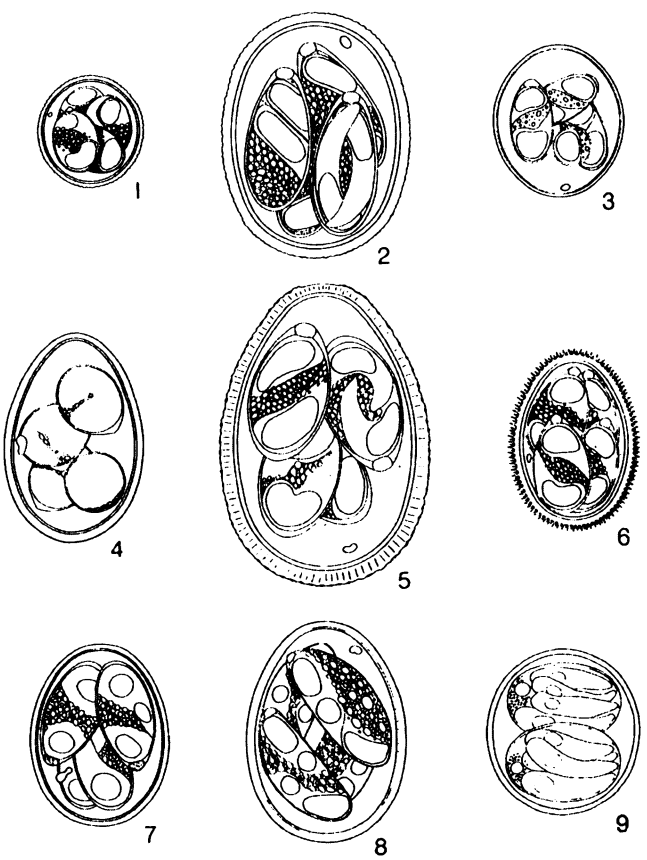

Fig. 531 Sporulated oocysts of coccidia from the pig [14]:

1 *Eimeria perminuta* (13 × 12 µm)
2 *Eimeria polita* (27 × 21 µm)
3 *Eimeria suis* (17 × 13 µm)
4 *Eimeria porci* (22 × 16 µm)
5 *Eimeria scabra* (32 × 23 µm)
6 *Eimeria spinosa* (20 × 13)
7 *Eimeria neodebliecki* (21 × 16 µm)
8 *Eimeria debliecki* (25 × 17 µm)
9 *Isospora suis* (20 × 17 µm)

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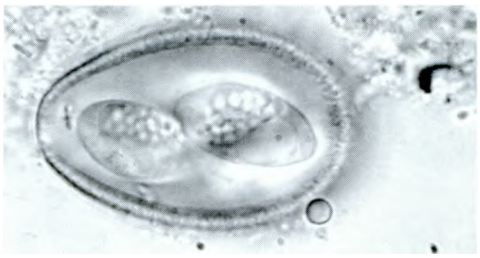

Fig. 532 Oocyst of *Eimeria scabra*; sporulated (32 × 23 µm) [4]

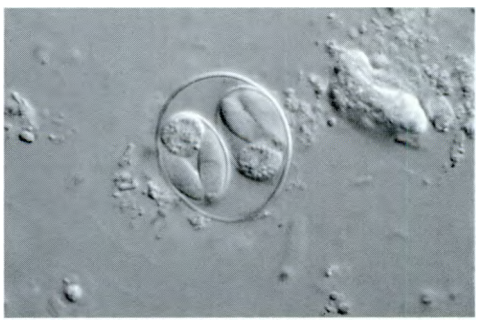

Fig. 533 Oocyst of *Isospora suis*; sporulated (20 × 17 µm) [4]

Cryptosporidium parvum

Remarks: Little is known about *C. parvum* in piglets. It is thought to be prevalent but usually of low morbidity and mortality. Infection may result in non-haemorrhagic diarrhoea (☞ CATTLE, 1).

Balantidium coli (syn. *B. suis*)

Location: Caecum, colon

Hosts: Pig, occasionally in peccary, man, dog and rat

Species description: The motile trophozoites are ovoid and variable in size, 30–150 × 25–120 µm. Cilia cover the entire body. The cyst form is spherical and measures 45–65 µm in diameter. *B. coli* reproduces in the lumen of the large intestine by binary fission where it is a commensal. Under normal conditions *B. coli* cannot penetrate intact intestinal mucosa. If another pathogen initiates a lesion, then *B. coli* may act as a secondary invader.

Geographic distribution: World-wide

Symptoms: Enteritis, diarrhoea, dysentery, colitis, anaemia, retarded growth and emaciation in young piglets

Significance: *B. coli* almost exclusively plays a role as a secondary invader in the large intestine of pigets. *B. coli* is much more pathogenic to man than to swine. It causes severe colitis, with ulcers of the mucosa and watery diarrhoea.

Diagnosis: *B. coli* can be easily recognized by microscopic examination of intestinal contents or by histologic examination of intestinal lesions.

Therapy: No treatment is necessary in pigs. Treatment of the primary infection is indicated.

Prophylaxis: Prevention of *B. coli* infections of swine is not indicated since it is a permanent inhabitant. General health care and good hygiene prevent primary infections which are necessary for *B. coli* to become pathogenic. Control of the transmission from pig to man depends on good sanitation.

(Figures 534, 535, 536)

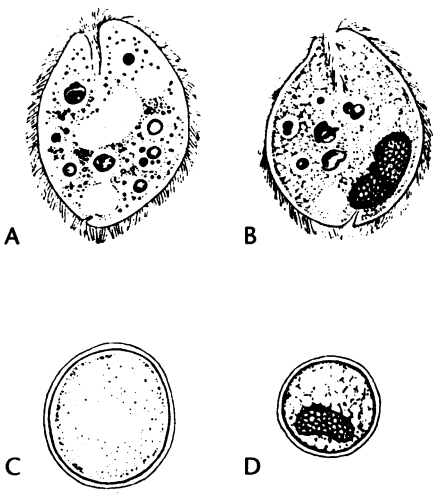

Fig. 534 *Balantidium coli* (schematic); living trophozoite (A), stained trophozoite (B), fresh cyst (C) and stained cyst (D) [14]

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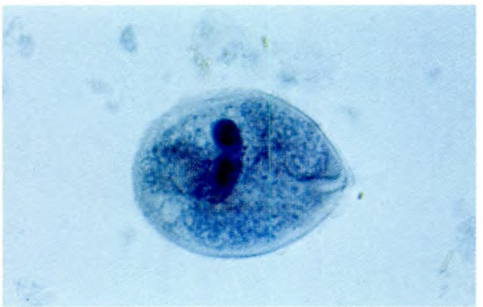

Fig. 535 *Balantidium coli*; trophozoite stained (50–150 µm) [4]

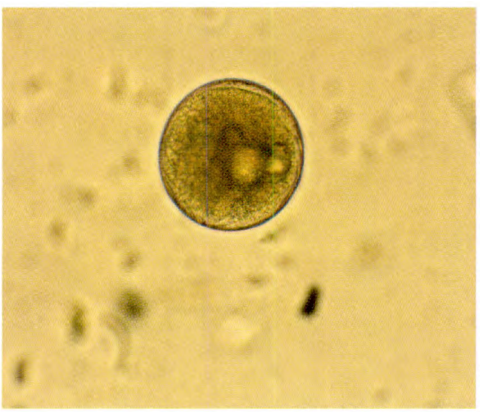

Fig. 536 *Balantidium coli*; cyst (50–100 µm in diameter)

Tritrichomonas suis

Remarks: This is the largest of the pig trichomonads and occurs in the stomach, nasal passages, caecum and small intestine and occasionally in the vagina; non-pathogenic.

Trichomonas (syn. *Trichomitus*) *rotunda*

Remarks: It occurs in the caecum and colon of swine; non-pathogenic.

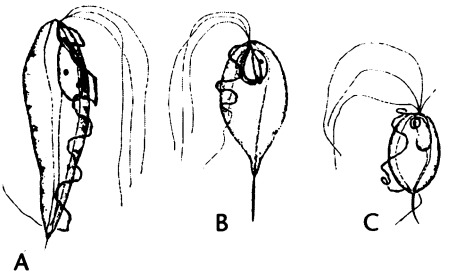

Fig. 537 Trichomonads of swine; (A) *Tritrichomonas suis* (11 × 3 µm), (B) *T. rotunda* (9 × 6 µm) and (C) *Tetratrichomonas buttreyi* (6 × 3 µm) [14]

Tetratrichomonas buttreyi

Remarks: It is the smallest of the pig trichomonads. It occurs in the caecum and colon; non-pathogenic.
(Figure 537)

Giardia lamblia

Remarks: It occurs in the duodenum, jejunum and ileum of man, primates (highly pathogenic) and many other mammals including swine where it is non-pathogenic (☞ SHEEP AND GOATS, ☜ 1).

Entamoeba suis (syn. *E. polecki*)

Remarks: It occurs in the caecum and colon of swine and is generally non-pathogenic. Occasionally enteritis and diarrhoea may be found but the pathogenic role of *E. suis* is in debate.

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HELMINTHS

(Figure 538)

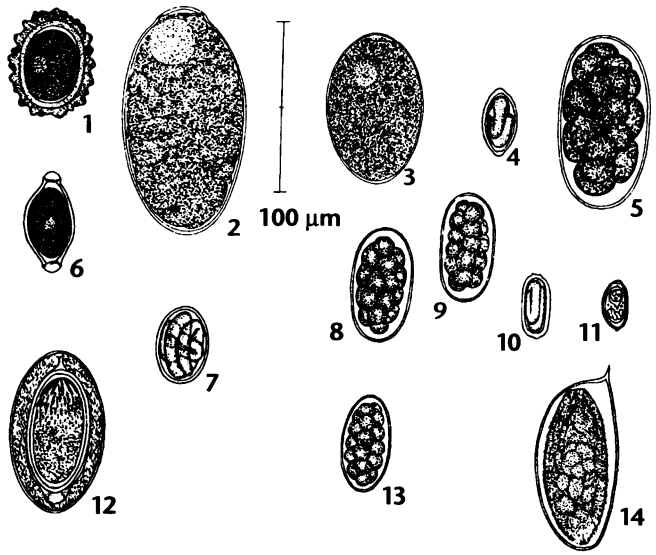

Fig. 538 Eggs of helminths found in swine [3]

1 *Ascaris suum*
2 *Fasciola hepatica*
3 *Paragonimus* sp.
4 *Ascarops strongylina*
5 *Stephanurus dentatus*
6 *Trichuris suis*
7 *Metastrongylus apri*
8 *Oesophagostomum dentatum*
9 *Hyostrongylus rubidus*
10 *Physocephalus sexalatus*
11 *Brachylaemus* sp.
12 *Macracanthorhynchus hirudinaceus*
13 *Globocephalus* sp.
14 *Schistosoma suis*

- **Trematoda eggs found in the faeces and adult trematodes living in the gastrointestinal tract**

Gastrodiscus aegyptiacus Intestinal fluke

Remarks: It occurs in the small and large intestine of equines, pig and warthog in Africa and India. If present in high numbers the parasite can significantly affect pigs (☞ HORSES AND DONKEYS, 1).

(Figure 539)

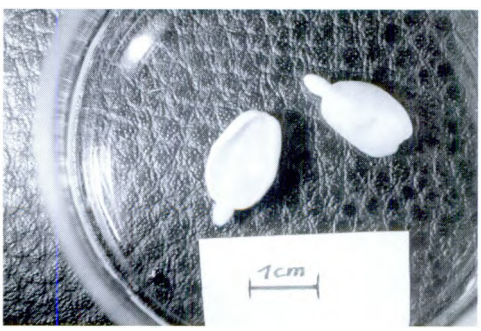

Fig. 539 *Gastrodiscus aegyptiacus*; ventral view (right) and dorsal view (left)

Gastrodiscoides hominis

Remarks: This is a common trematode found in the caecum and colon of pigs in South and East Asian countries.

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Postharmostomum suis

Remarks: It has been reported from Tunisia where it was found in the small intestine of pigs. Intermediate hosts are land snails of the genus *Xerophila*. It is not very pathogenic (☞ POULTRY, 1).

- **Eggs of the following trematodes may occasionally be found in the faeces of pigs**

Schistosoma bovis, S. mattheei, Dicrocoelium spp. and *Fasciola* spp.
(☞ SWINE, ■ 4.2 and CATTLE, 1)

Eurytrema pancreaticum
(☞ SWINE, ■ 4.5 and CATTLE, 1)

- **Nematoda eggs found in the faeces, adult nematodes living in the gastrointestinal tract and first-stage larvae of lungworms (*Metastrongylus* spp.)**

Gongylonema pulchrum Gullet worm

Remarks: *G. pulchrum* are occasionally found at necropsy in the oesophageal mucosa of swine but they are of little or no clinical significance (☞ CATTLE, 1).

Hyostrongylus rubidus Red stomach worm

Location: Stomach
Hosts: Pig
Species description: *H. rubidus* belongs to the trichostrongylids, is 4–10 mm long, bright red (blood sucker) and has a direct nematode life cycle. *H. rubidus* is found under a heavy catarrhal exudate and produces lesions similar to those of *Ostertagia* spp. in ruminants, except that hyperaemia of the mucosa is more common. Inhibition of larvae during periods of adverse environmental conditions (winter, dry season) occurs. In sows these inhibited larvae resume their development prior to parturition with the result that the environment of the piglets is contaminated.
Geographic distribution: World-wide
Symptoms: Variable appetite, anaemia, gastritis, diarrhoea associated with weight loss.
Significance: Moderate by contributing to mixed worm infections, resulting in reduced feed utilization.
Diagnosis: Demonstration of strongyle-type eggs in the faeces which resemble those of other strongyle worms (*Oesophagostomum, Globocephalus, Trichostrongylus*). Faecal culture is required to differentiate the different eggs. Mucosal scrapings for microscopical examination is essential for the detection of immature *Hyostrongylus* infection.
Remarks: Apart from the thin stomach worm, there are also two thick nematode species found in the stomach: *Ascarops strongylina* and *Physocephalus sexalatus* which belong to the Spiruridae and are > 12 mm long, much stouter and have coprophageous beetles as intermediate hosts. The eggs are thick-shelled and contain a larva when laid and measure 35–40 × 15–20 µm. At necropsy the adult *Physocephalus* and *Ascarops* are readily seen, whereas *H. rubidus* is more difficult to recognize. The stomach worms are more common in grazing pigs.
Therapy: Most modern benzimidazoles and probenzimidazoles and ivermectin (300 µg/kg, sc.) are highly effective against adult and immature stages (including hypobiotic larvae) of the stomach worms. Pyrantel (12.5 mg/kg, po.) and levamisole (5 mg/kg, im. or sc.) are effective against the adult stomach worms (Table 20).
Prophylaxis: Frequent removal of faeces and provision of dry quarters effectively reduce the infection risk.
(Figures 540, 541, 542, 543, 544, 545, 546)

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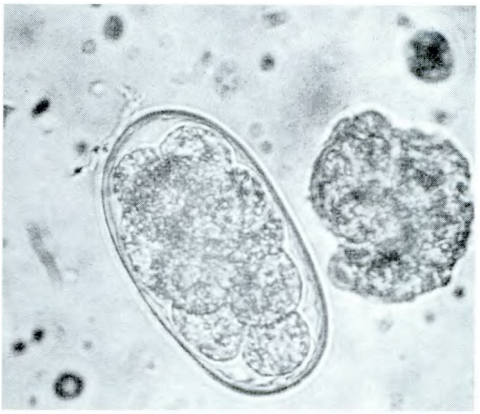

Fig. 540 Egg of *Hyostrongylus rubidus* (69–85 × 39–45 µm; typical strongyle-type egg) [11]

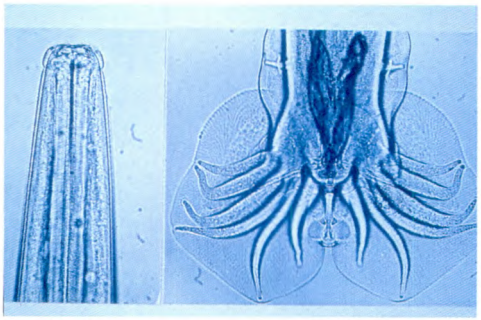

Fig. 541 *Hyostrongylus rubidus*; anterior end (left) and male bursa (right); males are 4–7 mm long, females are 5–10 mm long [4]

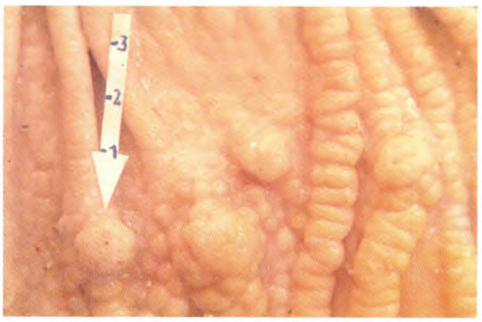

Fig. 542 Nodule formation in the gastric mucosa caused by *Hyostrongylus rubidus* [4]

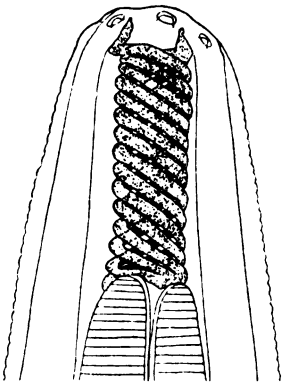

Fig. 543 *Ascarops strongylina*; anterior end; males are 10–15 mm long, females are 16–22 mm long [3]

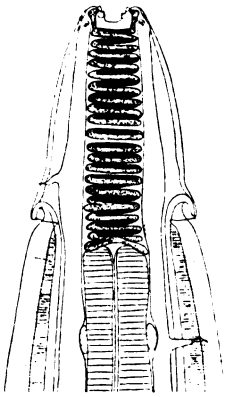

Fig. 544 *Physocephalus sexalatus*; anterior end; males are 6–13 mm long, females are 13–22.5 mm long [3]

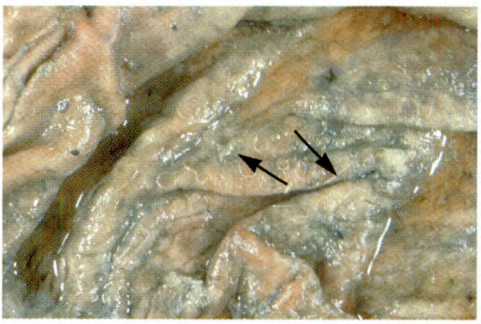

Fig. 545 *Physocephalus sexalatus* on the gastric mucosa [4]

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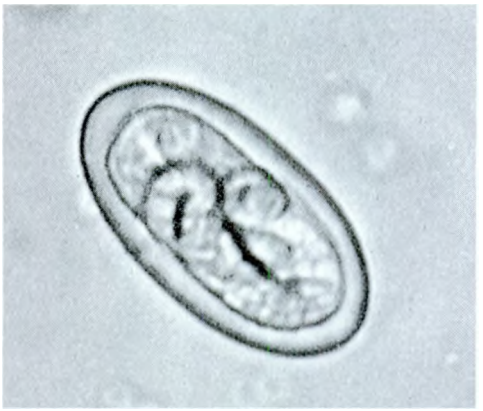

Fig. 546 Egg of *Physocephalus sexalatus* (34–39 × 15–17 µm) [11]

Table 20 Nematodicidal drugs for swine

Compound	Single dosing[1]	Multiple dosing[1]
Flubendazole	5.0	5–10 days x 30 ppm
Mebendazole	–	5–10 days x 30 ppm
Parbendazole	30, po.	7–10 days x 3000 ppm
Fenbendazole	5.0, po.	6–15 days
Febantel	5.0, po.	5 days x 0.5–0.7 mg/kg
Netobimin	7.5, po.	–
Thiabendazole	500, po.	5–10 days x 500 mg/kg
Oxibendazole	15, po.	10 days x 1g/kg/day
Oxfendazole	4.5, po.	–
Dichlorvos	30–40, po.	–
Levamisol	5–7.5, po.	–
Piperazine	250–312, po.	–
Pyrantel tartrate	12.5, po.	–
Thiabendazole	50–100, po.	–

[1]mg/kg body weight; po = orally

Gnathostoma hispidum

Location: Stomach

Hosts: Pig, warthog and man (exceptionally)

Species description: Males are 15–25 mm and females 22–45 mm long. The whole body is covered with spines. The eggs measure 73 × 40 µm. Eggs develop in water to second-stage larvae before hatching. Larvae develop to third-stage larvae when ingested by the intermediate hosts which are members of *Cyclops* spp. and related Crustacea. Infection of pigs by ingesting infected *Cyclops* or paratenic hosts (e.g. fish, birds, small mammals). Migration of larvae through the liver. Adults are embedded deeply in the gastric mucosa, producing cavities which contain reddish fluid and are surrounded by inflamed areas.

Geographic distribution: Europe, Asia, Africa (Zaire)

Symptoms: Inappetence, gastritis, weight loss in heavy infections. Mild infections are inapparent.

Significance: Severe infections cause gastritis.

Diagnosis: Demonstration of eggs in the faeces

Therapy: ☞ Therapy of *Hyostrongylus rubidus*

Prophylaxis: Water bodies containing the intermediate hosts should be avoided as a drinking place for pigs.

(Figures 547, 548)

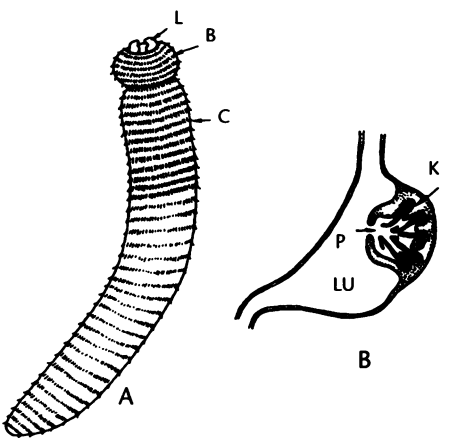

Fig. 547 *Gnathostoma hispidum*; adult parasite (males are up to 25 mm long, females are up to 45 mm long) covered with cuticular spines (A) and adults embedded in cyst-like cavities (B); L = lips, B = bulbus, C = cutucular spines, K = cyst wall, P = porus and LU = gastric lumen [33]

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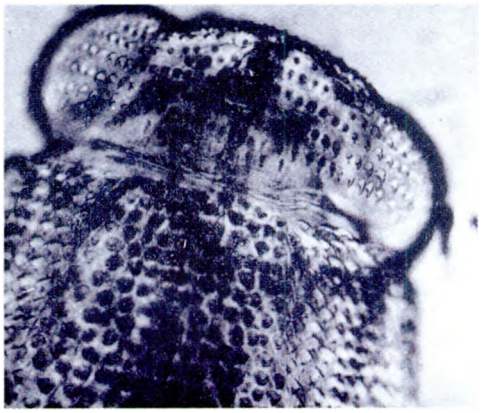

Fig. 548 *Gnathostoma hispidum*; anterior end with cuticular spines [49]

Trichostrongylus axei Stomach hair worm

Remarks: *T. axei* may occasionally be found in the stomach of pigs. It is of minor importance in pigs (☞ CATTLE, ☞ 1).

Ascaris suum Large roundworm of swine

Location: Small intestine

Hosts: Pig

Species description: *A. suum* is a large, white worm, 15–40 cm in length. The life cycle is direct. Infective larvae develop within 10–15 days and remain in the egg. After ingestion, the larvae are released in the intestine and burrow into the wall of the gut. The majority of the larvae pass to the liver via the hepatoportal bloodstream. Destruction of hepatic tissue and haemorrhage occurs, signs of which appear as "milk-spots" after the healing of these tracks. Larvae are carried in the blood to the lungs where they penetrate the blood vessels to enter alveoli to proceed to the pharynx to be finally swallowed. Further development to the adult stage occurs in the small intestine. Adult females are known to produce an enormous number of eggs (up to 200,000/day) which are very resistant to adverse climatic conditions and may be infective for years in the environment.

Geographic distribution: World-wide, widespread throughout the tropics

Symptoms: Damage caused by migrating larvae is of principal importance. Migration through the liver causes the white scar-like "milk-spots" which are the major reason for swine liver condemnation. Massive migration through the lungs may cause pneumonia followed by bacterial infections. Death may occur following heavy infections. Repeated infections, accompanied by lung haemorrhage and oedema and emphysema may result in a chronic, asthma-like condition characterized by unthriftiness. Adult ascarids in moderate numbers are relatively non-pathogenic but compete with their host for food and can therefore markedly reduce feed efficiency. High numbers of adult worms can sometimes "ball up" causing intestinal obstruction, perforation and peritonitis. Obstruction of the bile ducts and icterus may also occur.

Significance: *A. suum* is the most important swine endoparasite, causing heavy economic losses due to weight loss, mortality and condemnation of livers. *A. suum* infections can exacerbate swine influenza and endemic pneumonia and secondary bacterial infections.

Diagnosis: Demonstration of thick shelled eggs (60 × 45 µm) which are uneven in outline during the patent period. Young pigs often show signs (especially respiratory) during the prepatent period. A presumptive diagnosis can be made based on the history and signs and the demonstration of migrating larvae, especially in the lungs and liver at necropsy. Adult worms are found at necropsy and occasionally within the faeces of pigs.

Therapy: Piperazine preparations are cheap but effective only against adult worms. The benzimidazoles and probenzimidazoles, dichlorvos, ivermectin, levamisole and pyrantel are effective against immature and adult stages and have a broader spectrum of activity than piperazine. Pyrantel and ivermectin (300 µg/kg, sc.) are effective against migratory larvae.

Prophylaxis: Since the destruction of migrating

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immature larvae is difficult, control measures should prevent or reduce the ingestion of eggs by pigs. Apart from a good basic hygiene in pig quarters control is based on anthelmintics. In intensive pig rearing systems a simple anthelmintic program is as follows: treat sows 10–14 days before breeding and again before farrowing; treat weaners before entering clean pens; treat boars at 6-month intervals. In traditional pig rearing systems where pigs are kept freely around the compounds no effective prophylaxis is possible. Regular treatment is required.

(Figures 549, 550, 551, 552, Table 20)

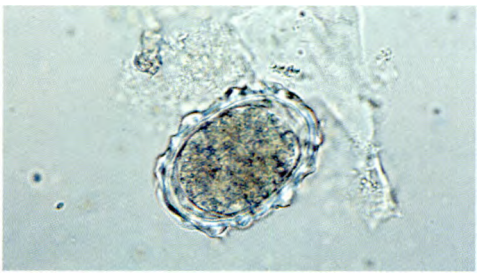

Fig. 549 Egg of *Ascaris suum* (50–70 × 40–60 µm)

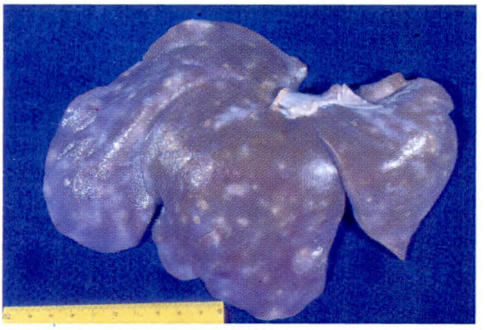

Fig. 550 "Milkspots" in the liver of a pig

Fig. 551 *Ascaris suum*; adult worms found in the small intestine

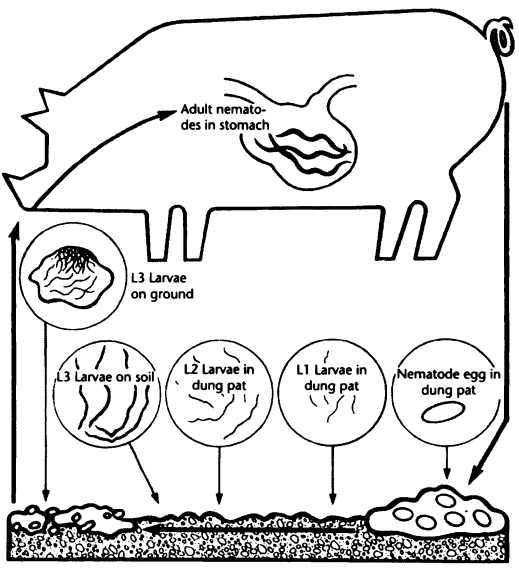

Fig. 552 Life cycle of a roundworm. Adult *Ascaris suum* worms are found in the small intestine. The larvae migrate from the intestine via liver, heart to the lungs [50]

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Globocephalus urosubulatus and other *Globocephalus* spp. Pig hookworms

Location: Small intestine
Hosts: Domestic pig and wild boar
Species description: Very little is known about the life cycle which is probably direct. Adults are 6–8 mm long and have a typical hookworm-like buccal capsule to suck blood. Eggs are passed in the faeces and develop to infective third-stage larvae within 8–12 days. Infection occurs by ingesting third-stage larvae or by transcutaneous penetration. Migration of larvae through heart, lungs, trachea, oesophagus, intestines.
Geographic distribution: World-wide
Symptoms: Anaemia, hypoproteinaemia, progressive weight loss and emaciation may occur in heavy infections.
Significance: *Globocephalus* spp. infections are generally of little significance although, if the parasite occurs within a particular population it may cause severe losses. Piglets are generally most severely affected.
Diagnosis: Strongyle-type eggs occur in the faeces. Adult worms are found at necropsy.
Therapy: ☞ *Hyostrongylus rubidus*
Prophylaxis: ☞ *Hyostrongylus rubidus*
(Figures 553, 554, 555)

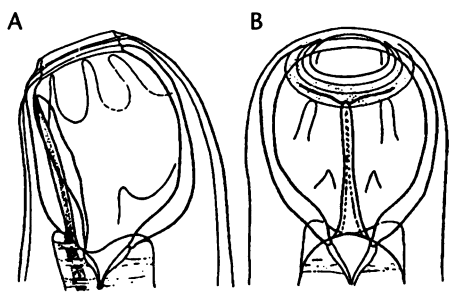

Fig. 553 *Globocephalus urosubulatus*; lateral view of anterior end (A) and dorsal view of anterior end (B) [34]

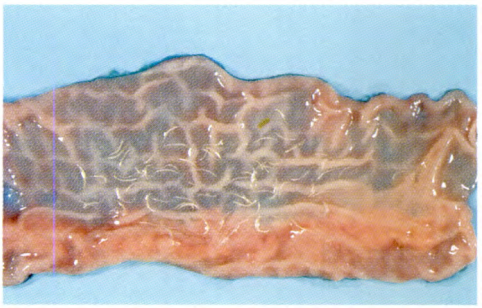

Fig. 554 *Globocephalus urosubulatus* in the small intestine of a pig

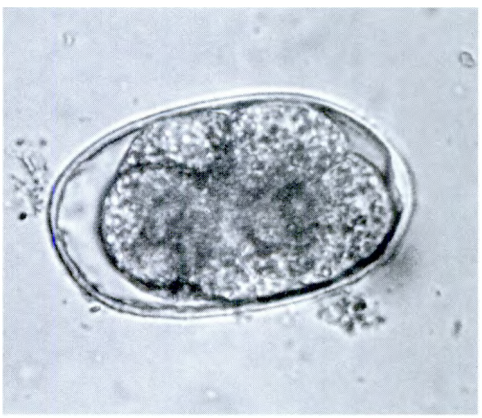

Fig. 555 Egg of *Globocephalus urosubulatus* (50–56 × 26–35 µm) [11]

Strongyloides ransomi Intestinal threadworm

Location: Small intestine
Hosts: Pig
Species description: The life cycle is similar to that of *S. papillosus* (☞ CATTLE, 1). Transmission of larvae in the colostrum is the most common route of infection of nursing piglets. The adult worms which are exclusively female, burrow into the intestinal wall and cause irritation and inflammation. Infection of the host can occur by both skin penetration and ingestion of infective larvae. Prepatent period is 6–9 days. Adult breeding stock may be infected with dormant larvae in their subcutaneous fat. Pregnancy and farrowing appear

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Fig. 556 Eggs of *Strongyloides ransomi* (40–55 × 20–35 µm) contain a first-stage larva

to stimulate the re-emergence of these larvae which then may infect the piglets via the colostrum. In only one week after birth piglets may pass eggs in their faeces that can develop within 24 hours to infective larvae. Consequently a quick rise in worm burdens is typical for threadworm infections.

Geographic distribution: World-wide, particularly common in warm climates

Symptoms: In light infections the animals show no signs. In heavy infections bloody diarrhoea, anaemia, emaciation and sudden death (especially in piglets) may occur. During the migratory phase of infection coughing, muscle soreness, abdominal pain and vomitus can be observed.

Significance: Highly significant for neonatal piglets

Diagnosis: Demonstration of thin-shelled, embryonated eggs in the faeces or the adults in scrapings from the intestinal mucosa is diagnostic. Eggs must be differentiated from the larger *Metastrongylus* eggs. At necropsy immature worms may be recovered from minced tissues placed in a Baermann isolation funnel.

Therapy: Infections in suckling piglets can only be reduced by treating the sows before farrowing. The benzimidazoles, febantel and levamisole are effective against intestinal infections. Ivermectin (300 µg/kg, sc.) is effective against adults and if administered 1–2 weeks before farrowing, controls the milk borne infection.

Prophylaxis: Cleaning the pig's living quarters can reduce free-living larvae and thus the infection risk. Wherever this is not feasible, anthelmintic treatment of sows prior to parturition and close observation of neonatal piglets (incl. treatment) is required.

(Figure 556)

Trichinella spp. (*T. spiralis, T. britovi, T. nativa, T. nelsoni* and *T. pseudospiralis*) Trichina worm

Remarks: Adult worms occur in the small intestine. Encysted larvae are found in the diaphragm and masseter, lingual and intercostal muscles of pig and many other mammals. These cysts are found by microscopical examination of infected muscles (☞ SWINE, ■ 4.1).

Trichuris suis Whipworm

Location: Caecum and colon

Hosts: Pig and wild boar

Species description: *T. suis* is 5–8 cm long and has a typical whip-like tail. Direct life cycle. Infection occurs by ingesting infective eggs (containing first-stage larvae). *Trichuris* eggs may remain viable for several years. After being ingested *T. suis* larvae hatch and enter the intestinal wall and develop further to the second-stage larvae and finally proceed to the large intestine for final maturation. Prepatent period is 6 weeks.

Geographic distribution: World-wide

Symptoms: Heavy infections are accompanied by diarrhoea and unthriftiness. Chronic infections may cause anaemia.

Significance: *T. suis* causes economic losses by producing poor growth and reduced feed efficiency wherever pigs are reared.

Diagnosis: The typical, double operculated eggs are diagnostic. Adult worms are easily found at necropsy.

Therapy: Dichlorvos, levamisole and some benzimidazoles generally at increased dosage rates are effective. Ivermectin (300 µg/kg, sc.) resulted in a 80% reduction of the adult worm population (Table 21).

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Prophylaxis: Removal of the faeces from the quarters reduces the infection risk drastically. Eradication is difficult because *Trichuris* eggs may be infective for 6 or more years on soil.

(Figures 94, 557, Table 21)

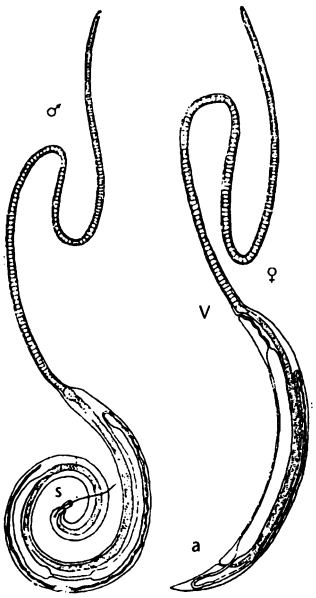

Fig. 557 *Trichuris suis* (4.5–5.5 mm long); male (left) and female (right); s = spicule, a = anus and v = vulva [9]

Table 21 Compounds to be used against *Trichuris suis* infections

Compound	Single dosing	Multiple dosing
Flubendazole	5.0, po.	5-10 days x 30 ppm
Mebendazole	–	10 days x 30 ppm
Oxibendazole	15.0, po.	10 days x 1 g/100 kg/day
Fenbendazole	30.0, po.	6-15 days x 16-20 ppm
Febantel	20.0, po.	
Levamisole	7.5, sc.	

po. = orally, sc. = subcutaneous

Oesophagostomum dentatum and other *Oesophagostomum* spp. Nodular worms

Location: Adult worms are found in the lumen of the large intestine. Larvae occur in the mucosa of the small and large intestine.

Hosts: Pig and wild boar

Species description: Adults are 8–14 mm long, white and have a shallow buccal capsule. The life-cycle is direct and infection results from ingestion of infective third-stage larvae. After ingestion the larvae moult and burrow into the intestinal mucosa anywhere between the pylorus and the rectum. After 5–7 days the larvae moult to fourth-stage larvae within the nodule and emerge into the intestinal lumen, where they mature and start to excrete eggs about 40–50 days after infection. Sows may have a periparturient rise in *Oesophagostomum* egg output, which is an important source of infection in newborn piglets.

Geographic distribution: World-wide

Symptoms: Diarrhoea, weight loss and anorexia may be observed in heavy infection.

Significance: The nodular worm is highly prevalent and among the most significant nematodes in pigs. It causes extensive nodule formations associated with decreased feed utilization, disturbed water and electrolyte metabolism and progressive weight loss.

Diagnosis: Typical strongyle eggs (75 × 40 µm) are found in the faeces. These can be differentiated from *Hyostrongylus* by larval culture. Adult worms are found sometimes in great numbers at necropsy. Multiple nodules, seen from the serosa side, may be found at slaughter (☞ CATTLE, 1).

Therapy: The benzimidazoles, levamisole, dichlorvos, pyrantel tartrate and ivermectin are effective (☞ *Hyostrongylus rubidus*).

Prophylaxis: Anthelmintic treatment does not always affect the larvae within the nodules. Therefore, repeated treatments several weeks apart are necessary to reduce the nodular worm incidence within a population.

Remarks: *O. quadrispinulatum* is similar to *O. dentatum* but does not occur in Africa.

(Figures 558, 559, 560, Table 22)

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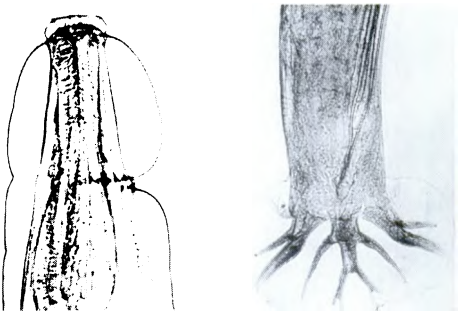

Fig. 558 *Oesophagostomum dentatum* (10–14 mm); anterior end (left) and posterior end of a male (right) [4]

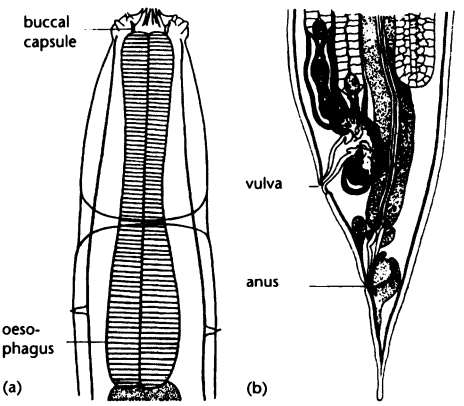

Fig. 559 *Oesophagostomum dentatum*; anterior end (a) and female tail end (b) [5]

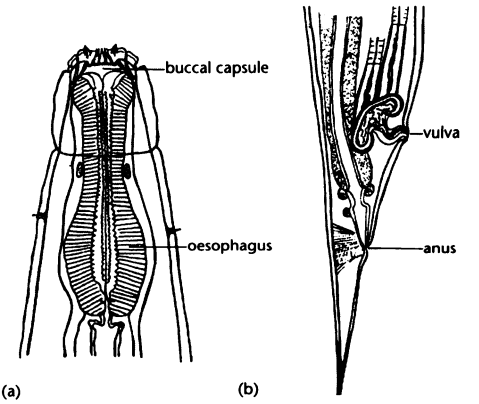

Fig. 560 *Oesophagostomum quadrispinulatum*; anterior end (10–15 mm) (a) and female tail end (b) [5]

Table 22 Comparison of the morphology of *Oesophagostomum dentatum* and *Oesophagostomum quadrispinulatum*

	Oesophagostomum quadrispinulatum	***Oesophagostomum dentatum***
Buccal capsule	Sides not parallel, diverging posteriorly	Sides papallel, not diverging
Oesophagus	Vase-shaped, swollen at anterior end	Club-shaped, not swollen at anterior end
Spicules	0.9–0.95 mm long	1.15–1.3 mm long
Female: posterior end	Long, tapering tail; distance between vulva and anus and tip of tail longer than that of *O. dentatum*	Relatively short tail; distance between vulva and anus and anus and tip of tail shorter than that of *O. quadrispinulatum*

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Metastrongylus spp. Lungworms

Remarks: Eggs of the lungworms occur in the faeces (☞ SWINE, ■ 4.3).
(Figures 561, 562)

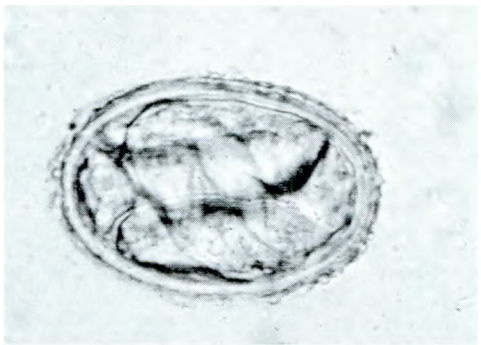

Fig. 561 Egg of *Metastrongylus apri* (51–63 × 33–42 µm) [11]

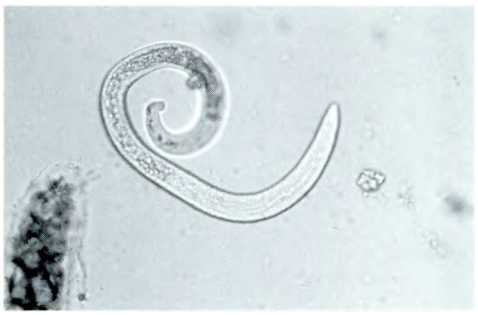

Fig. 562 First-stage larva of *Metastrongylus* spp.

ACANTHOCEPHALA

Macracanthorhynchus hirudinaceus
"Thorny headed worm"

Location: Small intestine
Hosts: Domestic and wild pig
Species description: *M. hirudinaceus* belongs to the Acanthocephala, a group of parasitic worms closely allied to the nematoda. Adults are 10–35 cm long, with reddish, transversely wrinkled bodies. The anterior end shows a spiny, retractable proboscis (rostellum) which anchors each worm to the intestinal wall. The eggs (67–110 × 40–65 µm) are ingested by the grubs of various beetles that serve as intermediate hosts.
Geographic distribution: World-wide, except western Europe (common in Madagascar).
Symptoms: Signs are not specific. Heavy burdens may cause diarrhoea, reduced weight gain and emaciation.
Significance: Mild infections are inapparent, heavy infections may cause irritation of the intestines, accompanied by weight loss. The site of the attachment often shows a necrotic center surrounded by a zone of inflammation. These lesions can usually be seen through the serosa. Perforation of the intestinal wall followed by peritonitis may occur.
Diagnosis: Eggs with four shells appear in the faeces. They do not reliably float in salt solutions and should therefore be looked for in the sediment. *M. hirudinaceus* is attached to the intestinal wall which is in contrast to *Ascaris suum* which lies freely within the small intestine.
Therapy: Levamisole (5–7.5 mg/kg, sc.) and fenbendazole (5 × 20 mg/kg, po.) at daily intervals are effective.
Prophylaxis: Control is only feasible if the use of contaminated hog lots can be avoided.
(Figures 563, 564, 565)

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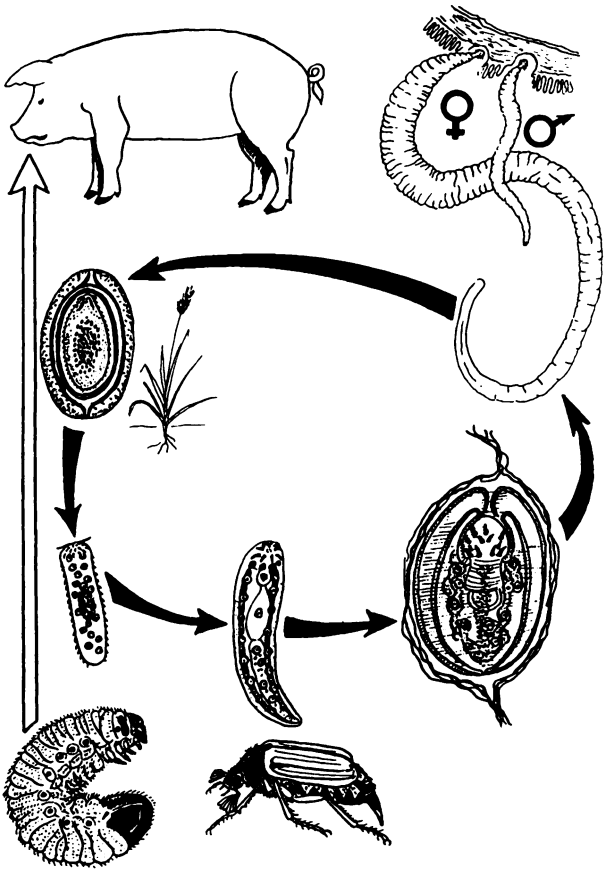

Fig. 563 Life cycle of *Macracanthorhynchus hirudinaceus* Larvae of different beetles act as intermediate hosts [51]

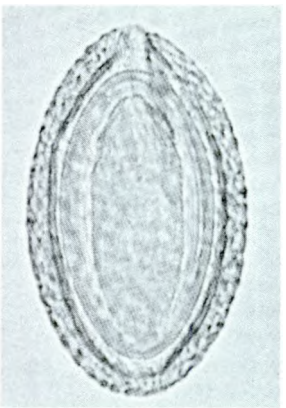

Fig. 564 Egg of *Macracanthorhynchus hirudinaceus* (70–110 × 40–65 µm) [50]

Fig. 565 *Macracanthorhynchus hirudinaceus*; anterior end with the typical spiny rostellum [10]

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2 Stages in the blood and circulatory system

PROTOZOA

TRYPANOSOMATIDAE

Trypanosoma congolense simiae

(syn. *T. porci, Nannomonas simiae*)

Hosts: Warthog, pig and camel

Vector: *Glossina brevipalpis* and *Glossina morsitans.*

Species description: This is a polymorphic form resembling *T. congolense.* Its natural host is the warthog and it is highly pathogenic for pig and camel. *Glossina* flies introduce *T. congolense simiae* to domestic pigs but thereafter mechanical transmission by *Stomoxys* spp. and tabanids is also possible. *T. congolense simiae* differs from *T. congolense* in that it is polymorphic instead of monomorphic. It is 12–24 µm long. About 90% of its forms are long and stout with a conspicuous undulating membrane, about 7% are long and slender with an inconspicuous undulating membrane and about 3% are short with an inconspicuous undulating membrane. A free flagellum is usually absent. The disease is characterized by disseminated intravascular coagulation, haemorrhage in the heart, lungs and meninges (☞ CATTLE, ■ 2 p. 55)

Geographic distribution: Tropical East and Central Africa

Symptoms: Hyperthermia, inappetence, depression, polypnoea and death within a few hours

Significance: *T. congolense simiae* is the most important trypanosome of domestic swine and causes a very acute and fatal disease in pigs.

Diagnosis: This is based on the peracute course, clinical signs of the disease and the demonstration of large numbers of organisms in the peripheral blood.

Therapy: Isomethamidium chloride (12.5–35 mg/kg, im.) at increased dosage rates or a combination of quinapyramine (7.5 mg/kg, sc.) and diminazene aceturate (5 mg/kg, im.) can be used to treat *T. congolense simiae* infections.

Prophylaxis: The suramin-quinapyrimidine complex (4 ml solution/5 kg, ☞ HORSES AND DONKEYS, ■ 2) protects piglets against *T. congolense simiae* for a period of 3 months and adults for 5 months. Insecticide application to avoid bites by tsetse flies may be necessary in endemic areas, where losses due to *T. congolense simiae* and other trypanosomes are high.

Trypanosoma (syn. *Pycnomonas*) *suis*

Hosts: Pig

Vector: *Glossina brevipalpis* (savannah regions) and Glossina vanhoofi (forest)

Species description: This suid specific trypanosome is poorly known. It is monomorphic, 14–19 µm in length, stout and with a short free flagellum. *T. suis* causes a chronic infection in adults and a more acute disease with death in less than 2 months in young pigs. It cannot be transmitted to sheep, goat and other domestic animals.

Geographic distribution: Central Africa (Zaire, Tanzania, Burundi)

Symptoms: Fever, apathy, progressive weakness and death in suckling piglets; unthriftiness in adult pigs

Significance: *T. suis* may cause great losses in suckling pigs.

Diagnosis: Clinical signs and the demonstration of the organisms in the blood

Therapy and Prophylaxis: ☞ *T. congolense simiae* and ☞ CATTLE, ■ 2

Trypanosoma congolense

Remarks: *T. congolense* occurs in swine but may regress spontaneously. Once the dis-

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ease sets in it develops a chronic course associated with emaciation, progressive anaemia, weakness and ataxia (☞ CATTLE, ■ 2).

Trypanosoma brucei

Remarks: *T. brucei* is not very pathogenic for pigs and develops as a chronic, usually mild or asymptomatic disease (☞ CATTLE, ■ 2).

Trypanosoma vivax

Remarks: *T. vivax* is generally non-pathogenic for pigs (☞ CATTLE, ■ 2).

Trypanosoma evansi

Remarks: *T. evansi* affects a wide range of hosts, including pig. The most severe disease occurs in camels, horses and dogs. The transmission is mechanical by biting flies (☞ HORSES AND DONKEYS, ■ 2). *T. evansi* infections in pig are chronic in nature and the clinical signs are unspecific (apathy, weakness) and often overlooked.

BABESIIDAE

Babesia (syn. *Piroplasma*) *trautmanni* Porcine piroplasmosis

Vector: *Rhipicephalus sinus*, *R. appendiculatus*, *R. sanguineus*, *Boophilus decoloratus* and *Dermacentor* spp.

Species description: The morphology of *B. trautmanni* is similar to that of *B. bigemina*. It is the large porcine babesia species (2.5–4 µm long and 1.5–2 µm wide), characteristically long and narrow. It occurs frequently in pairs, but the infected cells may also contain up to 6 organisms. Oval, amoeboid and ring forms may occur. Babesiosis of swine is seasonal, according to the vector. Wild boar and wart hogs may serve as natural reservoir. Mortality may reach 50%. Pigs of all age are affected.

Geographic distribution: Southern Europe, equatorial Africa

Symptoms: Fever, anaemia, haemoglobinuria, jaundice, oedema and incoordination. Abortion may occur in pregnant sows.

Significance: *B. trautmanni* infections may cause severe losses in pig farms in endemic areas. These losses are seasonal.

Diagnosis: Clinical signs, especially haemoglobinuria and icterus and demonstration of the parasites in Giemsa-stained blood smears.

Therapy: Diminazene aceturate (3.5 mg/kg, im.), trypan blue, phenamidineand quinuronium are also effective. (☞ CATTLE, ■ 2).

Prophylaxis: Regular tick control (☞ SWINE, ■ 5.1)

(Figure 566)

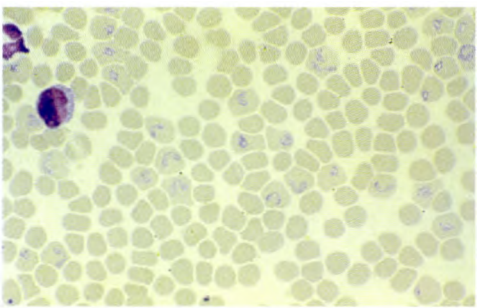

Fig. 566 *Babesia trautmanni* (2.5–4 × 2 µm); stained bloodsmear [4]

Babesia perroncitoi Porcine babesiosis

Vector: *Rhipicephalus sinus*, *R. appendiculatus*, *R. sanguineus*, *Dermacentor reticulatus* and other tick species to be established.

Species description: The morphology of *B. perroncitoi* is similar to that of *B. bovis*. It is the small porcine *Babesia* species, a small rounded form (0.7–2 µm in diameter). Oval to pyriform forms may occur (1.2–2.6 µm long and 0.7 –1.9 µm wide).

Geographic distribution: Mediterranean basin, West and Central Africa

Symptoms: Comparable to *B. trautmanni*. For significance, diagnosis, therapy and prophylaxis ☞ *B. trautmanni*

(Figure 567, 568)

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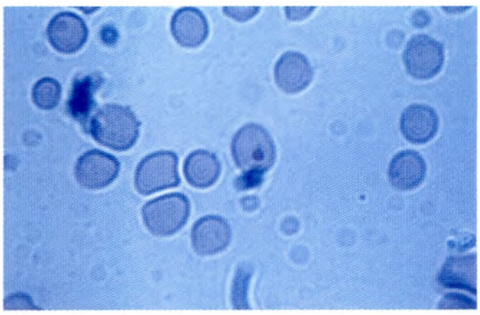

Fig. 567 *Babesia perroncitoi* (0.7–1.8 µm in diameter); stained bloodsmear [8]

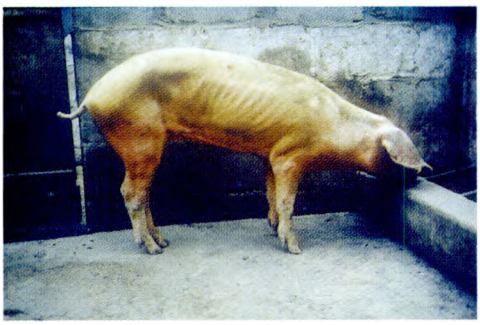

Fig. 568 Babesiosis in pigs; emaciation and icterus [8]

RICKETTSIALES

Eperythrozoon spp.

Eperythrozoon suis

Vector: *E. suis* is transmitted by parenteral routes. *Haematopinus suis* and probably other arthropods may transmit the infection. Mechanical transmission by surgical instruments (e.g. hypodermic needles) may also occur.

Species description: *E. suis* and *E. parvum* (☞ below) occur in pigs on the surface of erythrocytes. Severe cases generally occur in young pigs. Transplacental transmission may occur. *E. suis* is generally not very pathogenic but may assume increased significance together with other concomitant infections (babesiosis, trypanosomosis, etc.).

Geographic distribution: America, Europe, Asia and equatorial Africa

Symptoms: Varying degrees of haemolytic anaemia, icterus (yellow belly), fever, anorexia, weakness. Anaemia is the predominant clinical sign of *E. suis* infections in neonatal piglets. Abortion may occur in infected sows.

Significance: *E. suis* is of great economic significance in suckling pigs wherever it occurs.

Diagnosis: Acute eperythrozoonosis may be diagnosed by demonstrating large numbers of rickettsiae in Giemsa-stained bloodsmears (☞ METHODS, 3.1). The parasites may be found free in the plasma, surrounding platelets, or on the surface of erythrocytes as rings (2–3 µm in diameter). Serological tests, e.g. indirect haemagglutination, IFAT and ELISA are available and most useful for epidemiological studies. *E. suis* infection should be differentiated from *Anaplasma* infections.

Therapy: Oxytetracycline (6.6 mg/kg, im.) and other tetracyclines are effective in single dosages against *E. suis*. Haematinic drugs (e.g. iron dextran) may support recovery. If required, oral application of oxytetracycline is an effective herd treatment.

Prophylaxis: Control of ectoparasites (especially *Haematopinus suis* and *Sarcoptes suis*) may reduce *E. suis* infections (☞ SWINE, 5.1 and CATTLE, ■ 2)

Eperythrozoon parvum

Remarks: *E. parvum* is smaller than *E. suis* (0.5–0.8 µm in diameter) and non-pathogenic. It also occurs on the surface of erythrocytes.

(Figure 569)

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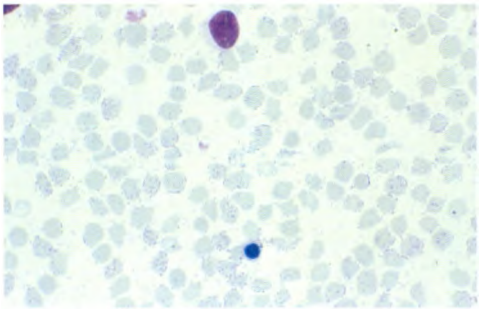

Fig. 569 *Eperythrozoon parvum* (0. 4–1 µm); stained bloodsmear [4]

HELMINTHS

- Nematoda larvae (microfilariae) found in the blood and circulatory system

Setaria congolensis

Remarks: Microfilariae occur in the peripheral blood and are ingested by mosquitoes (☞ SWINE, ■ 4.4 and CATTLE, ■ 2). The adult worms occur in the abdominal cavity of pigs.

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3 Stages in the urogenital system

HELMINTHS

- Nematoda found in the urogenital system

Stephanurus dentatus "Kidney worm"

Location: Adult *S. dentatus* worms are found in the kidneys, walls of the ureters and perirenal fat. Immature larvae may be found in the liver and peritoneal cavity and occasionally in other tissues or organs (e.g. thoracic organs or spinal cord, etc.)

Hosts: Pig

Species description: Adult *S. dentatus* worms (2– 4 cm long and about 2 mm in diameter) are usually found in pairs within cysts (up to 4 cm in diameter) in the kidney or nearby fat. It is mainly a parasite of pigs raised outdoors. Eggs pass out with the urine and hatch in 2 days. Infective third-stage larvae develop within 4 days and may infect pigs percutaneously or by being ingested. In addition earthworms may ingest and accumulate larvae. Consequently pigs may acquire heavy infections by eating earthworms. The larvae then migrate via blood vessels to the liver, where they wander for 3 months or more. The larvae proceed to migrate through the peritoneum to the kidney, where cysts are formed. Eggs do not appear in the urine until 9–16 months after infection. Patent infections in piglets < 5 months old were acquired prenatally. Female worms live as long as 3 years and produce up to 1 million eggs per day. The principal economic loss results from condemnation of organs affected by migrating larvae. The liver is usually most severely affected, showing cirrhosis, scar formation, extensive thrombosis of the portal vessels. Kidney and lung damage is also common.

Geographic distribution: Tropical and subtropical regions

Symptoms: Heavy infections result in reduced growth. Pleuritis and peritonitis are common. Inappetence, emaciation, ascites due to cirrhosis. The infection is a herd problem and lack of growth and wasting in the pig herd are the predominant signs.

Significance: The kidney worm causes great losses due to poor growth and condemnation of damaged organs (mainly liver) and tissues at slaughter.

Diagnosis: During the long prepatent period clinical signs may be present but no eggs can be found in the urine. Prepatent infections are therefore very difficult to diagnose and a definite diagnosis depends on the demonstration of the worms at necropsy.

Therapy: Ivermectin (300 µg/kg, sc.), fenbendazole (3 mg/kg/day for 3 days), oxfendazole (3–4.5 mg/kg, po.) and levamisole (8 mg/kg) are effective against *S. dentatus*.

Prophylaxis: This is largely a matter of hygiene. Indoor housing and rigorous outdoor sanitation (provision of a concrete platform under the feeding troughs) reduces the kidney worm incidence drastically. In traditional pig rearing systems control relies on regular treatment of young pigs and the elimination of old (inapparent but egg-excreting) animals.

(Figures 570, 571, 572, 573)

Fig. 570 *Stephanurus dentatus* (male: 20–30 mm; female: 30–45); adult worms found in a kidney cyst [8]

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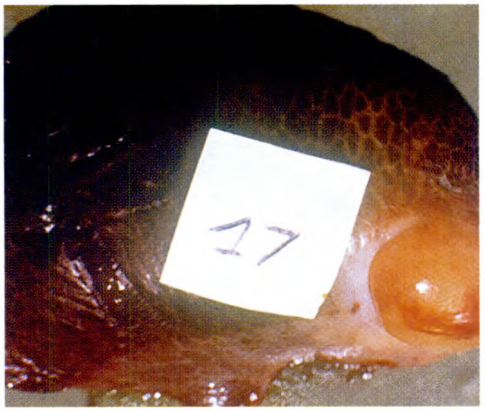

Fig. 571 Liver damage associated with *Stephanurus dentatus* is often an important cause of disease [8]

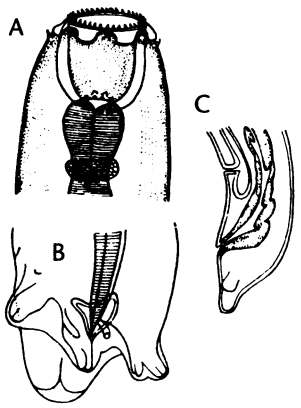

Fig. 572 *Stephanurus dentatus*; anterior end (A), posterior end of a male (B) and tail end of a female (C) [34]

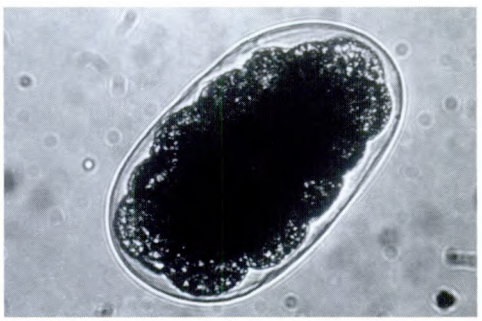

Fig. 573 Egg of *Stephanurus dentatus* (90–114 × 53–70 µm) appears in the urine [8]

Dioctophyma renale

Remarks: *D. renale* occurs most commonly in mink, dog and many other species. It may occasionally be found in pig. The females are the largest nematodes known (75–100 cm long and > 1 cm in diameter). The adults live in the renal tissue and destroy the renal parenchyma gradually as they grow. Pitted, thick shelled eggs with bipolar plugs are passed in the urine. Oligochaete annelids act as intermediate hosts. Infection is acquired by ingesting larvae or transport hosts (e.g. fish, frogs) which contain encysted larvae. After ingestion larvae migrate from the stomach to the peritoneal cavity and liver before maturing in the kidneys. Diagnosis is made on demonstration of eggs in the urine and mature parasites in the kidneys at necropsy. *D. renale* infections are incidentally found at necropsy and are something of a curiosity. Most modern benzimidazoles are expected to be effective. Surgical removal may often be necessary.

(Figures 574, 575, 576)

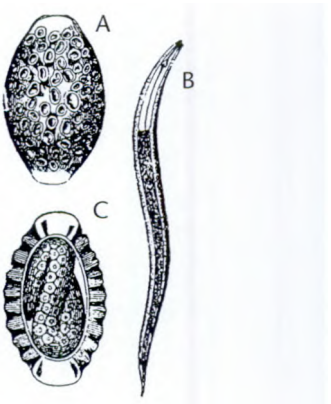

Fig. 574 *Dioctophyma renale*; (A) egg (71–84 × 46–52 µm); (B) embryonated egg and (C) larva after hatching [9]

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Fig. 575 *Dioctophyma renale*; male (35 cm × 3–4 mm long) [9]

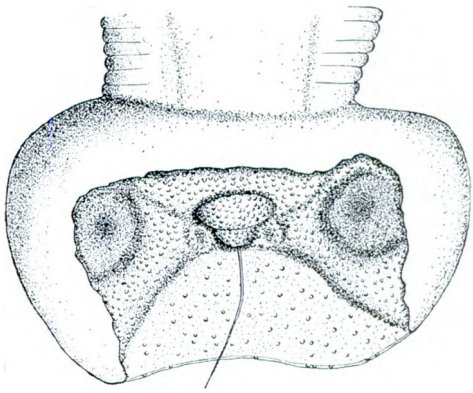

Fig. 576 *Dioctophyma renale*; male bursa (schematic) [9]

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4 Stages in internal organs

4.1 Locomotory system

4.1.1 Muscles

4.2 Liver

4.3 Respiratory system

4.4 Abdominal cavity

4.5 Pancreas

4.6 Central nervous system

4.1 Locomotory System

4.1.1 Muscles

PROTOZOA

Toxoplasma gondii

Location: Oocysts are produced in the epithelial cells of the small intestine of cats and other Felidae. *Toxoplasma* cysts (containing merozoites) may be found in a wide variety of mammals and in several organs (including brain).

Hosts: Felidae are definitive hosts, many mammal species (including all domestic animals) and man act as intermediate hosts.

Species description: Infection of intermediate hosts by ingesting sporulated oocysts or infected meat or infected animals. The definitive host can also be the intermediate host. After infection parasitaemia (trophozoites or tachyzoites, crescent-shaped, 4–8 × 2–4 µm) occurs and susceptible tissues all over the body are invaded and the parasites multiply. The parasites finally remain in cysts (bradyzoites) and only in the most receptive tissues. In general, the parasite cysts appear in the brain, eye, lungs, liver, skeletal and cardiac musculature. *T. gondii* cysts in muscles are 100 µm in diameter, the tachyzoites in the cysts are 6–8 µm long. Infection of the intermediate hosts occurs by ingestion of sporulated oocysts from the faeces of the final host (cat) or *Toxoplasma* cysts by raw meat from infected intermediate hosts (horse, swine, sheep and other species). Transplacental infection is common (☞ SHEEP AND GOATS, ■ 4.1.1). In carnivores, infection is acquired by ingestion of fresh, infected meat or carcasses of a wide range of intermediate hosts. In herbivores, most infections are thought to result from ingestion of herbage contaminated by *Toxoplasma* oocysts derived from cat faeces. In omnivores, including man, infection seems to result both from ingestion of undercooked meat and from accidental ingestion of oocysts.

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Cave: Raw meat of pigs is an important source of infection for humans. Populations at risk are serologically negative pregnant women and immunosuppressed persons.

Geographic distribution: World-wide

Symptoms: The majority of infections is subclinical. Fever, apathy, diarrhoea, respiratory distress (coughing, dyspnoea) and CNS symptoms may occur during the acute phase. Pregnant sows may abort or farrow weak or stillborn pigs.

Significance: *T. gondii* is one of the most common parasites in animals and probably also in man. Uncooked meat of swine is the major source of infection for man. Toxoplasmosis is an important cause of abortion and stillbirths in sheep, goats and sometimes in pigs.

Diagnosis: Demonstration of *Toxoplasma* cysts in stained biopsy sections or in artificially digested tissue. The diagnosis should be supported by immunohistochemistry (using mono- or polyclonal antibodies), isolation of the organisms and serological testing. Isolation may be based on intraperitoneal injection of suspected material into mice free of natural *Toxoplasma* infections in order to detect cysts in mouse brain after 4–6 weeks. Isolation may also be carried out by inoculation of the suspected material onto a tissue culture (e.g. Vero cells). Several serological tests are available (also as kits) for the detection of *Toxoplasma* antibodies (Sabin-Feldman dye test, Indirect Fluorescent Antibody Test, indirect haemagglutination, ELISA (☞ METHODS, 5). The B1 gene-derived PCR provides a highly sensitive tool for the diagnosis of *T. gondii* in clinical material. As little as one tachyzoite may be detected with this method.

Therapy: Specific treatment is not recommended for domestic animals. Sulfadiazine (73 mg/kg, po.) acts synergistically with pyrimethamine (0.44 mg/kg, po.) in laboratory animals and man. Clindamycin (10–40 mg/kg, po.; 25–50 mg/kg, im.) is the drug of choice for treatment of *Toxoplasma* infections in dogs and cats.

Prophylaxis: Raw meat of pigs is the main source of infection in man. Prevention is based on the interruption of the complex life cycle. Cats and pigs should not be kept together. Control of rodents is an essential part of the prevention of *Toxoplasma* infection in domestic animals, especially pigs, since rodents attract cats but can also be a source of infection if they are eaten by pigs. Feeding of uncooked garbage should be eliminated.

(Figures 577, 578, 579, 580)

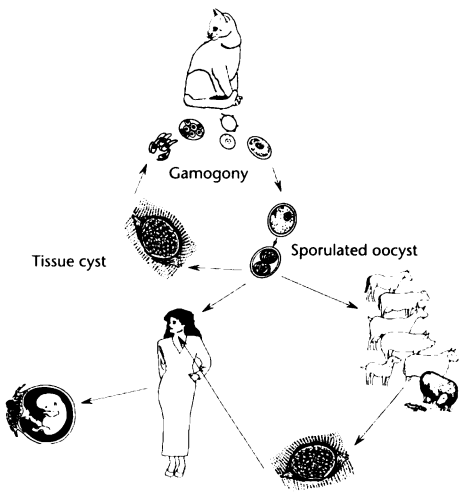

Fig. 577 Transmission cycle of *Toxoplasma gondii* Gamogony occurs in the final host (cat) and oocysts are excreted which infect domestic animals and man where tissue cysts are formed. *T. gondii* is of great importance when seronegative pregnant women become infected. The parasite may then affect the fetus [10]

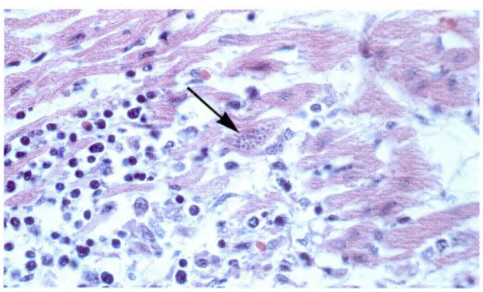

Fig. 578 *Toxoplasma gondii*; muscle cysts

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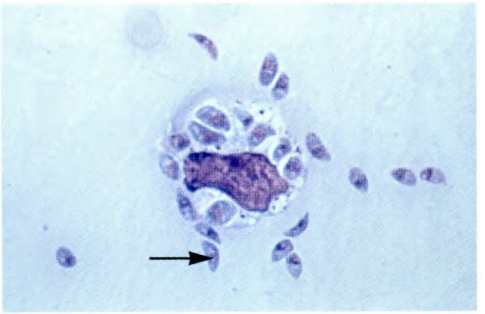

Fig. 579 *Toxoplasma gondii*; tachyzoites (6–8 µm long) [4]

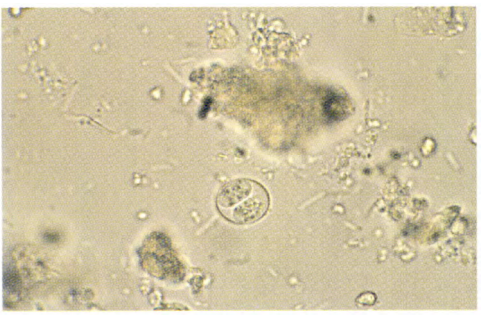

Fig. 580 Sporulated oocyst (12.4 × 10.5 µm) of *Toxoplasma gondii* are excreted exclusively in the faeces of Felidae. Sporulated oocyst are infectious for animals and human beings [10]

Sarcocystis spp.

Cysts of the following three species are found in swine: *S. suihominis*, *S. suicanis* (syn. *S. miescheriana*) and *S. porcifelis*.

Location: Cysts (Sarcocysts) are found in the muscles of swine. Infective sporocysts are passed in the faeces of the final host.

Hosts: The pig is the intermediate host of all three species is the pig. Definitive hosts of *S. suihominis* are man and primates, of *S. suicanis* the dog, wolf, fox and of *S. porcifelis* the cat.

Species description: *Sarcocystis* spp. develop in a 2-host cycle consisting of an intermediate host (prey) and the final host (predator). *S. suihominis* (common), *S. suicanis* (common) and *S. porcifelis* (uncommon) are the three species found in swine. In all three species swine acts as intermediate host, where the asexual part of the *Sarcocystis* life cycle takes place and cysts are formed in striated or cardiac muscles. These cysts contain infective, banana-shaped merozoites 60 days after infection. The sexual cycle is found in intestinal epithelial cells of the final hosts where gametogony occurs. Infection is acquired by ingestion of infective sporocysts of the final host.

Geographic distribution: World-wide

Symptoms: Most infections are asymptomatic and the parasite (cysts) is discovered only at slaughter. Intensity of clinical signs depends on the number of ingested sporocysts. More than 1 million is lethal. Acute sarcocystosis shows a biphasal fever between 5–9 days and 11–15 days after infection. During the second phase apathy, dyspnoea, anaemia and cyanosis (skin, ears, tail), muscle spasms, hyperexcitability and prostration are predominant. Abortion following heavy infection is reported in cows and may also occur in pregnant sows.

Significance: *Sarcocystis* infections are very common and generally asymptomatic in swine. However, when high numbers of sporocysts are ingested they may cause a severe disease condition.

Diagnosis: Generally not possible with the naked eye. Demonstration of muscle cysts in histological sections or following artificial digestion of infected muscles. Acute cysticercosis in swine may be diagnosed by Giemsa-stained bloodsmears taken from the surface of organs at necropsy or by stained histological sections. Serological techniques may be employed to discover chronic infections (important is the presence of cysts).

Therapy: Therapy is generally not indicated. Halofuginone (0.5 mg/kg, po.) may be used.

Prophylaxis: Dogs and other carnivores should not be allowed to eat raw meat, offal or dead animals. Also, man should not consume uncooked meat. Supplies of grain and feed should be kept covered. Dogs and cats

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should be kept away from buildings, used to store feed or house animals. Amprolium (100 mg/kg, po.) daily for 30 days and Halofuginone 3 ppm) may be used for prophylaxis in heavily infected herds.
(Figures 581, 582, 583)

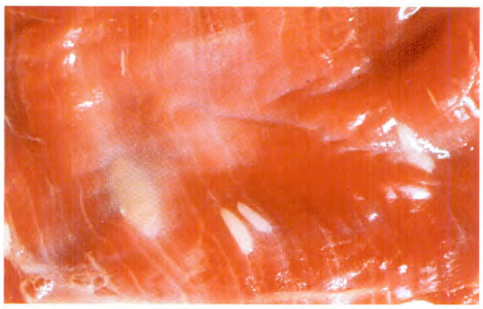

Fig. 581 Cysts of *Sarcocystis suicanis* in diaphragm (up to 1.5 mm long)

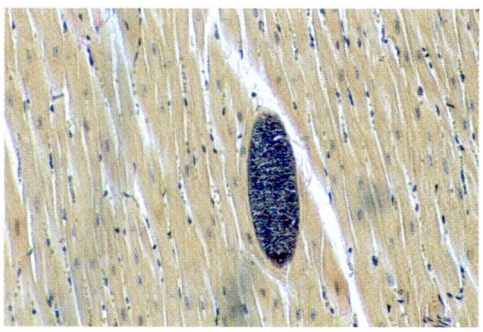

Fig. 582 Cysts of *Sarcocystis* sp. in the musculature

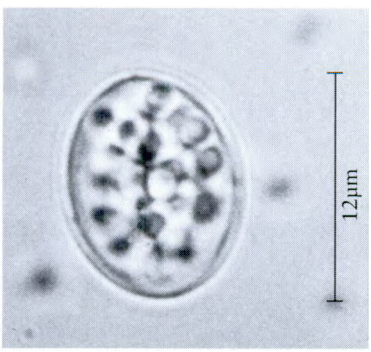

Fig. 583 Sporocyst of *Sarcocystis suicanis*; excreted by the dog [4]

HELMINTHS

- Cestoda larvae found in the muscles

Cysticercus cellulosae Cyst of the human tapeworm *Taenia solium*

Location: *C. cellulosae* cysts are found in the skeletal and cardiac muscles of pigs and man. Adult tapeworms (*T. solium*) are found in the small intestine of man.

Hosts: Pig is an intermediate host of the human tapeworm *T. solium*. Man can be both final and intermediate host of *T. solium*.

Species description: Cysts (cysticerci) are white, 5–18 mm in diameter and contain one protoscolex with a hook collar. Infection of pigs occurs by ingesting gravid proglottids or eggs which are passed in the faeces of infected man. These proglottids contain embryophores, releasing oncospheres which migrate to skeletal muscle and the heart via blood stream. Cysticerci develop within 2–3 months after the ingestion of the eggs and remain infective up to 2 years. Man acquires infection by eating infective pig muscle. Cysticerci are released in the human small intestine where the scolices evaginate and attach to the intestinal wall. The prepatent period is 7–8 weeks.

Geographic distribution: World-wide

Symptoms: Cysticercosis in swine is generally asymptomatic.

Significance: Cysticercosis in swine is not associated with pathology or clinical disease. Pathology in man is related to the number and localization of cysticerci. Neurocysticercosis in humans is a serious condition often with fatal consequences. Adult tapeworms produce no significant pathological changes in man.

Diagnosis: Antemortem diagnosis is based on the detection of specific antibodies (e.g. ELISA). Cysticercosis in swine is diagnosed at slaughter where macroscopic cysts may be discovered.

Therapy: Therapy of cysticercosis in swine is generally not indicated. *T. solium* can be treated with niclosamide or praziquantel,

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the latter is also effective against cerebral cysts of *T. solium* in man. Fenbendazole (5 mg/kg, po.) administered for 7 days and mebendazole (25 mg/kg, po.) for 5 days were described to be effective against cysticercosis in swine.

Prophylaxis: Man should not consume raw meat of pigs. Freezing for one week destroys the larvae that are a source of human infection. Improved sanitation (e.g. separation of human excrements and pig feeding ground) reduces the incidence of autochthonous infection and the infection of swine.

(Figures 154, 584, 585)

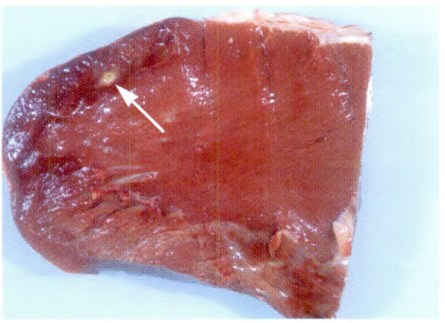

Fig. 584 *Cysticercus cellulosae* in muscle tissue (0.5–2 cm in diameter)

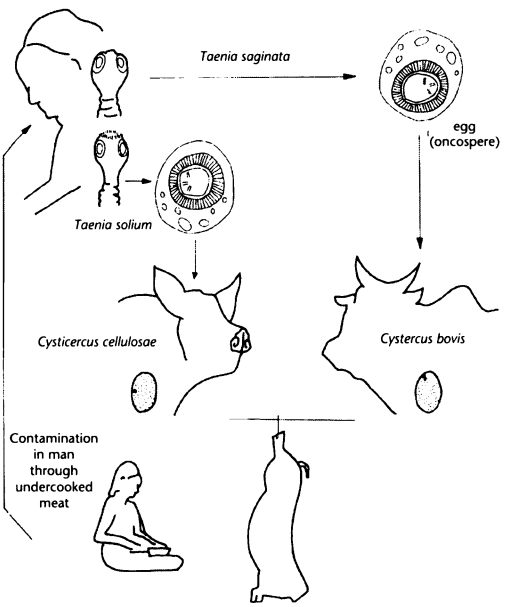

Fig. 585 Transmission cycle of *Taenia solium* (larval stage: *Cysticercus cellulosae* in swine) and *Taenia saginata* (larval stage: *Cysticercus bovis* in cattle) [16]

- **Nematoda larvae and adult nematodes found in the muscles**

***Trichinella* spp. (*T. spiralis, T. britovi, T. nativa, T. nelsoni* and *T. pseudospiralis*)**

Trichina worm

Location: Adult worms occur in the small intestine. Encysted larvae are found in muscles.

Hosts: Pig, wild boar, rat, man and many other mammals

Species description: Adult trichina worms are very small nematodes, 2–4 mm long. A females produces several hundreds of larvae which penetrate the intestinal wall and migrate via the lymph and blood vessels to the muscles where they encyst and remain viable for years. Further development only occurs if the infected tissue is ingested by another host, often a rat, man, or pig. Swine may therefore be both intermediate and principal host for trichina worms. After the ingestion of trichina cysts the larvae are liberated and mature in several days. The adult worms copulate in the small intestine and the females penetrate the mucosa to produce larvae for 2 weeks.

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Geographic distribution: World-wide

Symptoms: Infection of the intestines may cause diarrhoea and fever but is generally not typical to allow diagnosis. Infection of the muscles may cause myositis, muscle pain, stiffness, polypnoea, oedema, eosinophilia and in severe cases death.

Significance: *Trichinella* spp. are less pathogenic in swine than in man. The larvae accumulate in the diaphragm and jaw muscles of pigs. Adults cause few problems in swine whereas in man they cause nausea, diarrhoea and abdominal pain. 4–6 days after infection death may occur in humans and is usually caused by paralysis of the respiratory muscles but also infection of the brain.

Diagnosis: At necropsy, encysted larvae may be found in muscles (diaphragm) which are squeezed in a compressorium for subsequent microscopic examination. Muscles may also be artificially digested to detect trichina larvae (☞METHODS, 2.1 and 2.2). Serological tests (ELISA, IFAT) are available to detect infections in domestic animals and man (☞METHODS, 5.2 and 5.3).

Therapy: *Trichinella*-infected pigs are generally not treated. Their meat should be condemned!

Prophylaxis: To prevent infection of pigs, garbage must be cooked before being fed to pigs. Human infections can be controlled by thorough cooking or freezing of pork and game animals before consumption.

Cave: T. nativa and related species may survive freezing.

(Figures 586, 587, 588)

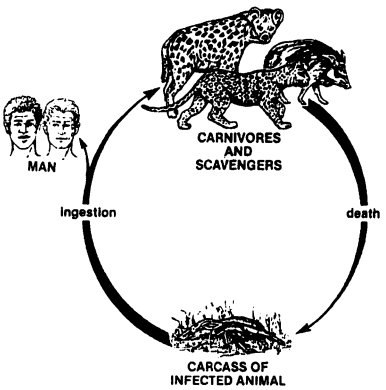

Fig. 586 Transmission cycle of *Trichinella* spp. in tropical Africa; the bushpig and other wild suids are the principal source of infection for man [21]

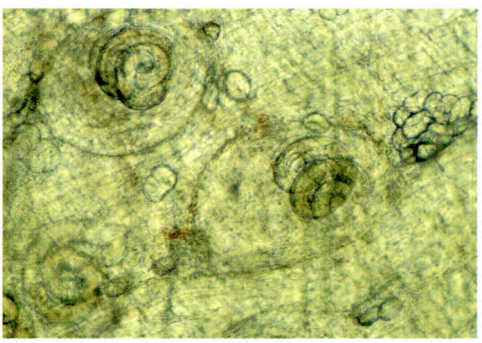

Fig. 587 Muscle cyst containing a *Trichinella spiralis* larva (0.4–0.6 × 0.25 mm)

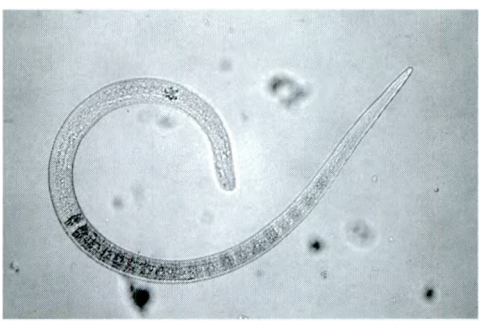

Fig. 588 *Trichinella spiralis* larva (800–1000 µm long) obtained from a digested muscle cyst

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4.2 Liver

HELMINTHS

- Trematoda found in the liver

Fasciola gigantica and *Fasciola hepatica* Large liver flukes

Remarks: *Fasciola* spp. are normally found in ruminants (☞ CATTLE, ■ 4.2) but many other mammal species including pig can also be affected. Pigs acquire *Fasciola* infections especially if they are grazing in swampy areas where the intermediate hosts are present. Fasciolosis in swine is generally asymptomatic. Losses occur by liver condemnation at slaughter.

Dicrocoelium spp. Small liver flukes

Remarks: *Dicrocoelium* spp. are normally found in ruminants but many other mammal species including pig can also be affected (☞ CATTLE, ■ 4.2).

Eurytrema pancreaticum Pancreatic fluke

Remarks: *E. pancreaticum* is a parasite of the pancreatic ducts and occasionally of the bile ducts of sheep, pigs and cattle. There are no obvious clinical signs and *Dicrocoelium*-like eggs can be demonstrated in the faeces (☞ SHEEP AND GOATS, ■ 4.5).

- Cestoda found in the liver

Echinococcus granulosus Hydatid cysts

Location: Larvae (hydatid cysts) of *E. granulosus* are found in the liver and the lungs of swine and other intermediate hosts. Adult tapeworms are found in the small intestine of the dog and other canids.

Hosts: Canids are the final hosts of *E. granulosus*. Intermediate hosts are herbivores, swine, humans and many other domestic animals.

Species description: Pigs are known to be intermediate hosts in many countries but generally there are only little data available about the role of pigs as intermediate hosts in African countries. Intermediate hosts acquire infection by eating eggs shed in the faeces of canids. Eggs develop to hydatid cysts which may reach the size of an orange over the period of several months. These cysts are infective for canids.

Geographic distribution: World-wide

Symptoms: Generally inapparent infections in pigs. Pressure atrophy of the liver, digestive disturbances and ascites may be found in heavy infections. Dyspnoea and coughing may be observed when lungs are greatly affected.

Significance: Hydatid cysts generally cause no clinical disease in swine.

Diagnosis: Demonstration of hydatid cysts at necropsy or at slaughter. Cysts containing "*Echinococcus* sand" which is composed of thousands of typical protoscolices with a hook collar. Antemortem diagnosis is difficult. Serological tests are available and of great importance for epidemiological studies.

Therapy: Therapy is generally not indicated. Most of the modern benzimidazoles applied orally over the period of 10–20 days at doubled dosage rates may be used to reduce the number and size of hydatid cysts.

Prophylaxis: In endemic areas a strict separation of dogs from pigs is necessary to reduce the incidence of hydatidosis in swine.

(Figure 589)

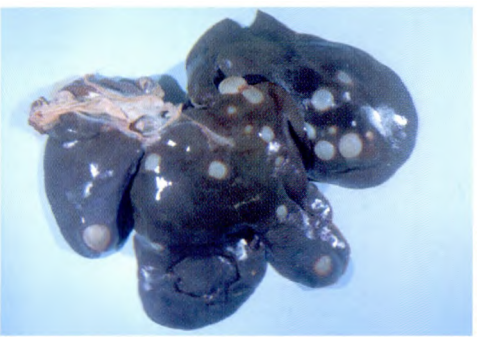

Fig. 589 Hydatid cyst of *Echinococcus granulosus* found in the liver of a pig

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Cysticercus tenuicollis
Cysts of *Taenia hydatigena*

Remarks: Larvae of the canine cestode *Taenia hydatigena* may also be found in swine. Larval migration through the liver may produce haemorrhagic tracks and masses of larvae may cause a severe condition associated with anaemia, fever, inappetence and death (hepatitis cysticercosa). Larvae wander under the liver capsule and form thin-necked cysts. These cysts are always covered by a layer of serosa, and therefore they seem to be attached to the peritoneum. Preferred serosal sites of cysticerci are the omentum and mesentry although they may also be found attached to the serosa of the liver. Cysticerci are up to 8 cm in diameter and cause no pathology. Diagnosis is made at necropsy or slaughter (☞ CATTLE, ■ 4.4).
(Figure 590)

Fig. 590 Haemorrhagic tracks in the liver parenchyma due to a high number of migrating *Cysticercus tenuicollis* larvae

- Nematoda found in the liver

Ascaris suum

Remarks: Migration of larvae through the liver causes haemorrhage, necrosis and fibrosis that appears as white spots ("milk-spots") under the capsule after healing. Condemnation of livers at slaughter is one of the major losses due to *A. suum* infections in pig farming (☞ SWINE, 1).
(Figure 591)

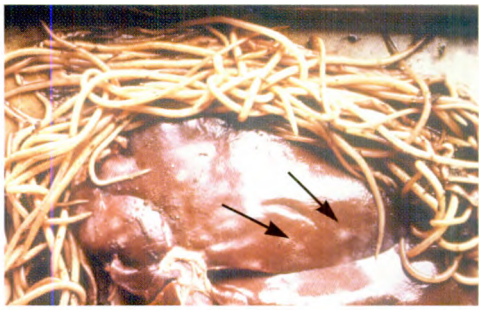

Fig. 591 Liver of swine with "milk spots" (arrows) and masses of adult *Ascaris suum* found in the small intestine [10]

4.3 Respiratory system

HELMINTHS

- Nematoda found in the lungs and trachea

Metastrongylus spp. (***M. apri***, syn. *M. elongatus*, ***M. pudendotectus***, ***M. salmi*** and ***M. madagascariensis***) Porcine lungworms

Location: Bronchi and bronchioles
Hosts: Pig and wild boar
Species description: The adult parasites are slender and white, with males measuring up to 25 mm in length and females up to 48 mm. *Metastrongylus* spp. have an indirect life cycle in which most earthworms may serve as intermediate hosts. Eggs are laid in the bronchi, coughed up and passed in the faeces. After the ingestion by earthworms the eggs hatch and within 10 days infective third-stage larvae develop within the earthworms. Pigs become infected by ingesting earthworms. The third-stage larvae penetrate small intestine of the pig and proceed via lymph and blood vessels to the lungs. The prepatent period is 2–4 weeks. *M. elongatus* is the most common species.

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Natural infections may involve one or more *Metastrongylus* species.

Geographic distribution: World-wide, with the exception of *M. madagascariensis* which has only been found in Madagascar.

Symptoms: Bronchitis, coughing, pneumonia, retarded growth in pigs kept on permanent pastures

Significance: Lungworms cause considerable economic losses due to reduced weight gain, respiratory disturbances following secondary infections. *Metastrongylus* spp. were also suspected to carry the virus of swine influenza and classical swine fever (hog cholera). Lungworms primarily cause clinical disease in young pigs, whereas older pigs show very few signs.

Diagnosis: Characteristic eggs are found in the faeces. At slaughter, the adult worms can be demonstrated. Serodiagnostic tests may also be used to detect the infections.

Therapy: For individual therapy ivermectin (300 µg/kg, sc.) and levamisole (7.5 mg/kg, sc.) are highly effective against adult lungworms. The broad spectrum anthelmintics such as fenbendazole (5 mg/kg, over 5– 15 days), flubendazole (30 ppm, over 10 days) are also effective when administered orally for several days. Febantel (20 mg/kg, po.) and mebendazole (30 ppm) are effective against *Metastrongylus* spp. at increased dosage rates.

Prophylaxis: Lungworm control is closely associated with earthworm control. Pigs kept clear of soil, especially during the rains may reduce disease incidence. Young pigs (< 6 months) should not come in contact with earthworms. If swine are not housed in clean quarters, nose rings will prevent pigs from rooting for earthworms

(Figures 538, 561, 562, 592, 593, 594)

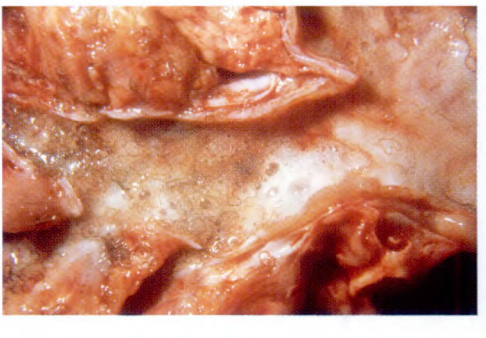

Fig. 592 *Metastrongylus apri* in the trachea of a pig (males: 11–26 mm long; females: 28–60 mm long) [8]

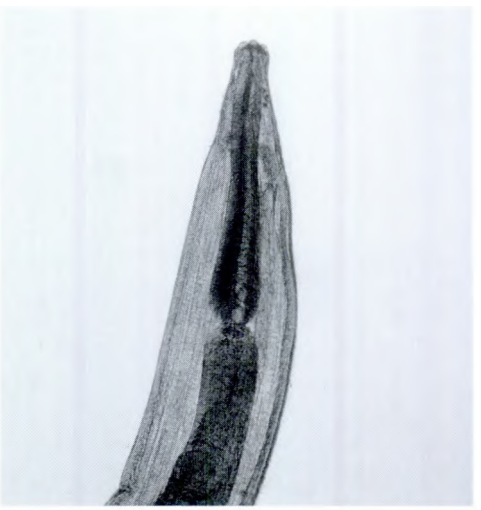

Fig. 593 *Metastrongylus apri*; anterior end

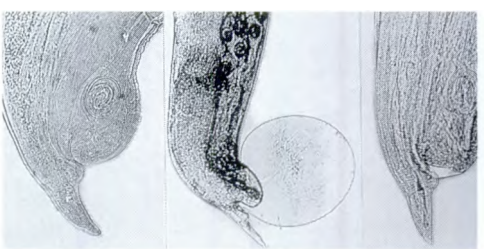

Fig. 594 Posterior end of female lungworms of pigs: (a) *Metastrongylus apri*, (b) *M. pudendotectus* and (c) *M. salmi* [4]

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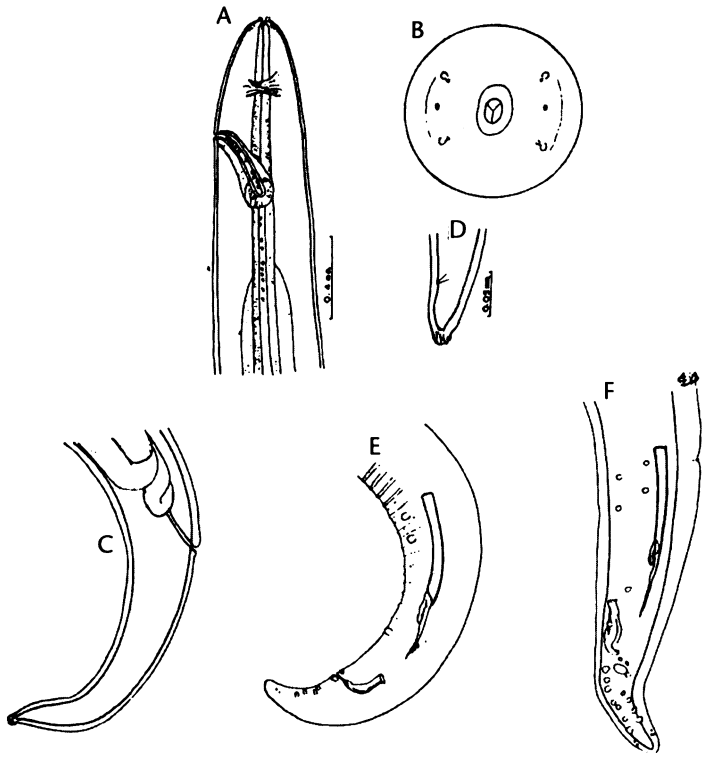

Fig. 595 *Setaria congolensis*; female anterior end (A), anterior end frontal view (B), female posterior end (C), female posterior end (D), male posterior end lateral (E) and male posterior end ventral (F) [34]

4.4 Abdominal cavity

HELMINTHS

- Nematoda found in the abdominal cavity

Setaria congolensis

Remarks: *S. congolensis* occurs in the peritoneal cavity of pigs in Africa (Zaire). Microfilariae occur in the blood and are ingested by mosquitoes (*Aedes* spp., *Culex* spp. and *Anopheles* spp.) which act as vectors. The exact life cycle of *S. congolensis* is unknown. Adult worms in the abdominal cavity are non-pathogenic. Pathogenic effects may occur when microfilariae migrate into the eye or the CNS or other organs. Little is known about *Setaria* infections in pigs and it seems that the worms are occasionally discovered at slaughter without pathological alterations or previous clinical signs. If therapy and prophylaxis are required ☞ *Setaria equina* (☞ HORSES AND DONKEYS, ■ 4.4 and *Setaria labiato-papillosa* (☞ CATTLE, ■ 4.4). (Figure 595)

4.5 Pancreas

HELMINTHS

- Trematoda found in the pancreas

Eurytrema pancreaticum Pancreatic fluke

Location: It occurs in the pancreatic ducts and occasionally the bile ducts (☞ SHEEP AND GOATS, ■ 4.5 and CATTLE ■ 4.5).

Hosts: Pigs, sheep and cattle

Species description: These flukes are 8–16 mm long, 6 mm wide and belong to the Dicrocoeliidae. The first intermediate hosts are terrestrial snails (*Bradybaena* spp.) and the

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second intermediate hosts are grasshoppers (*Conocephalus* spp.) where the cercariae encyst. After the ingestion of grasshoppers larvae migrate to the pancreatic and bile ducts where they mature and produce eggs 11–14 weeks after infections.

Geographic distribution: Asia, South America, East Africa

Symptoms: There are no obvious clinical signs.

Significance: Losses may occur due to fibrotic, necrotic and degenerative lesions of the pancreas and biliary ducts and reduced growth in animals with heavy infections.

Diagnosis: *Dicrocoelium*-like eggs can be demonstrated in the faeces, adult flukes may be seen at slaughter or necropsy.

Therapy: The same compounds may be used as in *Dicrocoelium* infections (☞ CATTLE, ■ 4.2).

Prophylaxis: The control of intermediate hosts may not be feasible, especially if pigs are kept permanently on contaminated pastures or soil.

4.6 Central nervous system

PROTOZOA

Toxoplasma gondii

Remarks: *Toxoplasma* cysts may occur also in the brain of pigs (☞ also SWINE, ■ 4.1). Fever, weakness, and incoordination, associated with a high mortality in neonatal piglets may indicate the brain invasion by the parasite in the acute phase. Chronic infections (*Toxoplasma* cysts in the brain) may be asymptomatic.

(Figure 596)

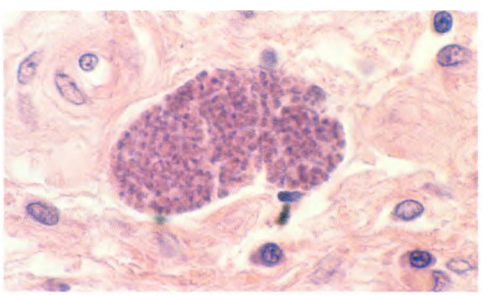

Fig. 596 *Toxoplasma gondii*; thin-walled cyst in the brain (25 × 50 µm)

Sarcocystis suihominis

Remarks: Tissue cysts may occur in the skeletal muscle, the heart and also in the brain. These cysts contain merozoites. *S. suihominis* generally does not cause CNS symptoms, except during the acute phase of heavy infections, when death may occur. Final host is man (☞ SWINE, ■ 4.1).

PROTOZOA

- **Cestoda cysts found in the central nervous system**

Cysticercus cellulosae

Remarks: Larvae (cysticerci) of the human tapeworm *Taenia solium* are occasionally found in the CNS of pigs (☞ SWINE, ■ 4.1).

- **Nematoda found in the central nervous system**

Trichinella spp.

Remarks: Larvae may sometimes be found in the CNS of pigs (☞ SWINE, 1)

Stephanurus dentatus

Remarks: Larvae may occasionally invade the CNS of pigs (☞ SWINE, ■ 3)

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5 Stages on the body surface

5.1. Skin and coat

5.1 Skin and coat

HELMINTHS

- Nematoda found in the skin

Suifilaria suis

Location: Subcutaneous and intermuscular connective tissue

Host: Pig

Species description: The adult parasites are difficult to see. Males are 17–25 mm long and the female worms are 32–40 mm long and 0.15–0.17 mm wide. The hind end of the male is spirally coiled and bears two unequal spicules. The tail of the female shows a number of small tubercles. The life cycle is not known. The female worms lay eggs in the skin.

Geographic distribution: Southern Africa

Symptoms: Numerous small vesicular eruptions on the skin

Significance: This filaroid worm produces skin vesicles, containing eggs, which may burst and become secondarily infected. Apart from producing nodules *S. suis* does not affect the health of pigs.

Diagnosis: Multiple, whitish nodules in the skin and connective tissues. These nodules contain eggs which measure 51–61 x 28–32 µm. The hard, white nodules of *S. suis* should be distinguished from *Cysticercus cellulosae* (larval stage of *Taenia solium*) larvae which contain fluid and a scolex.

Therapy and Prophylaxis: Unknown and generally not indicated

ARTHROPODS

Arthropods are divided into two main groups:

Arachnida (arachnids) including:
- *Ticks*
- *Mites*

Insecta including:
- *Lice*
- *Fleas*
- *Dipterida*

- Arachnida found in/on the skin

– **Ticks**
(Figure 597)

Ticks are found on a wide variety of host animals and are generally not very host-specific. They are not considered to be common parasites of pigs. The economic importance of ticks is considerable because of the annoyance to the host (direct noxious effects, tick worry), transmission of diseases (Babesioses) and the fatal tick toxicoses (paralysis, sweating sickness, general toxicosis). In intensive pig production systems, ticks are seldom a problem because animals are raised in close confinements. If pigs are kept in free range systems, tick infestations may assume increased significance (☞ SWINE, ▩ 2).

Significance: Heavy tick infestations cause blood loss and skin wounds. Infested pigs itch, bite and scratch, causing self-inflicted skin trauma, susceptible to secondary infections and myiasis. Anaemia and weight loss may also occur. Ticks of potential importance to pigs belong to two families: the Ixodidae ("hard ticks") and the Argasidae ("soft ticks").

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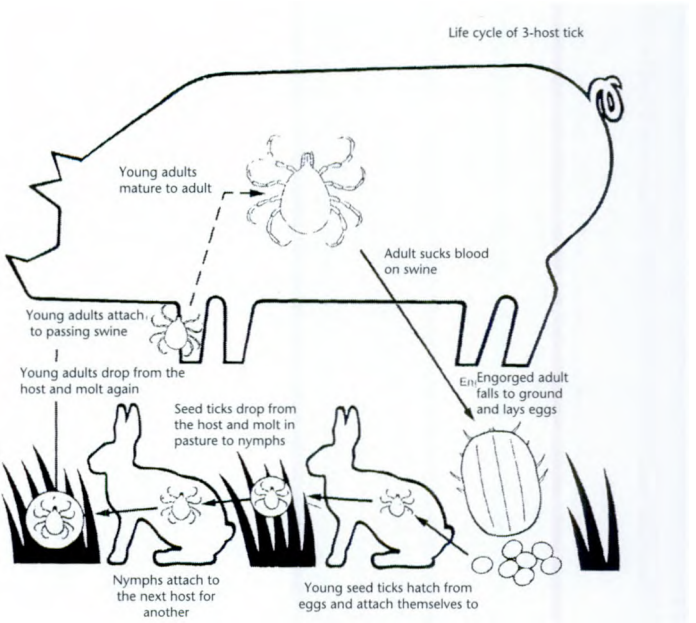
Fig. 597 Life cycle of a 3-host tick [50]



IXODIDAE ("hard ticks")

Boophilus decoloratus The blue tick

Remarks: This is a one-host tick and known to be a vector of *Babesia trautmanni* in East Africa. It occurs in Africa south of the Sahara.
(Figure 598)

Fig. 598 *Boophilus* sp.; vector of *Babesia trautmanni* adult engorged female [10]

Rhipicephalus spp. (*R. sinus, R. appendiculatus* and *R. sanguineus*)

Remarks: These species (mainly ticks of cattle) are vectors of *Babesia trautmanni* and *Babesia perroncitoi* in pigs.

Dermacentor spp.

Remarks: One-host or three-host ticks. Some species of this genus are suspected to be vectors of *Babesia trautmanni* and *B. perroncitoi* in pigs. Some species are associated with tick paralysis.

Amblyomma spp.

Remarks: This are three-host ticks. Several species of *Amblyomma* ticks are found on pigs in central and southern Africa. Apart from skin irritation at the site of attachment they may also cause tick paralysis.
(Figure 599)

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Fig. 599 *Amblyomma* sp.; adult male; members of this genus may cause severe irritation and tick paralysis in pigs [10]

***Ixodes* spp.**

Remarks: *Ixodes* spp. are three-host ticks and occur on pigs and other animals in Europe, North America and South Africa and may cause tick paralysis in domestic animals (☞ CATTLE, ■ 5.1).

ARGASIDAE ("soft ticks")

Ornithodorus moubata The eyeless tampan

Remarks: This tick lives in the native huts and in the sand under trees where animals and human beings frequently seek shelter. In huts they live in cracks in the floor or under loose soil, from which they emerge at night to feed. In burrows they bury themselves in the earth lining, coming out to feed when a suitable host is available. Adult females lay batches of about 100 eggs and broods over the eggs in the sand. Development includes a larval stage which remains quiescent until it has moulted to the nymphal stage. Several nymphal stages are passed through and the nymphs, like the adults, attack their host for short periods to feed. This tampan sucks blood from its hosts. It is extremely resistant to both starvation and desiccation (survival time off the host up to 5 and more years). *O. moubata* constitutes the main reservoir host for the virus of African Swine Fever. It is also an important vector of the virus among domestic and wild *Suidae* (warthogs, bush pigs, etc.; ☞ SHEEP AND GOATS, ■ 5.1, Figure 371).

Ornithodoros moubata porcinus
(syn. *O. porcinus*)

Remarks: This subspecies infests the burrows of warthogs and acts as the reservoir and vector of African Swine Fever. So does *O. erraticus* (syn. *marocanus*) in Spain and *O. puertoricensis* on the Caribbean islands. *O. coriaceus* transmits the virus of epizootic bovine abortion, blue tongue and possibly African Swine Fever. *O. turicata* and *O. talaje* are vectors of Mexican and American Relapsing Fever.

Otobius megnini The spinose ear tick

Remarks: Its larval and nymphal stages are parasites of the ears of dog, sheep, horse, cattle, pig and many other domestic and wild animals. The nymphs bear numerous spine-like processes. The body colour is bluish-grey and the legs, mouth parts and sines are pale yellow. Adult ticks are not parasitic and live in cracks and crevices in walls and woodwork, under stones or under the bark of trees. *O. megnini* may severely affect the ears of its hosts. Animals become restless, do not feed well and rapidly lose condition. The affected ears become filled with masses of ticks, wax and debris and are liable to be invaded by secondary organisms. These organisms may perforate the ear drum and set up infections in the middle and inner ears and death may occur from meningitis (☞ CATTLE, ■ 5.1).

- **Tick control in swine**

A number of acaricides is available as sprays or dips. Toxaphene (chlorinated hydrocarbone) as a 0.5% spray protects against reinfes-

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tations for more than 2 weeks. Other effective acaricides include coumaphos, dioxathion and malathion and the pyrethrinoids. A 5% coumaphos dust applied to the ears effectively controls the spinose ear tick (☞ CATTLE, ■ 5.1).

– Mites

Sarcoptes suis Mange mite, itch mite

Location: Skin of the head, ears, shoulders, neck, legs and tail. In heavy infestations skin lesions may occur all over the body.

Hosts: Pig and wild boar

Species description: *S. suis* is a tiny mite, 0.3–0.5 mm long with a round body, blunt mouthparts and 8 stumpy legs with an unsegmented pedicel. The legs end in bell-shaped suckers (caruncles). Female mites burrow tunnels in skin and lay eggs which develop within 17 days in a cycle of incomplete metamorphosis (eggs, nymphs, adults) in adult mites. 4–5 days after mating the new generation of adult mites starts to lay eggs. The burrowing of *Sarcoptes* mites causes great irritation, itching and lesions with exudates which become crusts. Chronic skin inflammation, accompanied by proliferation of the subcutaneous connective tissue results in thickened skin that may crack and leave wounds which may become secondarily infected. In temperate zones *S. suis* mites can survive for a few weeks off the host. In dry areas the mites die within a few days after leaving the pig. *S. suis* is transmitted directly via skin contact of infested pigs.

Geographic distribution: World-wide

Symptoms: Infested pigs are restless, and continually scratch and rub. The annoyance caused by *S. suis* distracts pigs from feeding, resulting in reduced weight gain.

Significance: Sarcoptic mange is of great economic importance wherever pigs are raised.

Diagnosis: Restlessness, continuous scratching and rubbing. Crusts and barks on the skin, especially on the inside of the ears, which are inflamed and scabby. Microscopical investigation of skin scrapings, taken from the alterated skin reveals the typical mites.

Therapy: Infested animals can be washed with an acaricide or treated systemically with ivermectin (300 µg/kg, sc., 2 × at 28 days interval). It is recommended to treat all the animals of a herd at the same time. The following acaricides are effective against Sarcoptic mange: Organophosphates (coumaphos, crotoxyphos + dichlorvos, malathion, trichlorfon, diazinon, phosmet and phoxin), chlorinated hydrocarbons (lindane), permethrin and pyrethrin. Infested animals first must be washed and then treated (spray) thoroughly twice at 3-week-intervals. Washing with acaricides results in a reduction of skin lesions and symptoms but animals generally remain infested. Treatment with ivermectin seems to kill all the mites and animals are free of infestation as long as no infected pig is introduced to the treated herd.

Prophylaxis: Infested pigs should not be introduced to the herd to avoid reinfection. Elimination of predisposing factors (e.g. malnutrition, intestinal parasites and other chronic infections).

(Figures 216, 217, 220, 221, 600, 601, 602, 603)

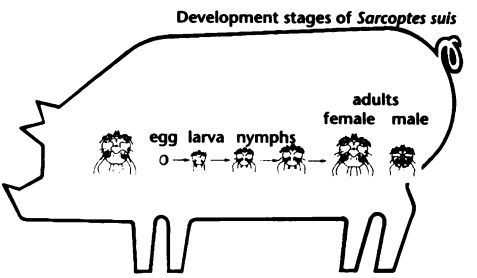

Fig. 600 Life cycle of *Sarcoptes suis* [10]

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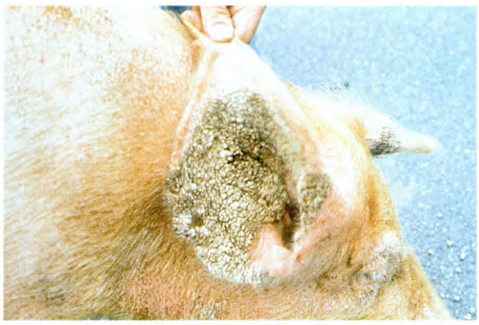

Fig. 601 Chronic *Sarcoptes suis* infestation results in thickened skin and crusts in the ears

Fig. 602 Crusts and barks on the skin of a pig due to Sarcoptic mange

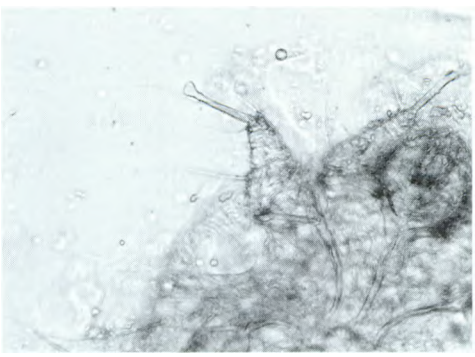

Fig. 603 Details of the *Sarcoptes suis* mite; tarsal suckers have long, unjointed pedicels

Demodex suis Follicle mite

Location: Within the hair follicles and the sebaceous glands

Hosts: Pig

Species description: The follicle mite is about 0.25 mm long, with a cigar-shaped body and 8 stubby legs. Several *Demodex* species which are morphologically identical occur on many other host species. *Demodex* spp. are regarded as distinct species and are host-specific. The entire life cycle is spent on the host and completed in about 3 weeks.

Geographic distribution: World-wide

Symptoms: Infested pigs scratch and rub, and large pustules may occur. Loss of hair, thickened and wrinkled skin may be seen.

Significance: Follicle mite in pigs cause great irritation which may develop to large abscesses. The exact prevalence of follicle mite infestations is unknown.

Diagnosis: Mites may be demonstrated by microscopic examination of deep skin scrapings.

Therapy: The same treatment may be applied to *Demodex* infestations as to *Sarcoptes suis*, although available treatments are less effective for *Demodex* infestations.

Prophylaxis: There is no effective prophylaxis because inapparent carriers may transmit the infestation.

- **Insecta found on the skin**

– **Lice**

ANOPLURA

Haematopinus suis Swine louse

Location: Lice may be found all over the body surface

Hosts: Pig

Species description: The swine louse is easily seen with the naked eye. It is 5–6 mm long. It has a long narrow head, long mouthparts adapted for sucking blood, and large claws on each of the six legs. *H. suis* is the

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only louse found on pigs. The pig louse spends its entire life cycle on the pig. Eggs are glued to the hairs and the development is completed in 2–3 weeks. Lice cannot survive off the host. Heavy infestations may cause severe anaemia, discomfort and annoyance. Lice are blood suckers. Self trauma of the skin as a result of the excessive scratching results in excoriation, thickening and soreness of the skin. Infested pigs are more susceptible to other diseases.

Geographic distribution: World-wide

Symptoms: Infested animals rub and scratch vigorously on any convenient object. They have a reduced weight gain. Anaemia may occur in heavy infestations.

Significance: Louse infestation is one of the most important diseases in pigs. *H. suis* transmits the swine pox virus, *Eperythrozoon suis* (☞ SWINE, ■ 2) and probably swine fever.

Diagnosis: Presence of these large insects on pigs

Therapy: A number of insecticides is recommended for use as sprays to control swine lice. Thorough applications are necessary and usually 1–2 l/mature animal are required. The following insecticides are effective against the pig louse: organophosphates (coumaphos, crotoxyphos + dichlorvos, malathion, stirofos, trichlorfon, diazinon, phosmet and phoxin), chlorinated hydrocarbons (lindane, methoxychlor) and permethrin. Dips, overall body treatment (dust) or spraying are the possible methods of treating infected animals. Fenthion (3%) pour-on is a ready-to-use formulation for lice control. Insecticidal treatment should be repeated 1 week after the initial treatment since eggs may survive. Ivermectin (300µg/kg, sc.) provides an effective lice control.

Prophylaxis: Regular treatment is required. In particular, sows should be treated prior to farrowing to prevent lice from moving onto the piglets.

(Figures 604, 605)

Fig. 604 *Haematopinus suis*; the swine louse (5–6 mm long) [4]

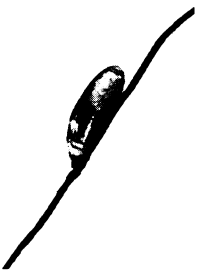

Fig. 605 *Haematopinus suis*; louse egg attached to the hair

– Fleas

SIPHONAPTERA

Remarks: Fleas are not host-specific and may attack any convenient mammal or bird for a blood meal. The two fleas most commonly found on swine are *Pulex irritans*, the human flea and *Echidnophaga gallinacea*, the stickfast flea. *Ctenocephalides felis*, the cat flea, may occasionally be found on young piglets. *Tunga penetrans*, the jigger or chigger flea occurs in Africa.

Fleas are wingless insects 2–4 mm long, have a thick brown chitinous exoskeleton and powerful legs. Female fleas lay eggs which drop off the host into the animal bedding. Larvae hatch in 2–16 days and feed on organic material such as dry blood, faeces, etc. Larvae mature in 1–2 weeks and pass through a pupal stage of varying length, depending on environmental conditions. Only the adult flea is parasitic and requires periodic blood meals. Fleas can survive for many months (*Pulex irritans* up to 2 years) in the absence of a host.

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Symptoms: Restlessness, stampeding when masses attack animals. Allergic dermatitis may be seen, but should be differentiated from other similar conditions (e.g. sarcoptic mange).

Significance: Fleas are generally of minor significance in pig production, but when masses of fleas occur in sheds they may severely irritate and debilitate pigs. An allergic dermatitis, similar to that of dogs may occur in pigs, and its signs resemble those of sarcoptic mange. Fleas are also capable of transmitting Classical Swine Fever. Masses of *Tunga penetrans* were found to produce agalactia in sows (in Zaire) and death of piglets. Ovigerous females (chiggers) were localized in the teats and obstructed the ducts.

Diagnosis: It is not easy, as adult fleas may leave the host and eggs and larvae are difficult to find. The bites are similar to those of mosquitoes, lice and mites.

Therapy and Prophylaxis: These are based on locating the flea-breeding area. Both the surrounding and the animals should be sprayed with an insecticide (organophosphates, chlorinated hydrocarbons or pyrethrinoids). Infested litter, bedding and manure should be burned and removed from the pig quarters. Housing can be sprayed with malathion (2.5%) and the pigs dusted with rotenone (1%) powder, lindane (1%) or malathion (6%) powder. Insecticidal sprays combined with growth regulators (e.g. deflubenzuron, fenoxycarb, methoprene) eliminate the adult fleas and interrupt larval development of fleas for several months.

(Figures 606, 607, 608, 609, 610)

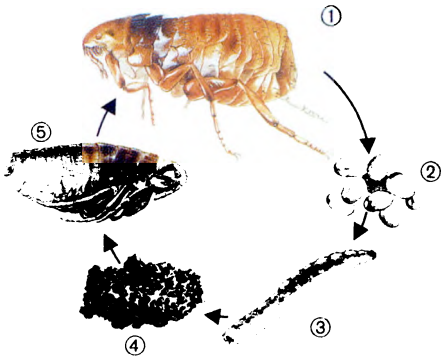

Fig. 607 Life cycle of fleas
Adult females (1) deposit around 100 eggs (2) after a blood meal, 4–12 days later spiny larvae develop (3) which develop in a pupal stage (4) from which adult fleas (5) hatch after a period varying from 3 weeks to several months [52]

Fig. 608 *Tunga penetrans*; the jigger flea (1 mm long) [4]

Fig. 609 *Ctenocephalides felis*; the cat flea (male: 2–2.5 mm long, female: 2–3.2 mm long) [52]

Fig. 606 *Pulex irritans*; the human flea (male: 2–2.5 mm long, female: 2–3.3 mm long) [52]

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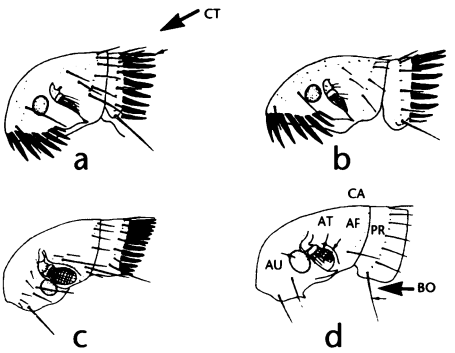

Fig. 610 Heads of some flea species which may affect domestic animals including pigs: (a) *Ctenocephalides canis*, (b) *C. felis*, (c) *Ceratophyllus gallinae* and (d) *Pulex irritans*; CT = ctenidia, AU = ommatidium, PR = pronotum, AT = antennae and CA = caput (head) [33]

– Dipterida

CULICIDAE Mosquitoes

***Aedes* spp., *Anopheles* spp., *Culex* spp. and others**

Remarks: Mosquitoes may also attack livestock, causing discomfort and irritation. Massive attacks cause skin lesions in form of raised, oedematous swellings (a few mm in diameter) on the legs and abdomen which tend to disappear within 1–2 days but this can make pigs unacceptable for marketing at that time. Control within the piggery is possible by using diazinon aerosol spray in the late evening as a regular procedure. Mosquito screening and insect repellants are also helpful in minimizing mosquito troubles in piggeries. Where possible, the breeding ground of the mosquitoes should be eliminated (e.g. draining water bodies in the surroundings, covering the surface, etc.) (☞ CATTLE, ■ 5.1.). (Figure 611)

Fig. 611 *Aedes* sp. in feeding position (5–6 mm long)

Flies are of importance in pig production world-wide. They may annoy animals by their painful bite while others transmit diseases. The larvae of some species invade pre-existing skin wounds and cause myiasis which may cause severe losses.

SIMULIIDAE

***Simulium* spp.** Black flies, midges or buffalo gnats

Remarks: This small fly occurs world-wide and is found in swarms near free-running well-aerated streams. Simulids cause severe irritation to livestock, including pigs when they occur in large numbers. Bites are inflicted on all parts of the body, giving rise to vesicles which burst exposing the underlying flesh. Skin wounds caused by simulids heal very slowly. Certain areas of the tropics are rendered uninhabitable by simulids (☞ CATTLE, ■ 5.1). (Figure 612)

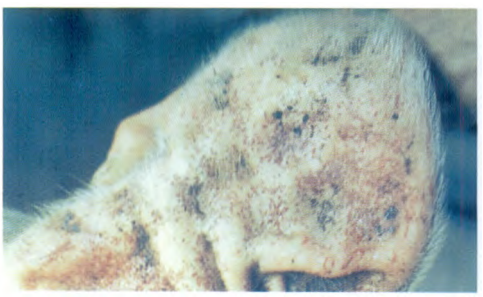

Fig. 612 Massive skin irritation on the ears of a pig due to *Simulium* sp. bites [30]

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TABANIDAE Horse flies

Remarks: These robust flies have powerful wings and breed on leaves of plants in the vicinity of water. They attack pigs and other large animals by biting in different places and then taking blood, frequently on the back, head, ears and the udder of sows. They are capable of transmitting infectious diseases, including hog cholera (☞ CATTLE, ■ 5.1).

MUSCIDAE

Musca domestica The common house fly

Remarks: Apart from being attracted to organic material this non-biting muscid fly may also be attracted to wounds and other moist parts of the body. *M domestica* is known to transfer pathogenic bacteria mechanically from one wound to another. It is also capable to spread hog cholera, eggs of nematodes and cestodes, stages of protozoa and several viruses. The preferred breeding place is in the faeces of animals or in decaying organic matter (☞ CATTLE, ■ 5.1).

Stomoxys calcitrans The stable fly

Remarks: It is a biting muscid and occurs world-wide. Both sexes of this fly are bloodsuckers and can become extremely irritating pests of man and domestic animals, including pigs. Its preferred breeding ground is in decaying vegetable matter or in moist straw or hay. This fly prefers strong light rather than dark stables. It can be a carrier of hog cholera and possibly other infectious pathogens (☞ CATTLE, ■ 5.1).

GLOSSINIDAE

Glossina **spp.** Tsetse flies

Remarks: Both sexes suck blood and are equally capable or transmitting trypanosomes. Tsetse flies, especially in great numbers can severely annoy and debilitate pig populations (☞ CATTLE, ■ 5.1).

CALLIPHORIDAE The blow flies and their allies

Callitroga (syn. *Cochliomyia*) ***hominivorax*** The "American screwworm"

Remarks: *C. hominivorax* is a primary blowfly and has recently been found to occur also in North Africa. Pigs and many other domestic animals may be affected. The larvae of the screwworm fly (maggots) penetrate into pre-existing wounds and extend the lesions considerably (☞ CATTLE, ■ 5.1). The life cycle differs from other flies in that breeding takes place in the wounds of the live animal and not in carcasses.

Chrysomya bezziana: The cattle screwworm in Africa and Asia

Remarks: Cattle are commonly infested (☞ CATTLE, ■ 5.1)) but also horses, sheep, dogs, sometimes man and rarely pigs. *C. bezziana* is also a primary blowfly.

Calliphora **spp.**

Remarks: Blowflies of the subfamily *Calliphorinae* differ from screwworm flies in that they are secondary blowflies which deposit their eggs in necrotic wounds which have been previously struck by screwworms (primary blowflies).

The disease condition commonly referred to as "myiasis"

Most cases of myiasis occur in rainy weather. Maggots penetrate the wound tissue, which they liquefy and thus extend the lesions. There is a foul-smelling exudate and death from screwworm infections is frequent.

Prophylaxis: To prevent myiasis, surgical wounds should be avoided during the fly season (e.g. rainy season, summer) and attention should be given to skin wounds

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and injuries. Wounds can be protected from myiasis by the application of a wound dressing containing an insecticide (e.g. organophosphates such as coumaphos or chlorinated hydrocarbons such as lindane) and an antiseptic. After wounds are invaded by larvae they may effectively be treated with pressurized aerosols containing coumaphos, lindane or ronnel (korlan). These are very effective in killing the larvae in wounds but they will not protect the wounds from re-invasion.

- **Control of Flies**

Fly control in piggeries is a continuing exercise during the months of fly activity (summer, rainy season). Breeding of flies can be prevented if dung is removed regularly or liquefied. Manure should be removed at least weekly (complete life cycle of the housefly takes 15 days) and spread thinly on soil so that fly eggs and larvae are killed by desiccation. Destruction of flies within pig sheds can be carried out in a number of ways. Insecticides are applied in form of sprays, baits or impregnated strips. Malathion, trichlorfon, tetrachlorvinphos can be used to spray buildings (ceiling, wall spaces, etc.). Feed or water should be covered for spraying to avoid contamination. Space or aerosol sprays (foggers) used daily with knockdown insecticides are also effective and less prone to resistance development than are long-term acting pyrethroids. Baits applied to clean concrete surfaces and pen divisions are effective and usually contain insecticides such as ronnel, diazinon, malathion, trichlorfon and dichlorvos. Fly electrocutors and adhesive papers offer a non-chemical method of controlling flies. Screen doors reduce the number of flies entering the buildings where livestock is kept. Insect growth regulators (deflubenzuron, cyromazine and others) may be used as larvicides.

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Parasites of Poultry

CONTENTS

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PROTOZOA

Histomonas meleagridis

Infectious enterohepatitis or "Blackhead"

Location: Caecum and liver

Hosts: Turkey, less frequently chicken and guineafowl

Species description: *H. meleagridis* is the cause of enterohepatitis or "blackhead" in turkeys but also in chicken, guineafowl and other gallinaceous birds. Chickens appear to act as reservoirs of the parasite for the main susceptible host, the turkey. The caecal worm *Heterakis gallinarum* and its eggs can carry *H. meleagridis*. Turkeys may therefore acquire *H. meleagridis* by ingestion of infected *Heterakis* eggs. Blackhead is not transmitted through the egg of the turkey.

Geographic distribution: World-wide; in Africa: East, North, parts of West and Central Africa

Symptoms: Histomonosis is mainly a disease of young turkeys. The chicken is more resistant to the infection, although it may occasionally also become infected. Symptoms are weakness, inappetence, listlessness, standing for long periods in a dejected attitude with eyes closed, dropped and ruffled feathers. Especially characteristic is the appearance of sulphur-yellow faeces. Birds become progressively weaker and death occurs within a week of first symptoms. The term "blackhead" is derived from the cyanotic (blue-violet) appearance of the comb and the wattles but it is not a constant or pathognomonic feature of *H. meleagridis* infection. The caeca and liver show marked lesions. The caeca are enlarged, thickened, intensively inflamed and the mucosa is replaced by areas of necrosis. Casts may occupy the lumen of both caeca. The liver is enlarged and shows necrotic areas (0.5–2 cm in diameter), dark brown in colour often with a yellowish perimeter. The surface of necrotic areas is almost always depressed. The caecal lesions may be confused with those due to *Eimeria tenella* but this infection is accompanied by characteristic stages of the parasite in the casts.

Significance: The disease is known to affect primarily turkeys. However, it also affects chickens around 12 weeks of age. The disease in chickens involves mainly the caeca. Mild liver lesions occur occasionally. Mortality in chickens is low but the morbidity can be high in young animals.

Diagnosis: This is often based on necropsy findings. Necrotic lesions of the liver at postmortem are pathognomonic. Detection of the parasites is difficult. The organisms are polymorphic and the morphology depends on the organ location and the stage of the disease. Histological examination of stained sections of the liver shows necrosis and colonies of the organisms. (In a few cases confirmation may be obtained by demonstration of *H. meleagridis* from direct smears of the affected liver or caeca contents).

Therapy: Dimetridazole at 0.0125% (100–200 ppm) in the feed or in the drinking water at 0.63 g/l and for prevention 0.315 g/l. Aminonitrothiazole at 0.5% is curative or at 0.01% preventive in the feed. It should be administered for 14 days.

Ronidazole at 0.0045–0.006% (60–90 ppm) in water given for 3 days prevents mortality.

Ipronidazole (50–85 ppm) is highly effective. For short-term therapy 0.025% in the

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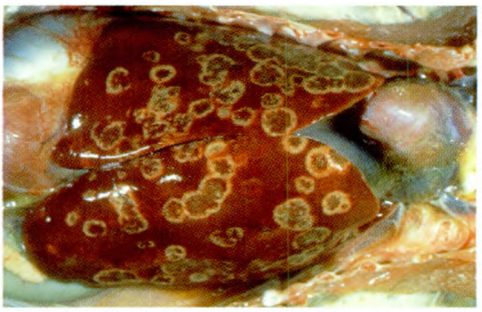

Fig. 613 *Histomonas meleagridis*; typical necrotic lesions in the liver of a turkey [53]

Fig. 614 *Histomonas meleagridis*; necrotic lesions in the caeca of a turkey. Casts occupy parts of the caecal lumen [53]

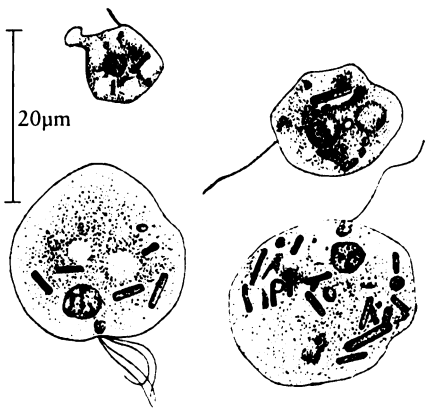

Fig. 615 Various forms of *Histomonas meleagridis* (schematic). The organism is pleomorphic, the morphology depends on the organ location, stage of infection and probably other factors [3]

feed or 0.0125% in the drinking water. For prevention continuous administration at the dosage of 0.00625% in the feed is suggested.

Prophylaxis: It is essential to avoid contact between turkeys and domestic chicken and where the two are kept on the same farm a separate area should be allocated to each of them. Regular treatment with anthelmintics to reduce *Heterakis gallinarum* infections is of benefit to reduce the overall incidence of histomonosis. Continuous administration of the above-mentioned drugs in prophylactic dosages may prevent outbreaks in populations at risk.

Remarks: *Parahistomonas wenrichi* is a non-pathogenic species resembling *H. meleagridis* and occurs in the liver. The most important difference is the size: *P. wenrichi* is about 1.5 times as large as *H. meleagridis* and has four flagella instead of one or two in the latter species.

(Figures 613, 614, 615)

Trichomonas gallinae

Location: Upper digestive tract (crop, oesophagus, pharynx, proventriculus) and liver

Hosts: Pigeon, turkey and chicken

Species description: *T. gallinae* is the cause of avian trichomonosis found particularly in pigeons but also in turkeys and chickens. It is basically a disease of young birds with inflammation and ulceration in the oesophagus, crop and proventriculus. Contaminated drinking water is the main source of infection. The incubation period is 3–14 days.

Geographic distribution: World-wide; in the Central African Republic, Zaire and southern Africa

Symptoms: Drowsiness, a pendulous crop and a foul odour from the mouth are usually observed in infected chickens. Primary lesions in the oropharyngeal mucosa, followed by parasite invasion into the pharyngeal glands and progressive penetration of the underlying tissue. Infection of the liver with multiple abscesses is considered as an important cause of death in infected birds.

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In pigeons suffocation and impaired feeding due to the massive lesions is often found to be an important cause of death.

Significance: *T. gallinae* causes a condition in chicken termed "canker". Turkeys, chicken and a wide variety of wild birds are parasitized with varying degrees of pathogenicity.

Diagnosis: At necropsy, yellowish necrotic lesions are evident in the mouth, crop and oesophagus with extension to the bones of the skull, the liver and elsewhere. Parasites are usually very numerous in the mouth and crop contents. A smear from crop or oesophagus lesions will usually demonstrate the parasite. This material or the carcass should be fresh for a successful identification of the trichomonads.

Therapy: In chickens treatment is generally not indicated. In pigeons metronidazole (60 mg/kg, po.) and dimetridazole (50 mg/kg, po.) in the feed or in drinking water (0.05%, during 5–6 days) may be used to suppress the disease.

Prophylaxis: Wild pigeons and other birds play an important role in introducing the infection to domestic birds. The drinking places should therefore be shielded to prevent the introduction of infection.

(Figures 616, 617, 618)

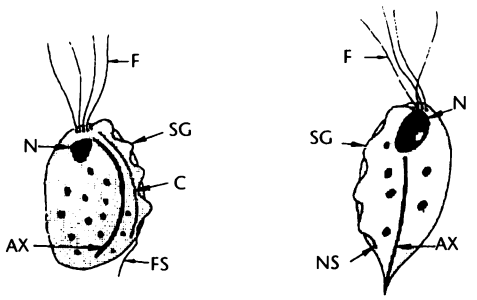

Fig. 616 *Trichomonas* species affecting birds; *T. gallinarum* (a), *T. gallinae* (b); AX = axostyl, C = costa, SG = recurrent flagellum, FS = free end of SG and F = flagellum [33]

Fig. 617 *Trichomonas gallinae*; caseous necrotic material in the crop of a young pigeon [4]

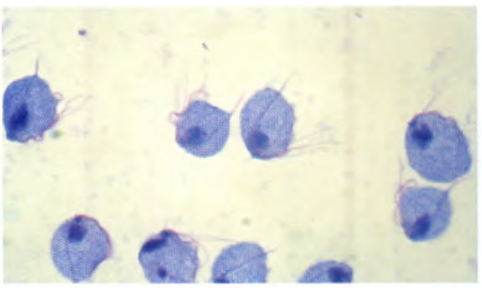

Fig. 618 *Trichomonas gallinae* (6–18 µm); stained [15]

Tetratrichomonas gallinarum
(syn. *Trichomonas gallinarum*)

Location: Lower digestive tract, particularly caecum and colon and sometimes liver

Hosts: Chicken, turkey, guineafowl and possibly other gallinaceous birds

Species description: *T. gallinarum* infections should be differentiated from the more pathogenic *Histomonas meleagridis*. Infection is acquired by ingestion of trichomonads in contaminated feed or water.

Geographic distribution: World-wide, the same as *T. gallinae*

Symptoms: Enterohepatitis with liver lesions resembling those due to histomonosis.

Significance: This is a common parasite which probably cannot cause disease by itself, although infected birds have been reported to show blackhead-like lesions of the Caecum and liver. Often a watery, slimy inflammation of the caecum is observed.

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Diagnosis: This is based on the demonstration of the organisms (up to 8 µm) in smears of the large intestinal mucosa.
Therapy: ☞ *T. gallinae*
Prophylaxis: ☞ *T. gallinae*

- **Coccidiosis in Chicken**

Coccidiosis in chicken is, following bacterial diseases, the second largest problem worldwide, wherever poultry is reared in crowded conditions. The following characteristics of Coccidia may be responsible for their great significance: short life cycle (5–7 days); no intermediate host is required and *Eimeria* spp. have a very high reproductive capacity.

In chickens six different species are important:

Eimeria tenella

It is very pathogenic, causes bloody caecal coccidiosis and often mortality.

Eimeria necatrix

It is very pathogenic, causes bloody intestinal coccidiosis and often mortality.

Eimeria maxima

It can be responsible for high morbidity and sometimes high mortality. It often causes subclinical coccidiosis associated with marked weight loss.

Eimeria acervulina

It is even less pathogenic than maxima, but reduces weight gain and feed conversion and impairs absorption of nutrients and pigments (subclinical coccidiosis).

Eimeria mitis

It is difficult to diagnose; in case of heavy infections, symptoms like *E. acervulina*.

Eimeria brunetti

It may be very pathogenic, causes weight loss and mortality. Difficult to diagnose because of the indistinct lesions in the lower intestinal tract. Sometimes necrosis in the lower small intestine and the large intestine.

- **General features of *Eimeria* spp. in chicken**

Species description: The life cycle of all *Eimeria* spp. involves two or more generations of an asexual development known as schizogony, followed by a sexual phase termed gametogony which results in the formation of oocysts. Oocysts are excreted in the faeces and become infective in the litter only after sporulation during which sporozoites are produced. Some *Eimeria* spp. cause clinical coccidiosis (e.g. *E. tenella*, *E. necatrix*), associated with bloody diarrhoea, weakness and death while other species cause subclinical coccidiosis (e.g. *E. maxima*, *E. acervulina*). The overall losses due to subclinical infections are enormous and result in markedly reduced production (growth and egg production). Immunosuppressive diseases may act in concert with coccidiosis to produce a more severe disease. Marek's disease may interfere with immunity to coccidiosis, and infectious bursal disease may exacerbate coccidiosis, placing a heavier burden on anticoccidial drugs.
Geographic distribution: World-wide
Diagnosis: This is based on both clinical observations and laboratory investigations. An outbreak of coccidiosis is often associated with the following signs: signs of illness in the flock, abnormal faeces, birds that have a moribund appearance with lethargy, ruffled feathers and loss of skin pigmentations. Coccidiosis may be followed by diarrhoea which may be bloody in case of *E. tenella* and *E. necatrix* or contain mucus in case of *E. acervulina* or *E. maxima*. Oocyst counting may contribute additional infor-

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mation about the overall herd situation but this cannot be used alone to identify outbreaks or species involved in outbreaks. Coccidiosis can best be diagnosed from birds killed for immediate necropsy. Many stages of Coccidia can be seen in smears taken from suspected intestinal lesions. Mucosal scrapings may be diluted with saline on a slide, covered with a coverslip and then examined with a microscope. Oocysts or macrogametes are most easily seen, but in many cases the lesions are caused by maturing schizonts. The presence of clusters of large schizonts in the midgut area is pathognomonic for *E. necatrix*, while a similar finding in the caecum indicates *E. tenella*.

In warm, moist areas the development of Coccidia is even more facilitated. Coccidiosis in chickens is exclusively represented by species of the genus *Eimeria*. For simplicity reasons the *Eimeria* infections found in domestic fowls are distinguished as 1) those causing marked haemorrhage in the intestinal tract and 2) those without marked haemorrhagic lesions.

(Figures 619, 620, 621)

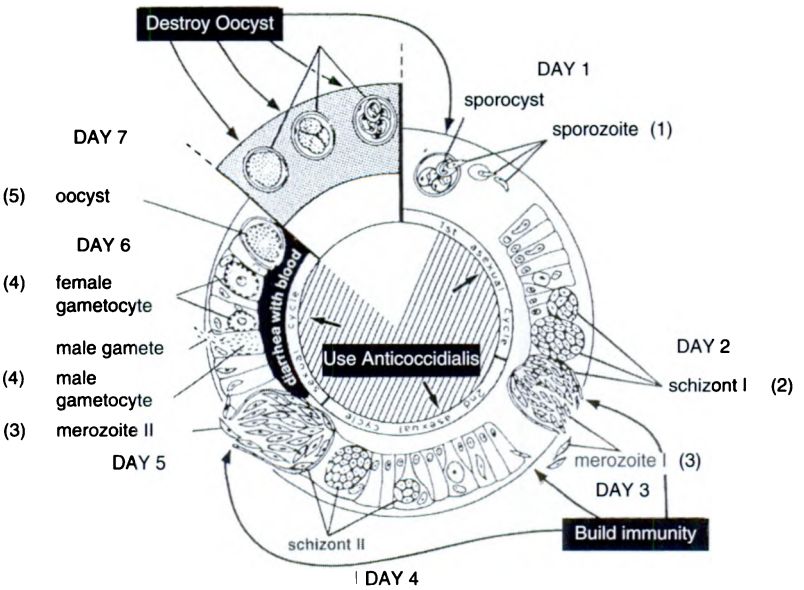

Fig. 619 Coccidia have a very definite pattern of development. There are two or more generations of an asexual development known as schizogony, followed by a sexual phase (gametogony) which results in the formation of oocysts. Oocysts are excreted in the faeces and become infective in the litter only after sporulation during which sporozoites are produced [54]

Development stages of coccidia: (1) sporozoites (motile infective stages), (2) schizonts (multiplying asexual reproductive stage), (3) merozoites (lst to 3rd-generation motile infective stage), (4) gametocytes (sexual reproductive stages, female non-motile macrogametocyte and male motile microgametocyte) and (5) oocysts (zygotes formed by the sexual union of gametocytes)

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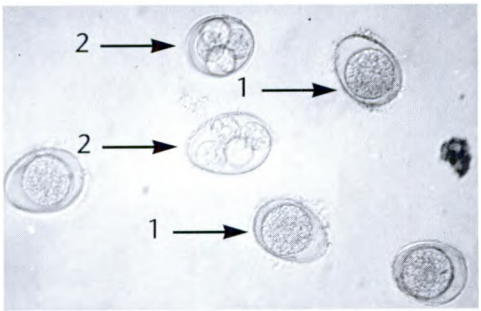


Fig. 620 Unsporulated (1) and sporulated (2) oocysts of *Eimeria maxima* (30 × 20 µm) [15]

Differential Characteristics for 9 Species of Chicken Coccidia

Diagnostic Characteristics in Red

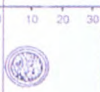
	Characteristics	E. acervulina	E. brunetti	E. maxima	E. mitis	E. mivati	E. necatrix	E. praecox	E. tenella	E. hagani (Species of Doubtful Validity)
Macroscopic Lesions	Zone Parasitized									
	Macroscopic Lesions	**light infection:** whitish round lesions sometimes in ladder-like streaks **heavy infection:** plaques coalescing, thickened intestinal wall	coagulation necrosis mucoid, bloody enteritis in lower intestine	thickened walls, mucoid-tinged exudate, petechiae	no discrete lesions in intestine, mucoid exudate	**light infection:** rounded plaques of oocysts **heavy infection:** thickened walls, coalescing plaques	ballooning, white spots (schizonts), petechiae, mucoid blood-filled exudate	no lesions mucoid exudate	**onset:** hemorrhages into lumen **later:** thickening, whitish mucosa, cores of clotted blood	pinhead hemorrhages petechiae
Microscopic Characteristics	Millimicrons Oocysts Redrawn from Originals	10 20 30 	10 20 30 	10 20 30 	10 20 30 	10 20 30 	10 20 30 	10 20 30 	10 20 30 	10 20 30
	Length x Width (µ) Length = Width =	AV = 18.3 x 14.6 17.7 – 20.2 13.7 – 16.3	24.6 x 18.8 20.7 – 30.3 18.1 – 24.2	30.5 x 20.7 21.5 – 42.5 16.5 – 29.8	15.6 x 14.2 11.7 – 18.7 11.0 – 18.0	15.6 x 13.4 11.1 – 19.9 10.5 – 16.2	20.4 x 17.2 13.2 – 22.7 11.3 – 18.3	21.3 x 17.1 19.8 – 24.7 15.7 – 19.8	22.0 x 19.0 19.5 – 26.0 16.5 – 22.8	19.1 x 17.6 15.8 – 20.9 14.3 – 19.5
	Oocyst Shape and Index-Length/Width	ovoid 1.25	ovoid 1.31	subspherical 1.09	ellipsoid to broadly ovoid 1.16	oblong ovoid 1.19	ovoidal 1.24	ovoid 1.16	broadly ovoid 1.08	ovoid 1.25
	Schizont, max in Microns	10.3	30.0	9.4	15.1	17.3	65.9	20.0	54.0	
	Parasite Location in Tissue Sections	epithelial	2nd generation schizonts subepithelial	gamatocytes subepithelial	epithelial	epithelial	2nd generation schizonts subepithelial	epithelial	2nd generation schizonts subepithelial	epithelial
Life Hist. Chracter.	Minimum Prepatent Period-HR	97	120	121	93	93	138	83	115	99
	Sporulation Time Minimum (HR)	17	18	30	15	12	18	12	18	18

Fig. 621 Differential characteristics for nine *Eimeria* species of chicken [54]

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- **Coccidiosis with marked haemorrhage**

Eimeria tenella

Location: Caecum

Significance: *E. tenella* is highly pathogenic. This acute infection occurs most commonly in young chicks. Heavy infections are characterized by the presence of blood in the droppings and by high morbidity and mortality.

Diagnosis: At postmortem in the acute phase, the caecum are distended by blood following erosion of the mucosa. Large second-generation schizonts and free merozoites can be detected in smears from the caecum mucosa. Caecal cores are composed of necrotic debris, gametocytes and oocysts may be found during the recovery period of the host. Acute deaths without the presence of oocysts may occur.

(Figures 622, 623, 624)

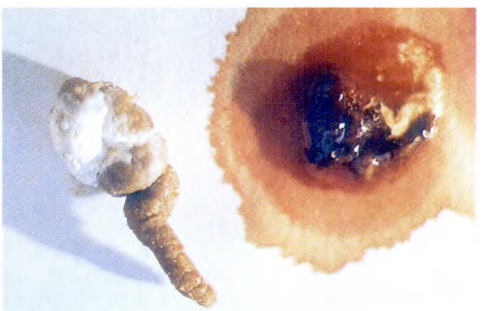

Fig. 622 Bloody faeces 4–6 days after infection with *Eimeria tenella* (right) and normal faeces (left) [54]

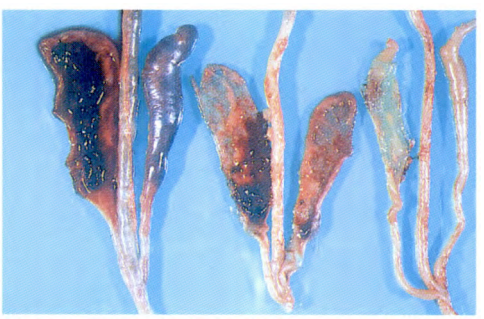

Fig. 623 Caeca of chicken, distended by blood due to *Eimeria tenella* infection (left and middle) and normal caecum (right) [53]

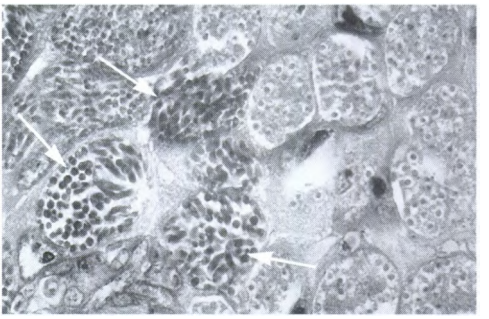

Fig. 624 Schizonts of *Eimeria tenella* in caecal mucosa (arrows) (60–70 µm in diameter) [54]

Eimeria necatrix

Location: Small intestine

Significance: *E. necatrix* is a highly pathogenic species which may cause heavy mortality. Lesions are confined to the small intestine where haemorrhage accompanies the maturation of the deeply penetrating schizonts.

Diagnosis: White spots (colonies of schizonts) are often visible in the intestinal wall. Oocysts occur in the caecum where macroscopic lesions are absent, an important distinction between this species and the *E. tenella*. It may be distinguished from *E. maxima* by the presence of very large schizonts in scrapings from the small intestine.

(Figure 625, 626)

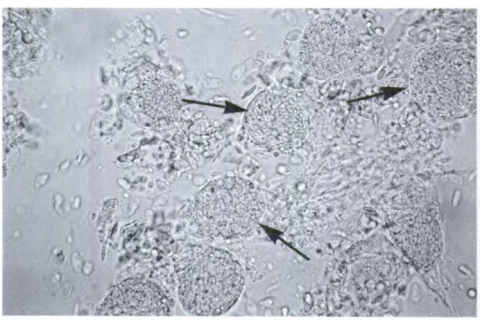

Fig. 625 Large schizonts (arrows) of *Eimeria necatrix* in the scraping of small intestinal mucosa (60–70 µm in diameter) [53]

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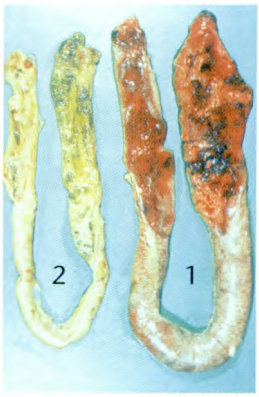

Fig. 626 *Eimeria necatrix* causes haemorrhage and necrosis in the mucosa of the small intestine (1); normal intestine (2) [53]

- **Coccidiosis without marked haemorrhage**

Eimeria maxima

Location: Mid-small intestine

Significance: *E. maxima* can be responsible for high morbidity and sometimes high mortality. *E. maxima* often causes subclinical coccidiosis associated with marked weight loss. Lesions occur most frequently in the mid-small intestine although the whole of the small intestine may be involved.

Fig. 627 Mucoid exudate and haemorrhage in the mid small intestine due to *Eimeria maxima* (left); normal intestine (right) [53]

Diagnosis: Typically the intestine appears thickened with pink mucoid exudate. Haemorrhage may be pronounced in heavy infections. Gametocytes or large yellowish oocysts may be seen in smears from the intestinal mucosa.

(Figure 627)

Eimeria brunetti

Location: Lower small intestine, caecum

Significance: The pathogenicity of this species is high. In heavy infections numerous small haemorrhages may occur in the mucosa with blood-stained exudates.

Diagnosis: This is difficult due to the indistinct lesions in the lower intestinal tract. Yellowish-white caseous material may be found in the lumen of the lower intestine and rectum. Rounded oocysts with a variable size occur throughout the intestine. Stained smears from the intestinal mucosa should be made to differentiate from *Clostridium perfringens* infections.

The following species are regarded as less pathogenic although they might cause weight loss in heavy or concomitant infections:

Eimeria mivati

Remarks: This species occurs in the upper small intestine. Heavy infections may produce mortalities of up to 10%. Light infections cause weight loss.

Eimeria acervulina

Remarks: This species occurs in the upper small intestine and is not very pathogenic but reduces weight gain and feed conversion and impairs the absorption of nutrients. It generally causes a subclinical coccidiosis.

(Figure 628, 629)

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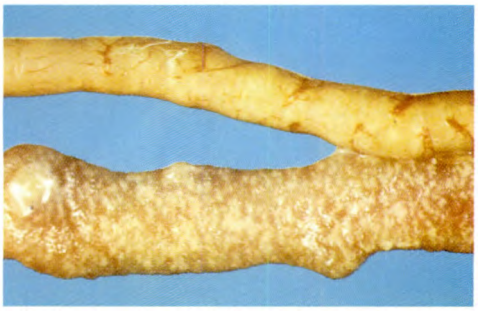

Fig. 628 *Eimeria acervulina* infection cause typical striation and thickening of the mucosa [55]

Fig. 629 Mucoid faeces due to *Eimeria acervulina*, *Eimeria mivati* and *E. maxima* [54]

Eimeria mitis

Remarks: This species is difficult to diagnose and heavy infections cause lesions similar to those of *E. acervulina*.

Eimeria praecox

Remarks: This species occurs in the upper third of the small intestine. It is of low pathogenicity.

- **Control of Coccidiosis**

In the past chemotherapy was used to treat outbreaks after signs of infection and losses were apparent. Soon the concept of preventive medication emerged with the realization that most of the damage is done once signs of coccidiosis occur. Today, almost all broiler flocks in intensive poultry systems receive preventive medication, and treatment is used as a last resort.

If layers are kept permanently in battery cages, the replacement flocks can be raised under permanent coccidiostatica application. If layers are kept in systems with direct contact to faeces, the coccidiostatica application to the replacement flocks should be stopped at the 11th–13th week of age, in order to achieve immunization. The contact to the contaminated faeces should then be maintained.

Therapy: Sulfachlorpyrazine (300 ppm in drinking water), sulfadimethoxine combined with ormetoprine (125 and 75 ppm in feed), sulfaquinoxaline combined with pyrimethamine (14 ppm and 45 ppm, in drinking water), nicarbazine (125 ppm in feed), amprolium (125 ppm in feed or via drinking water), amprolium combined with ethopabate (125 and 8 ppm via drinking water) or toltrazuril (25–75 ppm via drinking water during two consecutive days). Other combinations may also be used. Therapy should be continued until the symptoms have disappeared (3–6 days).

Prophylaxis: Anticoccidial drugs represent a wide variety of chemicals including one group of antibiotics (ionophores). The demand on control of coccidia in broilers, turkeys and replacement pullets is very different.

Different programs have been developed and are as follows:

Continuous feeding

A common program with 1 drug only, which is mainly used where coccidiosis one does not seem to be a major problem.

Shuttle programs

One drug is used in the starter feed (2–3 weeks) and another in the grower feed. Several shuttle programs using various drugs are practiced throughout the world. The drugs used should be complementary in spectrum of activity.

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Rotation programs

In these programs the same anticoccidial is used for several grow outs, and then replaced by another.

There are other programs which may also be useful in practice. General rules cannot be made for such a choice but it is necessary to change products every now and then. A wide variety of anticoccidials is currently available (Tables 23–25). Depending on the product used, the withdrawal periods and the indications should be strictly considered. Anticoccidials should not be administered to laying hens. Toltrazuril (7 mg/kg, po.) is a modern anticoccidial and not related to any other drug currently in use. It is effective against all the pathogenic *Eimeria* spp. of chicken and may be used for both therapy and prophylaxis of coccidiosis. Since toltrazuril does not interfere with the development of immunity it may be used for prevention of coccidiosis in replacement chickens. A strategic use every 3 to 4 weeks during rearing will provide reliable protection. Usually a 2-day treatment every 3 to 4 weeks up to 18 weeks of age will be sufficient. It is well established that outbreaks of coccidiosis are often related to stress (e.g. vaccination, debeaking, handling of birds, heat stress, change of diet). In all these cases a strategic administration of toltrazuril at these times will protect birds from coccidiosis.

Note: Even at recommended dosage anticoccidials demonstrate side effects such as depressed growth, toxicity in layers, interactions with minerals, immunosuppression. It is therefore essential to concentrate anticoccidial drugs correctly. Coccidial resistance has been reported with almost all commercially available drugs. Resistance of Coccidia against drugs may be expressed by decreasing weight gains, reduced utilization of nutrients and rather rarely by clinical coccidiosis and mortality.

(Tables 23, 24, 25)

Table 23 Therapeutic anticoccidial drugs, applied in drinking water

Generic names	Dosage (mg/kg body weight) in drinking water
Amprolium (1) + Ethopabate (2)	0,012-0,024% (1) + 0,0076-0,0152% (2) during 5-7 days
Diaverdin (1) + Sulfaquinoxaline (2)	0,00192% (1) + 0,00768% (2) during 3 days, 2 days break, and 3 days treatment again
Pyrimethamin (1) + Sulfaquinoxaline (2)	0,0015% (1) + 0,0045% (2) during 6 days or during 3 days, 2 days break, and 3 days treatment again
Sulfaclozin.natric.monohydrate	0,03% for 3 days, 2-3 days break, 3 days treatment or treatment on day 1.-3.-5.-7. and 9 or 1.-2.-5.-6. and 9.

Table 24 Combined prophylactic anticoccidial drugs for poultry

Generic names	Dosage (mg/kg) in feed
Amprolium (1) +	250-400(1) +
Ethopabate (2)= Acetaminoethoxy-methylbenzoat	20-32 (2)
Metichlorpindol (1), Methylbenzoquat (2)	100 (1) + 8,35 (2)
25% Aklomide (1) + 20% Sulfanitran (2)	250 (1) + 200 (2)
Amprolium (1) + Sulfaquinoxaline (2) + Ethopabate (3)	80 (1), + 60 (2) + 5(3)
Pancoccin (1) + Pyrimethamin (2)	145 (1) + 5 (2)
Sulfadimethoxine (1) + Ormetoprim (2)	125 (1) + 75 (2)
Bithionol (1) + Methiotriaziamin (2)	500 (1) + 100 (2)
Nitromide (1) + Sulfanitran (2) + Roxarsone (3)	250 (1) + 300 (2) + 50 (3)

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Table 25 Prophylactic anticoccidial drugs for poultry (applied in feed)

Generic names	Dosage (mg/kg) in feed
Aklomide (Chloro-Nitrobenzamid)	250
Amprolium (Amino-propyl-pyridinyl-methyl-picoloniumchlorid)	125-250
Arprinocid (9-(2-chloro-6-fluorobenzyl)-adenin)	70
Arsanilic acid or sodium-arsanilate	400
Beclotiamin (drug closly related to Amprolium)	40-125
Buqinolate (Alkyl-hydroxy-isobutoxy-chinolincarboxyl)	82,5
Clopidol; Meticlorpindol; Clopindol (Dichlor-dimethyl-pyridinol)	125-250
Decoquinate (Hydroxychinolin-derivate)	30
Diclazuril (2,6 dichloro-a (4-chlorophenyl)-4-(4,5-dihydro-3,5 dioxo-1,2,4-triazin-2 (3H)-yl)benzenacetonitril	1
Dinitrotolmid (Dinitro-ortho-toluamid)	40-250
Furazolidone (Nitrofuraldehyd-amino-oxazolidon)	55
Glycarbylamid (Imidazolcarboxamid)	30
Halofuginone (extract from *Dichroa febrifuga*)	3
Lasolocid (from *Streptomyces lasaloliensis*)	75-125
Madurimicin (from Actinomadura yumaensis)	5
Methylbenzoquat (Methyl-benzyloxy-butyl-dihydroxychinolin carboxylat	10-30
Monensin (from *Streptomyces cinnamonensis*)	100-120
Narasin (from *Streptomyces aureofaciens*)	60-70
Nicarbazin (Dinitrocarbanil-hydroxi-dimethyl-pyrimidin)	100-200
Nihydrazon (Nitrofuraldehyd-acetylhydrazon)	110
Nitrofurazone (Nitro-furfurylidin-semicarbazon)	55
Nitromide (Nitrobenzamid)	250
Nitrophenid (Dinitrophenyl-disulfid)	125
Robenidine (Chlorobenzyl-amino-guanidin)	33
Roxarsone (Hydroxy-nitrobenzen-arson-acid)	5
Salinomycin (from *Streptomyces albus*)	50-70

Cryptosporidium spp. (*C. baileyi* and *C. meleagridis*)

Location: Epithelial cells of respiratory and gastrointestinal tract and bursa. Predilection sites are caecum, posterior parts of colon, and bursa.

Hosts: *C. baileyi* occurs in chicken and *C. meleagridis* in fowls.

Species description: In chicken and turkeys *Cryptosporidium* spp. are primary pathogens that can produce respiratory and intestinal disease resulting in morbidity and mortality. The life cycle differs from that of *Eimeria* spp. in that the intracellular stages are confined to the microvillus region of the host cell and the oocysts which sporulate within the host cell are infective when released in the faeces. Another feature of *Cryptosporidium* spp. is the establishment of infections in the mucosal epithelium of a variety of organs. For example, *C. baileyi* can infect the cloaca, bursa of Fabricius, the upper and lower respiratory tracts and the eye lids.

Geographic distribution: World-wide

Symptoms: Disease associated with enteric infection is rare. By contrast, respiratory infections may produce a variety of clinical signs, depending on the particular sites involved. There may be inflammation of the air sacs, pneumonia, sinusitis or con-

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junctivitis with coughing, dyspnoea, nasal discharges and mortality.

Significance: Cryptosporidia are often found in healthy birds, although reduced weight gain and severe clinical signs were found following infection of the respiratory tract. An interaction between Cryptosporidia and viral infections (Marek, Turkey Viral Hepatitis) has repeatedly been reported in the sense that one infection may favour the other.

Diagnosis: This is done by demonstration of oocysts in the faeces (☞ CATTLE, 1) or by histological examination of infected tissue at necropsy. Smears of the tracheal mucosa may reveal the parasites after staining with carbol-fuchsin. An ELISA test may be used for epidemiological studies.

Therapy: There is no specific therapy. Cryptosporidia are unsusceptible to all tried anticoccidial agents.

Prophylaxis: This is based on strict disinfection of the chicken quarters. *Cryptosporidium* oocysts are very resistant to most of chemical agents, except ammonia, 10% formalin or hydrogen peroxide (☞ CATTLE, 1).

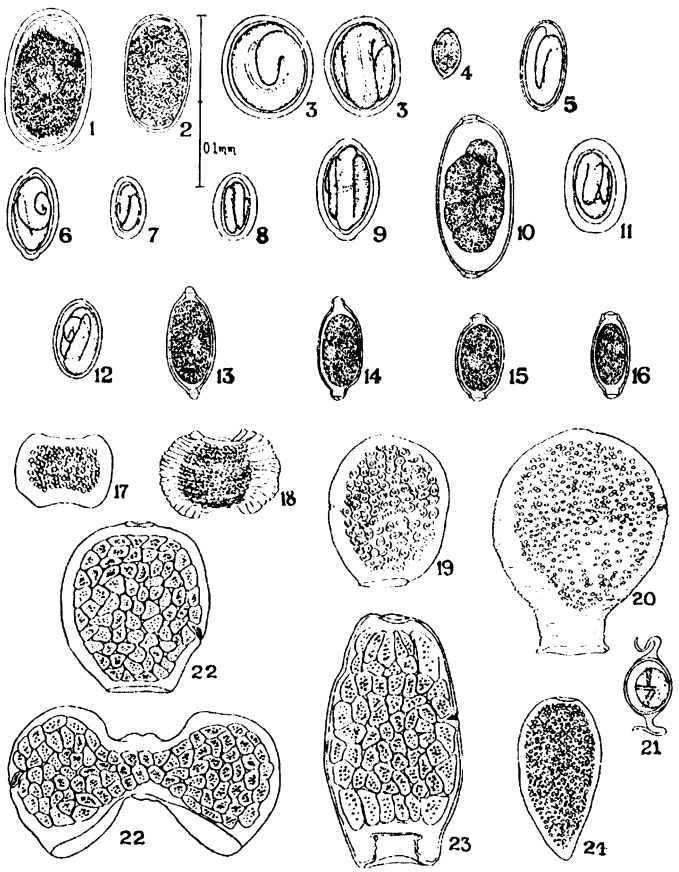

Fig. 630
Helminth eggs and segments of cestodes found in chicken [3]

1 *Ascaridia galli*
2 *Heterakis gallinarum*
3 *Subulura brumpti*
4 *Prosthogonimus* sp.
5 *Strongyloides avium*
6 *Tetrameres americana*
7 *Acuaria spiralis*
8 *Acuaria hamulosa*
9 *Gongylonema ingluvicola*
10 *Syngamus trachea*
11 *Hartertia gallinarum*
12 *Oxyspirura mansoni*
13 *Capillaria annulata*
14 *Capillaria retusa*
15 *Capillaria columbae*
16 *Capillaria longicollis*

Ripe segments of tapeworms (not drawn to scale)
17 *Amaebotaenia sphenoides*, 18 *Hymenolepis carioca*, 19 *Raillietina cesticillus*, 20 *Choanotaenia infundibulum*, 21 single egg of *C. infundibulum*, 22 *Raillietina echinobothrida* and 23 *R. tetragona*

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HELMINTHS

(Figure 630, ☞ p. 349)

- **Trematoda eggs found in the faeces and adult trematodes living in the gastrointestinal tract**

Echinostoma revolutum

Location: Rectum, caecum
Hosts: Duck, goose and other water birds and rarely chicken and turkey
Species description: The first intermediate host can be one of a variety of species of water snails. The second intermediate host can be the same or another water snail species or tadpoles of *Rana esculenta* and of other frogs.
Geographic distribution: World-wide
(Figure 631, 632)

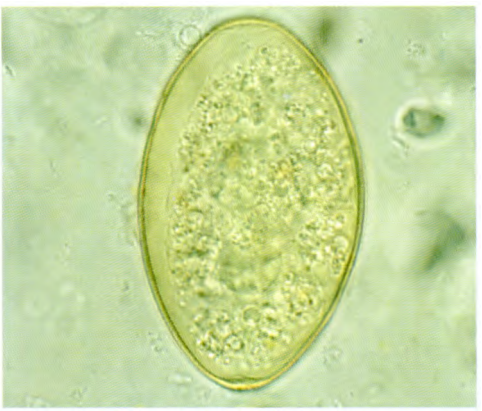

Fig. 631 Egg of *Echinostoma* sp. (97–126 × 60–70 µm) [4]

Fig. 632 *Echinostoma* sp., stained (10–22 × 2 mm) [4]

Echinoparyphium recurvatum

Location: Small intestine, especially duodenum
Hosts: Duck, goose, rarely chicken, pigeon and turkey
Species description: Two intermediate hosts similar to *Echinostoma revolutum*
Geographic distribution: World-wide, especially Asia, North Africa

Hypoderaeum conoideum

Location: Posterior part of the small intestine
Hosts: Duck, goose and other water birds, chicken, turkey and pigeon
Species description: Two intermediate hosts similar to *Echinostoma revolutum*
Geographic distribution: World-wide

Notocotylus attenuatus

Location: Caecum, rectum
Hosts: Chicken, duck, goose and wild aquatic birds
Species description: Intermediate hosts are water snails of the genera *Planorbis, Lymnaea* and *Bulinus.*
Geographic distribution: World-wide
(Figures 633, 634)

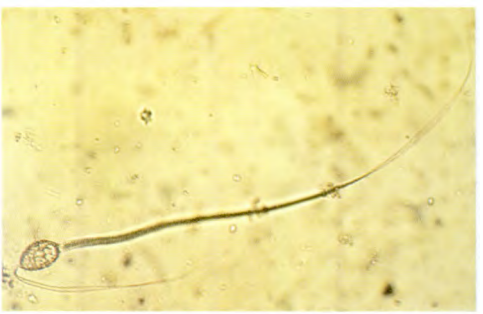

Fig. 633 Egg of *Notocotylus attenuatus* (20 × 10 µm) with two long filaments (up to 200 µm long) [11]

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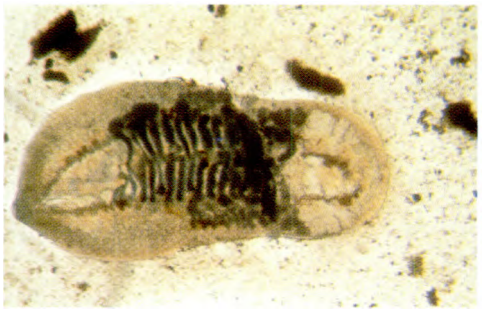

Fig. 634 *Notocotylus attenuatus* (2–5 × 0.6–1.4 mm) [53]

Catatropis verrucosa

Location: Caecum
Hosts: Chicken, duck, goose and wild aquatic birds
Species description: Intermediate hosts are water snails (Planorbis). Cercariae leave the snail and encyst on water plants which are ingested by the final host.
Geographic distribution: World-wide
(Figure 635)

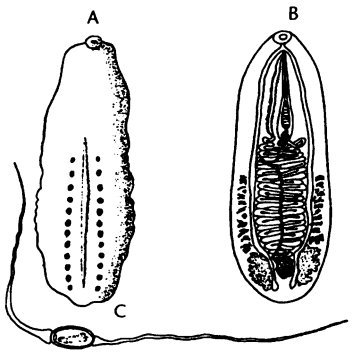

Fig. 635 *Catatropis verrucosa* (1–6 × 0.75–2 mm); ventral view (A), dorsal view (B); egg (18–28 µm long; (C) with two filaments (160–200 µm long) [9]

Postharmostomum commutatum
(syn. *P. gallinarum*)

Location: Caecum
Hosts: Chicken, pheasant, turkey, pigeon and guinea-fowl
Species description: First intermediate hosts are snails of the genera *Eulota, Subulina, Euhadra* and *Philomycus*. After liberation Cercariae may encyst in the same or other species of snails.
Geographic distribution: Southern Europe, Asia

- **General features of avian intestinal trematodes**

Symptoms: High numbers may cause marked irritation of the intestinal mucosa, enteritis, diarrhoea, emaciation. Secondary infections with other pathogens (e.g. *Eimeria* spp.) may cause high mortality rates.
Significance: Infections with trematodes may assume increased significance wherever birds have access to habitats where the snails live. Most of these trematodes are primarily parasites of aquatic birds. Chickens may also acquire infections when infected intermediate hosts are ingested. High mortalities may occasionally occur in young ducks, geese and chickens.
Diagnosis: This is based on the demonstration of trematode eggs in the faeces (sedimentation). Adult worms may be found at necropsy where a catarrhal, haemorrhagic enteritis may be seen in heavy infections.
Therapy: Niclosamide (60 mg/bird) is effective against Echinostomatidae. Niclosamid should not be used in geese. Fenbendazole (40 mg/kg, po.) was effective against *Echinostoma* spp. in geese. Praziquantel (20–25 mg/kg, po. during several days) and flubendazole (10–50 mg/kg, po. during 5 days) may be used in aquatic birds.
Prophylaxis: Chickens should be kept away from contaminated water bodies.

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- **Cestoda eggs and proglottids found in the faeces and adult cestodes living in the gastrointestinal tract**

Davainea proglottina

Location: Duodenum
Hosts: Chicken
Species description: This is a small tapeworm 1.5–4 mm long with only 4–7 whitish, transparent proglottids. The eggs are round and have embryonal hooks; they measure 55 × 36 µm. The suckers are armed with 3–6 rows of hooks. The intermediate hosts are various species of gastropod molluscs (e.g. *Limax*, *Arion*, *Cepaea*, *Agriolimax* spp.). The chicken acquires infection after ingestion of the infected mollusc. This microscopic tapeworm may be recognized in the duodenal mucosa by protrusion of the gravid proglottids above the villi if the open intestine is floated in water.
Significance: This species is highly pathogenic.
(Figures 636, 637, 638, 639, 640)

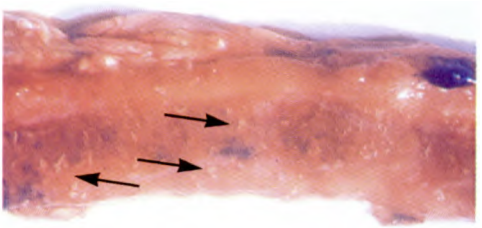

Fig. 636 *Davainea proglottina* (1.5–4 mm × 0.4–0.6 mm) attached to the intestinal mucosa [8]

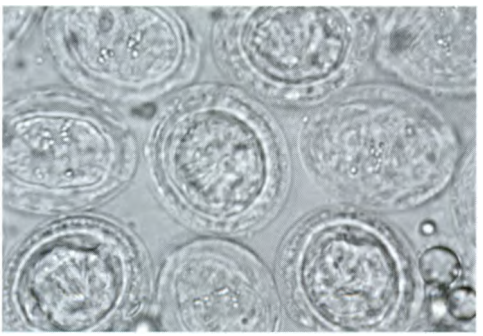

Fig. 637 Eggs of *Davainea proglottina* (55 × 36 µm) containing embryonal hooks

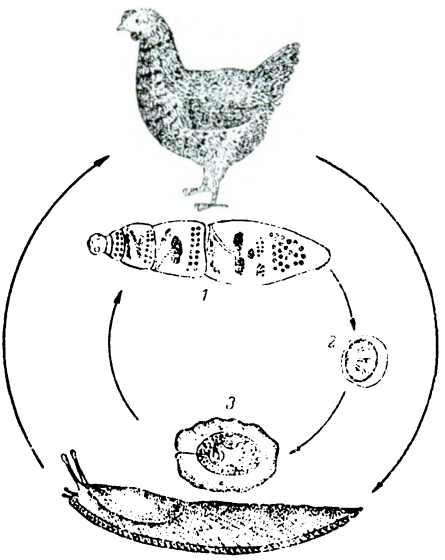

Fig. 638 Life cycle of *Davainea proglottina*; adult worm (1), eggs (2) and larval stage in the intermediate host (3)

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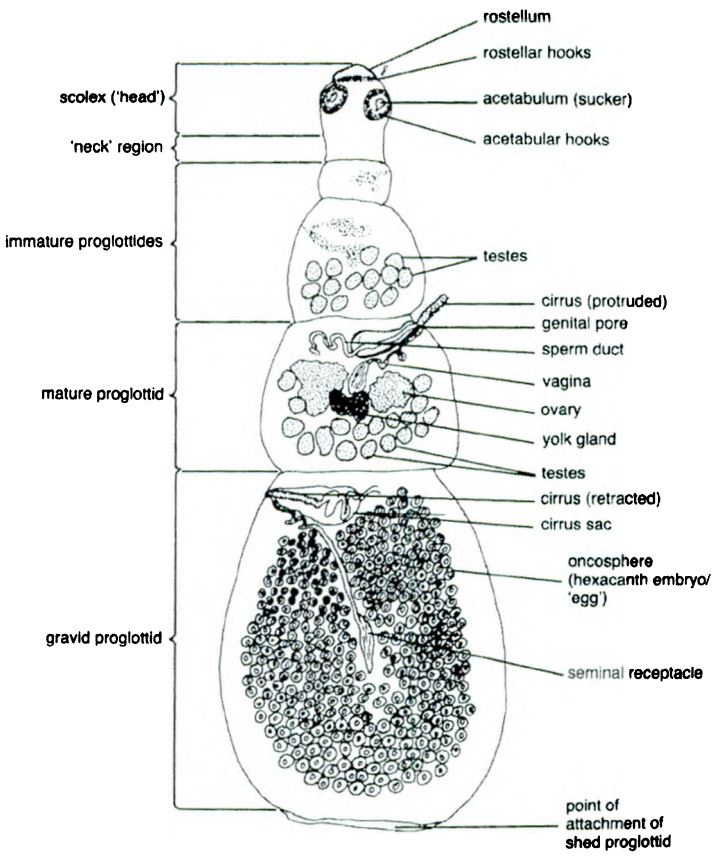

Fig. 639 *Davainea proglottina*; scolex and proglottids (schematic) [5]

	Davainea proglottina	*Raillietina (Raillietina) tetragona*	*Raillietina (Raillietina) echinobothrida*	*Raillietina (Skrjabinia) cesticellus*	*Houttuynia struthionis*	*Amoebotaenia sphenoides*	*Choanotaenia infundibulum*
Length	0,5–3 mm	25 cm	13 cm	4–13 cm	60 cm	4 mm	0,5–20 cm
Suckers	Armed, oval	Armed	Armed, circular	Unarmed	Unarmed	Unarmed	Unarmed
Scolex hooks:							
Shape	Hammer	Hammer	Hammer	Hammer	Hammer	Rosethorn	Rosethorn
Number	80–94	100	200	400–500	160	12–14	16–20
Size	7–8 μm	6–8 μm	10–30 μm	7–12 μm	Large 77,6 μm; Small 63 μm	—	25–30 μm
Eggs	One per capsule	6–12 per capsule	8–12 per capsule	One per capsule	15–25 per capsule	One per capsule	One per capsule *Musca*
Intermediate hosts	Slugs	*Musca* spp. ants	Ants	*Musca* spp. beetles	Unknown	Earthworms	spp. other arthropods

Fig. 640 Description of the morphology of cestode species of poultry [5]

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Raillietina spp. (*R. cesticillus*, *R. echinobothrida* and *R. tetragona*)

Location: Small intestine

Hosts: Chicken, turkey, pigeon and many other domestic and wild birds

Species description: *R. cesticillus*: length 9–13 cm, width 1.5–3 mm and size of eggs 93 × 74 µm; *R. echinobothrida* length up to 25 cm, width 1.2–4 mm and size of eggs 93 × 74 µm; *R. tetragona* length 10–25 cm, width 1–4 mm and size of eggs 93 × 74 µm. The size of the eggs is identical whereas the number of egg capsules within the gravid proglottids and the number of eggs in each capsule may be of diagnostic value.

Ants (Hymenoptera) act as intermediate hosts for *R. echinobothrida*; flies (*Musca domestica*) or ants (*Tetramorium* spp.; *Orthophagus* spp. and others) for *R. tetragona*; beetles (Coleoptera) for *R. cesticillus*. *R. tetragona* is one of the largest of the fowl tapeworms, the scolex is smaller than that of *R. echinobothrida* and the rostellum is armed with 1–2 rows of hooks and the suckers are oval and armed. *R. echinobothrida* resembles *R. tetragona* in size and shape but the rostellum is more heavily armed (two rows of hooks) than in *R. tetragona*. *R. cesticillus* is smaller and has a large scolex with a wide rostellum armed with 400–500 small hooks.

Significance: The pathogenicity of *R. echinobothrida* and *R. tetragona* is moderate to severe. *R. cesticillus* is mild or harmless.

(Figures 641, 642, 643, 644)

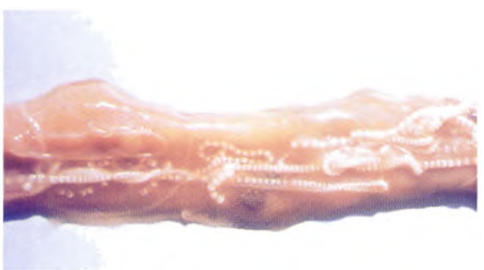

Fig. 641 *Raillietina* sp. (up to 25 cm long and 4 mm wide) in the small intestine [8]

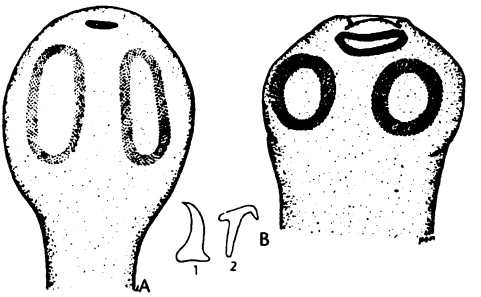

Fig. 642 Scolex of *Raillietina tetragona* (A) and scolex of *R. echinobothrida* (B); hook from sucker (1) and from rostellum (2) [3]

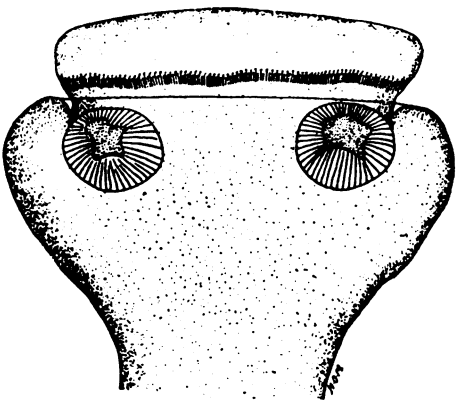

Fig. 643 Scolex of *Raillietina cesticellus* [3]

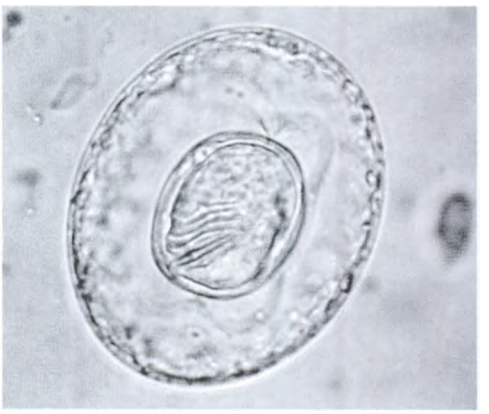

Fig. 644 Egg of *Raillietina* spp. (90–95 × 70–75 µm) [11]

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Amoebotaenia cuneata (syn. *A. sphenoides*)

Location: Small intestine
Hosts: Chicken
Species description: This is a short tapeworm (< 4 mm) and may be recognized as whitish projections among the villi of the duodenum. A triangular anterior end with a pointed scolex gives the entire worm a wedge-shaped anterior. The suckers are unarmed and the rostellum is armed with a single row of 12–14 hooks. The eggs are round and measure 47 × 42 µm. The intermediate hosts are earthworms (several genera).
Significance: This species is mildly pathogenic.
(Figures 630, 645)

Fig. 645 *Amoebotaenia* spp. (2–4 mm long) rostellar hook (a) [3]

Choanotaenia infundibulum

Location: Small intestine
Hosts: Chicken
Species description: The worms are up to 23 cm long and 1.5–3 mm wide. Eggs measure 54 × 47µm with a distinctive elongated filament. The segments are markedly wider posteriorly than anteriorly, giving the worm a characteristic shape. The intermediate hosts are the house fly (*Musca domestica*) and beetles of several genera.
Significance: This species is moderately pathogenic.
(Figures 630, 646)

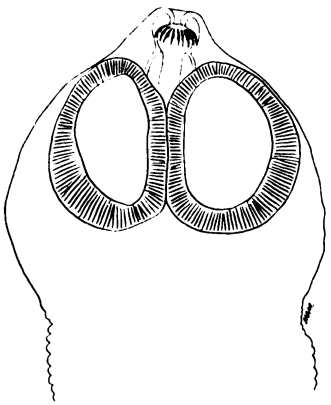

Fig. 646 Scolex of *Choanotaenia infundibulum* (up to 23 cm long and 1.5–3 mm wide) [3]

Hymenolepis spp. (*H. cantaniana, H. carioca*)

Location: Small intestine
Hosts: Chicken
Species description: The intermediate hosts are stable flies or dung beetles for *H. carioca* and other beetles for *H. cantaniana*. *H. cantaniana* is a short tapeworm with a maximum length of 2 cm. It resembles *H. carioca* which is much longer. The latter is about 1 mm in diameter and the proglottids look more like a thread than a worm.
Significance: The pathogenicity of these species is mild.
(Figure 647, 648)

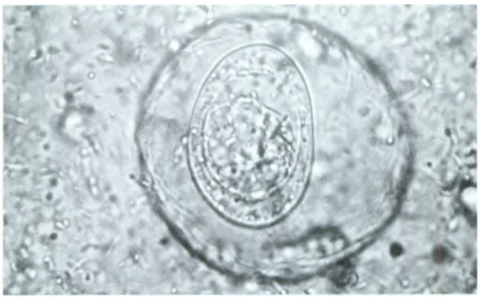

Fig. 647 Egg of *Hymenolepis* spp. (76 × 62 µm) [8]

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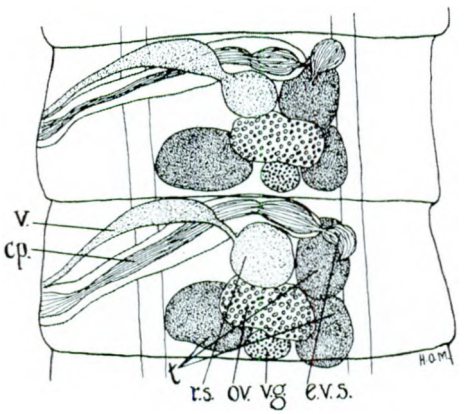

Fig. 648 Mature segments of *Hymenolepis carioca*; v = vagina, cp = cirrhus pouch, ov = ovary, t = testes, r.s. = receptabulum seminis, v.g. = vitelline gland and e.v.s = external vesiculum seminalis [3]

Fimbriaria fasciolaris

Location: Small intestine
Hosts: Chicken
Species description: The anterior portion of the body of this cestode forms a folded expansion or "pseudoscolex" by which the parasite attaches itself. The cestode is 40 cm long and 1.5 mm wide. Copepods (*Diaptomus* spp. and *Cyclops* spp.) act as intermediate hosts.
Significance: The pathogenicity of this species is mild.
(Figure 649)

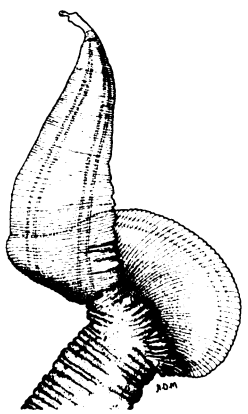

Fig. 649 Scolex and pseudoscolex of *Fimbriaria fasciolaris* (up to 40 cm long and 1.5 mm wide) [3]

- **General features of avian cestode infections**

Chickens kept in free range systems on pastures are often infected with tapeworms (Cestoda). In many African and Asian countries a high percentage of chickens are infected with tapeworms. In contrast, tapeworm infections are very rarely encountered in intensive poultry rearing systems.
D. proglottina is the most important species and highly pathogenic. The parasite may occur in high numbers in the duodenum and is known to penetrate deeply within the villi. Necrosis and haemorrhagic enteritis can occur following heavy infections. The infection may be fatal. Of the *Raillietina* species *R. echinobothrida* is the most pathogenic. Multiple nodules and hyperplastic enteritis at the attachment site of the scolex can occur. Other species are not normally harmful unless the infection is extremely heavy, which can cause significant decrease of production. Infection is acquired by ingestion of intermediate hosts in which the cysticercoids occur.

Symptoms: Chronic infections are characterized by reduced growth rate, emaciation, paralysis and weakness.
Diagnosis: Although proglottids are passed in the faeces, diagnosis is usually made at necropsy. Representative members of the flock should be examined and mucosal scrapings should be examined microscopically to detect the small cestode species. It is difficult to make a reliable diagnosis of all the individual species and also not necessary for chemotherapy.
Therapy: Praziquantel (1 × 10 mg/kg, po.) is highly effective against adult cestodes of chickens. Niclosamid (20 mg/kg, po. during 2–6 days) is effective against most of the chicken tapeworms. A single application of niclosamid (50–200 mg/kg, in

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drinking water; water should be consumed within 3–4 hours) may also be an efficient way of controlling cestode infections. Fenbendazole (100 ppm. via feed, during 4 days), mebendazole (60 ppm via feed, during 7 days), flubendazole (60 ppm, during 7 days), oxfendazole (10 mg/kg, po.) and febantel (30 mg/kg, po.) may be used to control tapeworms in chickens.

Cave: Niclosamid is toxic for geese! The combination of praziquantel and pyrantel tartrate is toxic for chickens and may cause severe neurological problems.

Prophylaxis: Control measures should be directed against the intermediate hosts which is normally impossible. Insecticides may be used for flies and ants in the chicken house. Beetles and earthworms are difficult to control but alternation of ranges may be helpful. The use of insecticides on the pastures is not justifiable.

- **Nematoda eggs found in the faeces and adult nematodes living in the gastrointestinal tract**

- **Nematodes of primary importance**

Ascaridia galli

Location: Small intestine

Hosts: Chicken, turkey, geese, duck

Species description: The males are 50–76 mm and the females are 72–116 mm long. The eggs are elliptical, thick-shelled and 77–94 × 43–55 µm in size. The head has 3 large lips and the tail of the male shows a typical circular preanal sucker. The life cycle is direct. Earthworms can ingest the eggs and when they are swallowed by the final host can transmit the infection mechanically. The eggs hatch in the intestine of the host and live for the first ten days in the lumen after which larval development continues within the mucosa of the intestines until 17 days after infection. Subsequently the larvae re-enter the lumen and reach maturity in 6 to 8 weeks. The incubation period is 3–5 days (clinical symptoms following invasion of the larvae) and the prepatent period is 6–8 weeks.

Geographic distribution: World-wide

Symptoms: Marked lesions may be produced when large numbers of young parasites penetrate into the duodenal mucosa. They cause haemorrhage and enteritis and the birds become anaemic and suffer from diarrhoea. Chickens become unthrifty, emaciated and weak.

Significance: *A. galli* may be a serious nematode for young chickens 3–4weeks old. The egg production may seriously be impaired and yield pale egg yolk.

Diagnosis: Detection of eggs in the faeces (*cave: Heterakis* eggs [see below] look very similar!) or of the adult worms in the intestine at autopsy.

Therapy: A wide variety of compounds may be used to treat *Ascaridia* infections in chickens. These are cambendazole (70 mg/kg, po.), febantel (60 ppm via feed over 6 days or 15 mg/kg, po. over 2 days), fenbendazole (60 ppm, over 3 days), flubendazole (30 ppm, over 7 days), levamisole (30 mg/kg, po. or 300 ppm via feed), mebendazole (60 ppm, over 7 days), piperazine (200–300 mg/kg, po.) and pyrantel tartrate (10–40 mg/kg po. over 2 days).

Prophylaxis: Special attention should be paid to the young birds. Eggs develop in moist environment. Therefore, litter should be removed regularly and kept dry.

(Figures 650, 651)

Fig. 650 *Ascaridia galli*; masses in the small intestine [53]

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Fig. 651 Egg of *Ascaridia galli* (77–94 × 43–55 µm) [11]

Diagnosis: Detection of eggs in the faeces (flotation). The eggs have thick, smooth shells; they measure 65–77 × 35–48 µm. The eggs are similar to those of *Ascaridia galli* but they are generally smaller than 77 µm. Adult worms may be found in the caecum at necropsy.

Therapy: Most of the compounds effective against *Ascaridia galli* may also be used to control *Heterakis gallinarum* (☞ above), except piperazine and pyrantel tartrate which are not effective against this parasite.

Prophylaxis: Preventive control of *H. gallinarum* is indicated in flocks where histomonosis occurs.

(Figures 652, 653, 654)

Heterakis gallinarum Caecal worm

Location: Caecum

Hosts: Chicken, turkey and guineafowl

Species description: The male is 7–13 mm and the female is 10–15 mm long. There are large lateral alae extending some distance down the sides of the body. The life cycle is direct. Earthworms may also ingest the eggs of the *Heterakis* and may be the means of causing infections in poultry. The larval development in the egg to the infective second larval stage (eggs are infective !) in the environment depends on the temperature and takes 2–4 weeks at 27°C.

Geographic distribution: World-wide

Symptoms: Primary infections are usually inapparent. Secondary infections are associated with nodule formation in the mucosa and submucosa of the caecum. Marked inflammation and thickening of the intestinal wall may occur in heavy infections. Heavy infections in laying hens may markedly reduce the egg production.

Significance: The principal economic importance of *H. gallinarum* lies in its role as carrier of *Histomonas meleagridis*, the causal pathogen of enterohepatitis ("blackhead") of turkeys. The protozoan remains viable in the egg of *H. gallinarum*.

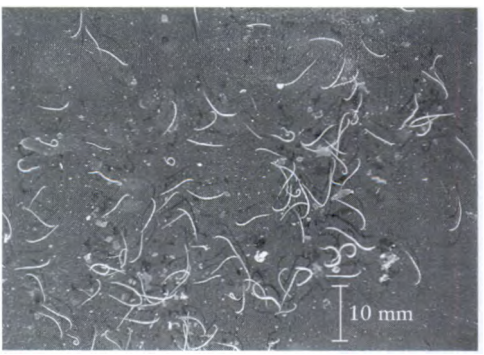

Fig. 652 *Heterakis* sp. in a petri dish; males are 7–13 mm and females 10–15 mm long

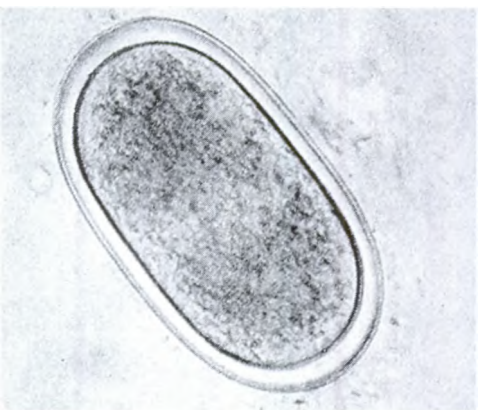

Fig. 653 Egg of *Heterakis gallinarum* (66–79 × 41–48 µm) [11]

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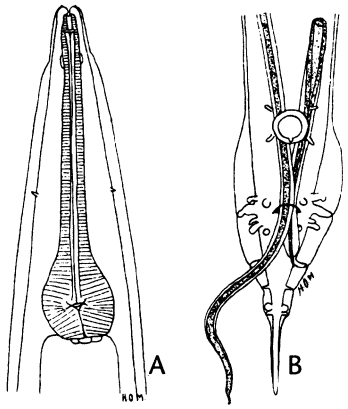

Fig. 654 *Heterakis gallinarum*; anterior end (A) and posterior end (B) of a female [3]

Capillaria annulata (syn. *Eucoleus annulatus*) **and *Capillaria contorta*** (syn. *Eucoleus contortus*) Crop capillariosis

Location: Mucosa of the crop and oesophagus

Hosts: Chicken, turkey, guineafowl and wild birds

Species description: The intermediate hosts for *C. annulata* are earthworms. *C. contorta* has a direct life cycle although earthworms may act as intermediate hosts. *C. annulata* can easily be identified by a cuticular swelling just back of the head. The male is 18–26 mm long and 52–74 µm wide. The spicules are 1.12–11.6 mm long and the spicule sheath is covered with fine spines. The female is 25–60 mm long and 77–120 µm wide. The eggs are operculated and measure 55–66 × 25 µm. *C. contorta* males are 8–17 mm long and 60–70 µm wide. The spicules are slender and about 800 µm long and the spicule sheath is covered with fine hair-like processes. Females are 15–60 mm long and 120–150 µm wide. The vulva is prominent and circular.

Symptoms: Heavy infections are characterized by marked thickening of the crop and oesophageal wall, a marked catarrhal or croupous inflammation and sloughing of the mucosa. The birds are emaciated and weak.

Significance: The *Capillaria* species of the crop and oesophagus are highly pathogenic and deaths may occur in heavy infections.

(Figures 630, 655, 656, 657)

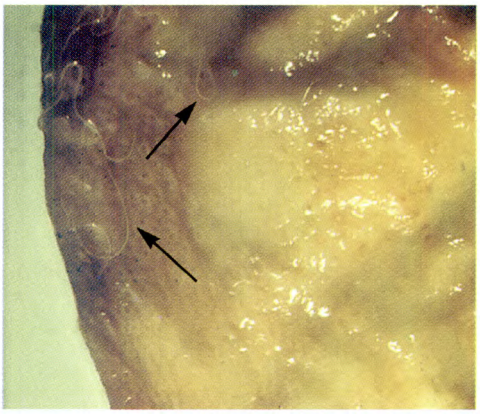

Fig. 655 *Capillaria* (syn. *Eucoleus*) *annulatus* in the crop of a turkey; males are 14–16 mm and females are 20–26 mm long [8]

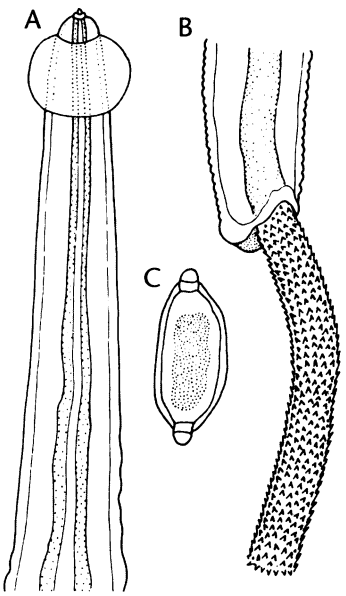

Fig. 656 *Capillaria* (syn. *Eucoleus*) *annulatus*; anterior end (A) and posterior end (B) of male with spiny spicule sheath and egg (C) [56]

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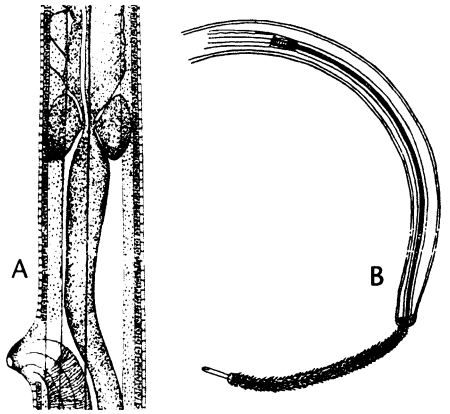

Fig. 657 *Capillaria contorta* (syn. *Thomix contorta*, *Eucoleus contorta*); female vulva (A) and posterior end of male with spiny spicule sheath (B) [56]

Capillaria caudinflata, Capillaria obsignata and other *Capillaria* spp.
Small intestinal capillariosis

Location: Small intestine, sometimes caecum
Hosts: Chicken, turkey and guineafowl
Species description: Earthworms are the intermediate hosts of *C. caudinflata*. *C. obsignata* has a direct life cycle. *C. caudinflata* females are 12–25 mm long. The vulva shows a characteristic appendage. The male is 9–18 µm long. *C. obsignata* males are 7–13 mm long and the spicules are very long (1.1–1. 5 mm) with a sheath with transverse folds without spines. The females are 10–18 mm long without special characteristics.
(Figures 630, 658, 659)

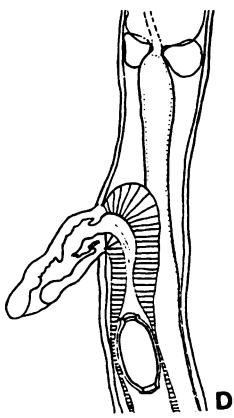

Fig. 658 *Capillaria caudinflata*; vulva with typical appendage [56]

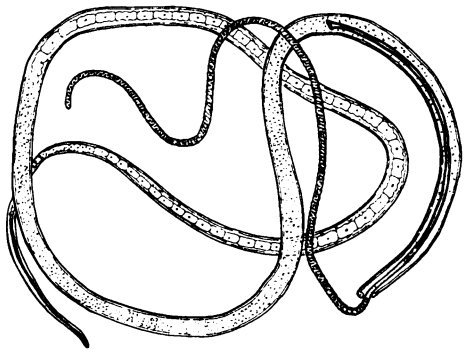

Fig. 659 *Capillaria obsignata*; male with spicule sheath without spines but with transverse folds [56]

Capillaria anatis Caecal capillariosis

Location: Caecum, rarely small intestine
Hosts: Duck, goose, chicken, turkey and guineafowl
Species description: Males are 8–15 mm and the spicules are 0.7–1.9 long. Females are 11–28 mm long. The eggs measure 46–67 × 22–29 µm. This is commonly a parasite of water birds but may also occur in chickens. The life cycle is direct.
Symptoms: The intestinal capillariosis (*C. caudinflata*, *C. obsignata* and *C. anatis*) is seen in birds of all ages. Older birds may act as carriers of the infection. Disease may occur during the prepatent period. There is emaciation, diarrhoea associated with a slimy, stringy mucous. Chronically infected birds have thickened intestinal walls covered with a catarrhal exudate.
Significance: Intestinal *Capillaria* infections cause marked losses due to reduced weight gains.

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- **General features of *Capillaria* spp. infections**

Diagnosis: Usually made at necropsy when large numbers of parasites can be found in the crop or in the intestine after an intestinal lavage. The lemon-shaped eggs may also be detected in large numbers in the faeces.

Therapy: The same compounds as for *Heterakis gallinarum* may be used to treat *Capillaria* infections (☞ above).

Prophylaxis: Regular investigations of faeces may provide information about the level of infection in intensive poultry systems. Regular anthelmintic control may reduce disease incidence. The control of earthworms on the pastures is not feasible.

(Figures 660, 661)

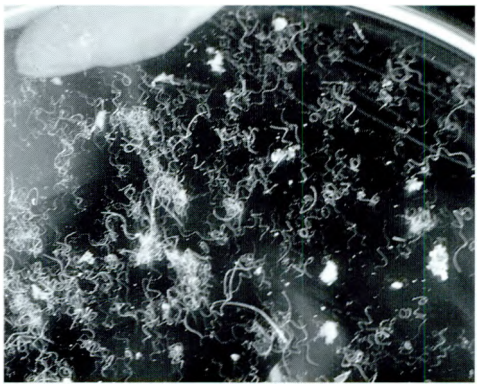

Fig. 660 *Capillaria* sp.; adult worms in a petri dish [53]

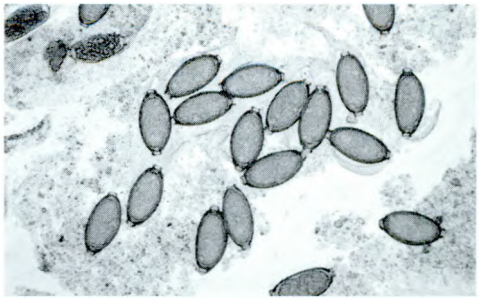

Fig. 661 Eggs of *Capillaria* sp. (43–65 × 20–35 µm) [53]

Trichostrongylus tenuis

Location: Caecum, sometimes small intestine

Hosts: Chicken, turkey, duck and other game birds

Species description: Excretory pore at the head end. Males are 5.5–9 mm and females 6.5–11 mm long. The spicules are dark and unequal in length (the longer 120–164 µm and the shorter 104–150 µm long) both much twisted and provided with an ear-like structure on the proximal end. The eggs are thin-shelled and measure 65–75 × 35–42 µm. The life cycle is direct and infective third-stage larvae develop in 2 weeks.

Geographic distribution: World-wide

Symptoms: Heavy infections cause weight loss, anaemia, inappetence, progressive emaciation. The faeces may be slimy, liquid and bloody.

Significance: This is a very pathogenic species. Heavy infections may cause severe losses especially in floor-reared birds and backyard flocks. *T. tenuis* may be fatal for young goslings.

Diagnosis: Typical strongyle-type eggs appear in the faeces. Adult parasites may be found at necropsy.

Therapy: Febantel (60 ppm via feed over 6 days or 15 mg/kg, po. over 2 days), fenbendazole (60 ppm, over 3 days), flubendazole (30 ppm, over 7 days) and mebendazole

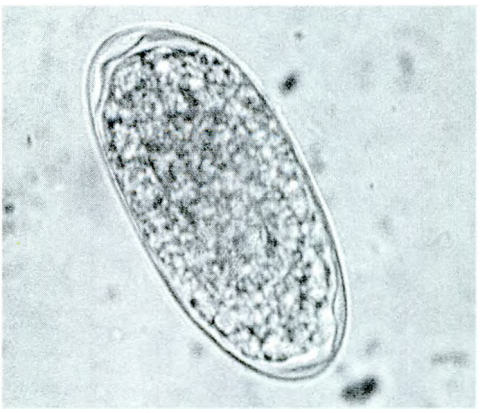

Fig. 662 Egg of *Trichostrongylus tenuis* (65–75 × 35–42 µm) [11]

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(60 ppm, over 7 days) are highly effective against *T. tenuis* in chickens.

Prophylaxis: Contaminated pastures should be avoided in flocks where the parasite causes problems. Regular anthelmintic treatments are indicated.

(Figure 662)

- **Nematodes of secondary importance**

Dispharynx spiralis (syn. *D. nasuta, Acuaria spiralis*)

Location: Proventriculus

Hosts: Chicken, turkey, pigeon and guinea-fowl

Species description: The body of this parasite is usually rolled in a spiral and four wavy cuticular cordons on the anterior end are typical. Males are 7–8 mm and females 9–10 mm long. Embryonated eggs are produced by the females. Intermediate hosts are the pillbug (*Armadillidium vulgare*) and the sowbug (*Porcellio scaber*) and probably other isopods. These roundworms are usually found with their heads buried deep in the mucosa. *D. spiralis* has been considered as the main cause of "grouse disease" in the USA.

Geographic distribution: Africa, Asia, the Americas

Symptoms: Deep ulcers may occur in the proventriculus and the wall may markedly be thickened in heavy infections. Affected birds rapidly loose weight and become weak and anaemic.

Significance: This parasite may cause severe losses in young birds.

Diagnosis: This is based on the demonstration of embryonated eggs in the faeces or the adult parasites at necropsy. The parasites are often completely concealed beneath the proliferating tissue.

Therapy and Prophylaxis: ☞ PREVENTION AND CONTROL OF INTESTINAL NEMATODE INFECTIONS, p. 366

(Figures 630, 663)

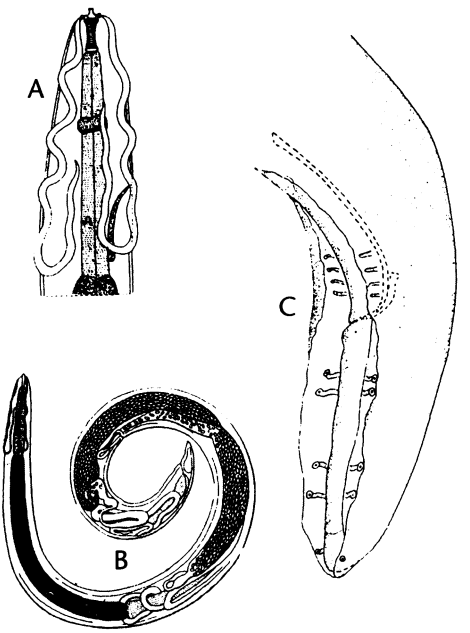

Fig. 663 *Dispharynx spiralis*; anterior end with wavy cuticular cordons (A), total female parasite (B; up to 10 mm long) and male posterior end (C) [56]

***Tetrameres* spp. (*T. americana* and *T. fissispina*)**

Location: Proventriculus, oesophagus and rarely intestine

Hosts: *T. americana* occurs in chicken, turkey, duck and other birds; *T. fissispina* occurs most commonly in duck and goose and rarely in other poultry. It may occur in chicken.

Species description: There is a marked sexual dimorphism. The females of this species are globular, blood red with four longitudinal furrows. Females of *T. americana* measure 3.5–4.5 mm by 3 mm. Males are 5–5.5 mm long. Eggs measure 42–50 × 24 µm and are embryonated when laid. Two double rows of posteriorly directed spines extend throughout the whole body length. The spicules are unequal in length and measure 100 µm and 290–312 µm. *T. fissispina* is similar in appearance to *T. americana*.

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Females are 1.7–6 mm long and 1.3–5 mm wide. Males are 3–6 mm long with 4 longitudinal rows of spines along the median and lateral lines. Eggs measure 48–56 × 26–30 µm and are embryonated when laid. Intermediate hosts include grasshoppers, cockroaches for *T. americana* and amphipods, grasshoppers, earthworms and cockroaches for *T. fissispina*.

Geographic distribution: *T. americana* has been reported from USA and South Africa. *T. fissispina* occurs world-wide.

Symptoms: Emaciation and anaemia may result in heavy infections. The wall of the proventriculus may be thickened so that the lumen is almost obliterated.

Significance: Heavy infections may cause severe digestive disturbances, weight loss and death.

Diagnosis: Embryonated eggs appear in the faeces and adult worms may be found at necropsy as black or red spots in the opening of the glands.

Therapy and Prophylaxis: ☞ PREVENTION AND CONTROL OF INTESTINAL NEMATODE INFECTIONS, p. 366

(Figures 664, 665, 666, 667)

Fig. 665 *Tetrameres fissispina*; females (2.5–4 mm long and up to 2 mm wide) [4]

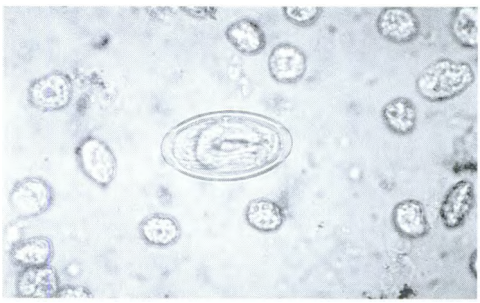

Fig. 666 Egg of *Tetrameres fissispina* (45–57 × 25–32 µm) [11]

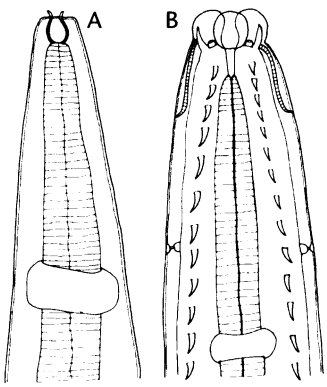

Fig. 664 Anterior ends of *Tetrameres americana* (A) and *T. fissispina* (B) [56]

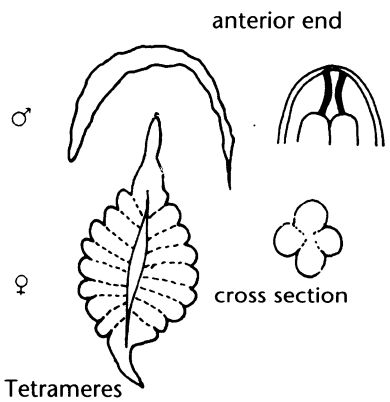

Fig. 667 Details of *Tetrameres* sp. [16]

Acuaria (syn. *Cheilospirura*) *hamulosa*

Location: Gizzard

Hosts: Chicken, turkey and game birds

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Species description: Intermediate hosts are grasshoppers and beetles. Males are 10–14 mm and females 16–29 mm long, with two large triangular lateral lips. The cuticle of the anterior part of the body is ornamented with four "cordons" which are epaulette-like thickenings and extending at least 2/3 of the length of the body. The male has four pairs of pre-cloacal and 6 pairs of post-cloacal papillae. The eggs measure 40–45 × 24–27 µm and are embryonated when passed in the faeces.

Geographic distribution: World-wide

Symptoms: Light infections are inapparent. Heavy infections cause inappetence, weakness, progressive emaciation and anaemia.

Significance: Low numbers of these worms are relatively non-pathogenic. Heavy infections cause massive alterations of the gizzard.

Diagnosis: Embryonated eggs are passed in the faeces. Several species of Spiruridae may occur in the faeces and the eggs are very similar and difficult to differentiate. The worms may be found after the horny lining of the gizzard has been removed. They may be found in soft, yellowish-red nodules in the thinner parts of the wall (Figure 630).

Therapy and Prophylaxis: ☞ PREVENTION AND CONTROL OF INTESTINAL NEMATODE INFECTIONS, p. 366 (Figures 630, 668)

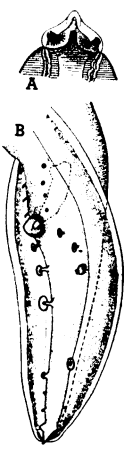

Fig. 668 *Acuaria hamulosa*; anterior end (A) and female posterior end (B) [56]

Hartertia gallinarum

Location: Small intestine

Hosts: Chicken

Species description: This worm resembles closely *Ascaridia galli*. The male is 28–40 mm long and the female is 60–100 mm long. The eggs are thick shelled and measure 45–53 × 27–33 µm and contain a fully developed embryo when laid. Intermediate hosts are termites and chickens become infected when eating infected termites.

Geographic distribution: South and West Africa, Asia

Symptoms: Diarrhoea, weight loss and decreased egg production, emaciation and weakness.

Significance: This parasite may cause losses of chickens kept in free range systems in Africa.

Diagnosis: This can be made by finding the eggs in the faeces but it is often difficult to distinguish them from eggs of other nematodes of chickens. Diagnosis should therefore be confirmed by autopsy of selected cases (Figure 630).

Therapy: ☞ *Ascaridia galli*, p. 357

Prophylaxis: Chickens should be kept away from termites when losses due to *H. gallinarum* occur. ☞ PREVENTION AND CONTROL OF INTESTINAL NEMATODE INFECTIONS, p. 366

(Figures 630, 669, 670)

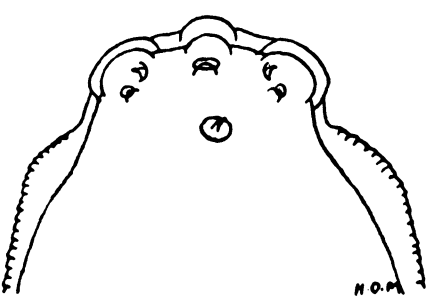

Fig. 669 *Hartertia gallinarum*; anterior end [3]

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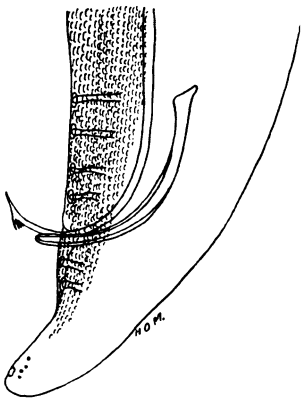

Fig. 670 *Hartertia gallinarum*; male posterior end, lateral view [3]

may greatly reduce the incidence of parasitic infections where this type of ground-living intermediate host occurs.
(Figures 630, 671)

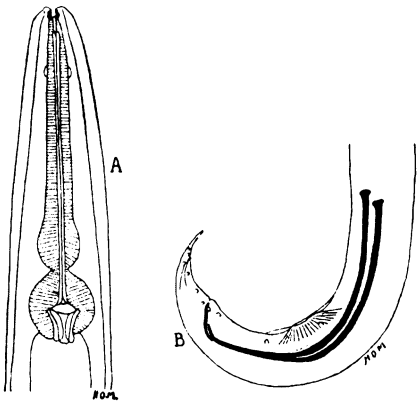

Fig. 671 *Subulura brumpti*; anterior end (A) and male posterior end, lateral view (B) [3]

Subulura brumpti

Location: Caecum
Hosts: Chicken, turkey, duck, guineafowl and other birds
Species description: This is a small nematode with anterior end curved dorsally and an oesophagus dilatated posteriorly, followed by a bulb. The males are 6.9–10 mm long and the females 9–17.5 mm. The eggs measure 52–64 × 41–49 µm and have a smooth shell. The eggs contain a fully developed embryo when laid. The intermediate hosts are various beetles and cockroaches.
Geographic distribution: Africa, the Americas, Asia
Symptoms: There are generally no marked clinical signs.
Significance: This parasite seems to be relatively non-pathogenic.
Diagnosis: This is based on the demonstration of the eggs in the faeces and the identification of adult worms at necropsy.
Therapy: Levamisole (30 mg/kg, po.) has been used for therapy.
Prophylaxis: This is difficult since the intermediate hosts are difficult to remove or destroy in the environment of chickens. Modern confinement rearing of poultry

Subulura suctoria and *Subulura differens*

Remarks: *S. suctoria* occurs in the caecum of chickens, turkeys, guinea fowls in Africa and South America. It is larger than *S. brumpti*: males are 11.8–13.8 mm and females 20–33 mm long. The eggs measure 51–70 × 45–64 µm. Intermediate hosts are beetles. The pathogenicity seems to be low although heavy infections may cause losses due to reduced performance. *S. differens* is similar to *S. brumpti* and occurs in chickens and guinea-fowls in southern Europe, Africa and Brazil. For therapy and prophylaxis ☞ PREVENTION AND CONTROL OF INTESTINAL NEMATODE INFECTIONS, p. 366.

Strongyloides avium

Location: Caecum, sometimes small intestine
Hosts: Chicken, turkey and goose
Species description: Only the females are parasitic. Parasitic adults are 2.2 mm long and the size of the eggs is 52–56 × 36–40 µm. Direct life cycle; percutaneous infections may occur.

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Geographic distribution: World-wide

Symptoms: Acute heavy infections cause weakness, emaciation and bloody, slimy diarrhoea. Young birds suffer more from infections.

Significance: This may be a serious pathogen especially for floor-reared young birds.

Diagnosis: The small embryonated eggs may be found in the faeces. Adult parasites may be demonstrated in mucosa scrapings taken from the caecal mucosa at necropsy (Figure 130).

Therapy and Prophylaxis: ☞ below PREVENTION AND CONTROL OF INTESTINAL NEMATODE INFECTIONS

(Figures 630, 672)

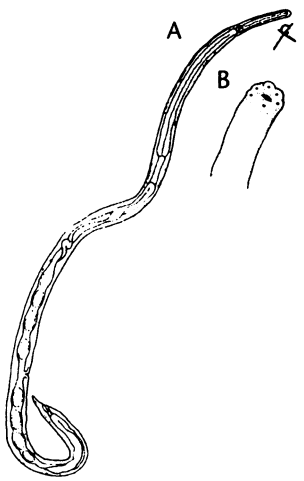

Abb. 672 *Strongyloides avium*; parasitic female (A) and head of female (B) [56]

- **Prevention and control of intestinal nematode infections**

Modern poultry management practices, especially confinement-rearing of chickens have greatly reduced the prevalence and spectrum of nematode infections in poultry. Many nematodes that caused extensive problems in "backyard" flocks are seldom seen in commercial poultry systems. However, nematode infections are still of great significance wherever chickens are reared on pens or on pastures. For most nematodes control measures consist of sanitation and breaking the life cycle rather than chemotherapy. Confinement-rearing on litter prevents infections with nematodes using outdoor intermediate hosts such as earthworms or grasshoppers. Conversely, nematodes with direct life cycles or those that utilize indoor intermediate hosts such as beetles and cockroaches may also develop indoors. Treatment of the soil or litter with insecticides (stirofos, ronnel, carbaryl) in order to kill the intermediate hosts has been described to be beneficial but it is no longer justifiable. Extreme care should be taken to insure that feed and water are not contaminated. To control beetles, the floor may be sprayed with permethrin after the old litter has been removed. Raising different bird species and/or different ages together or in close proximity is a dangerous procedure as regards parasitism.

In many situations nematodes can only be controlled effectively by means of chemotherapy. A wide variety of drugs is available and the poultry producers should only use drugs which are approved for birds that will produce eggs or meat for human consumption. The withdrawal periods should strictly be respected. Anthelmintics may be administered via feed or drinking water. The drugs listed under therapy of *Ascaridia galli* (p. 357) infections may be used to control the most important nematode infections in poultry. Tetramisole may be used to control *Subulura brumpti* and *Strongyloides avium*. Tetramisole is not effective against *Dispharynx spiralis* which can partially be controlled with mebendazole. Apart from the modern benzimidazoles piperazine may also be used to treat *Tetrameres* spp. infections. Ivermectin (0.2–0.4 mg/kg, po.) has been reported to be highly effective against *Ascaridia* and *Capillaria* and probably other intestinal nematodes.

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2 Stages in the blood and circulatory system

PROTOZOA

Trypanosoma spp. Avian trypanosomosis

Remarks: Several species of trypanosomes occur in avian hosts. They are usually not a problem in commercial flocks but widespread in wild birds. Avian trypanosomosis is reported to be seasonal in temperate zones, being discovered only during summer times. Bloodsucking arthropods, including mosquitoes, simuliids, red mites and hippoboscids are the vectors. Bird trypanosomes persist during periods of adverse environmental conditions (e.g. winter), are more or less restricted to the bone marrow, and reappear in the peripheral blood in spring. Avian trypanosomes are presumably non-pathogenic. *T. avium* occurs in Europe and *T. gallinarum* has been described from the chicken in central Africa. Avian trypanosomes are very polymorphic, sometimes attaining great size. They may be 26–69 µm long or even longer. There is a free flagellum and the body often appears to be striated.

Toxoplasma gondii

Remarks: Chickens kept on pastures, pens or in free range systems are often infected with *T. gondii* because of their continuous ingestion of oocysts with infected faeces of cats. The infection in adult birds is often inapparent although free merozoites may be seen in bloodsmears or in leucocytes. These merozoites are 6–7 µm long. Young chickens may seriously be affected during these acute stage of infection, showing weakness, emaciation, diarrhoea, atactic gait and even death (☞ POULTRY, ■ 4.1 and SWINE, ■ 4.1).

Sarcocystis spp. Sarcosporidiosis

Remarks: Chickens kept in free range systems often harbour cysts of *Sarcocystis* spp. in their muscles. The examination of blood within 14 days after infection may reveal free merozoites which are about 9 µm long (☞ POULTRY, ■ 4.1). These merozoites develop within the epithelial cells of the blood vessels.

Plasmodium spp. Avian malaria

Location: Gamonts are found in the erythrocytes and endothelial cells of birds.

Hosts: A wide variety of birds may be affected, including chicken, turkey, guineafowl and wild birds.

Vector: Mosquitoes (mainly *Culex* and *Aedes*)

Species description: The avian malaria is caused by several species of *Plasmodium*. Species with round or irregular gamonts which displace the nucleus of the host erythrocytes are *P. gallinaceum* and *P. juxtanucleare*.

Species with elongate gamonts which do not usually displace the nucleus of the host erythrocytes are *P. durae*, *P. fallax* and *P. lophurae*. The pathologic effects in avian hosts range from no apparent signs to severe anaemia. Zoo penguins are very susceptible and succumb very quickly. Infected mosquitos introduce sporozoites into the host.

Geographic distribution: World-wide

Symptoms: Affected birds are droopy and weak. Severely affected birds have a protruding abdomen and the liver and spleen are enlarged and discoloured (dark-brown to black). Birds become progressively weak and apathetic. Coma and death may occur in heavy infections after a short period.

Ocular haemorrhage may occur. Severe anaemia may also be observed. Central nervous dysfunction may result from blockage of capillaries by exoerythrocytic stages of *P. gallinaceum*.

Significance: Infections occur world-wide with high frequencies. *P. gallinaceum*, *P. juxta-*

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nucleare and *P. durae* are the most pathogenic species for chickens and may cause mortalities of up to 90%.

Diagnosis: Giemsa-stained bloodsmears usually reveal numerous organisms (gamonts and meronts) in the erythrocytes. Infected cells also have dark pigment granules (digested haemoglobin). As liquid blood samples cool, microgamonts produce microgametes that enter the plasma. Microgametes of *Plasmodium* spp. and related *Haematozoa* can be mistaken for *Borrelia spirochaetes*, when moving in wet smears.

Therapy: Affected animals or flocks may be treated with chloroquine (1 mg/kg, im., daily during 5 days; 250 mg/120 ml of drinking water) can be used. Grape or orange juice may be used to disguise the drug's bitterness. Quinacrine (1.6 mg/kg, im., daily during 5 days) and primaquine (100 mg/kg, po.) and sulfonamids combined with trimethoprime may be used. In penguins, 5 mg/kg daily in fish may be used for prevention. Pyrimethamine, chlorguanil or trimethoprime may also be used. Pyrimethamine is reported to be the best suppressive drug. Any antimalarial drug may be tried, but strain differences in drug susceptibility are found.

Prophylaxis: Mosquito screening of the chicken houses may prevent transmission by the vector.

(Figure 673)

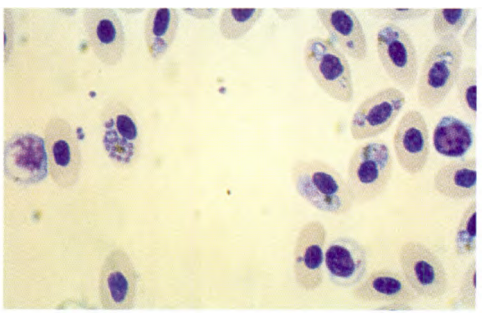

Fig. 673 *Plasmodium gallinaceum*; schizont and macrogametocyte; stained bloodsmear (1000 ×) [15]

Haemoproteus spp. (*H. columbae* and *H. danilewskii*)

Remarks: Over 120 species of *Haemoproteus* have been reported mostly from wild waterfowl, passerine birds and some other orders of birds. Chickens are not affected by this parasite. Vectors are hippoboscid flies and biting midges (*Culicoides* spp.) for some species. Gamonts may be demonstrated in stained bloodsmears and schizonts in the endothelial cells of the blood vessels of the lungs. Schizonts may be found in the liver, spleen and kidneys. Antimalarial drugs may be used for treatments (☞ above *Plasmodium* spp.).

(Figure 674)

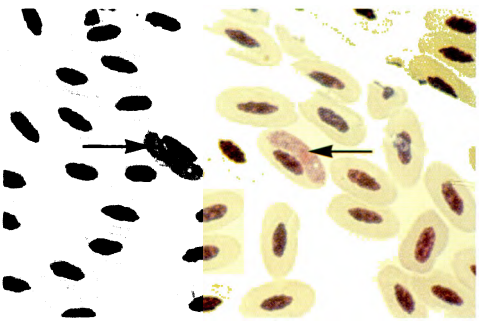

Fig. 674 *Haemoproteus* sp.; gamonts (arrows); stained bloodsmear (1000 ×) [4]

Leucocytozoon spp. (*L. caulleryi* and *L. sabrazesi*)

Location: Gamonts occur in the red blood cells and white blood cells which are often distorted by the large size of the organism.

Hosts: Many wild and domestic birds are affected by these parasites.

Species description: Parasites of this genus undergo schizogony in the endothelial cells and parenchymatous cells of the liver, heart, kidney and other organs of avian hosts. Large schizonts are produced. The gamonts occur in the circulating blood, and the infected host cells become grossly distorted and assume a spindle-shape. The

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vectors are black flies (*Simulium* spp.) or biting midges (*Culicoides* spp.). Of the numerous *Leucocytozoon* species many are restricted to a certain group or order of birds.

Geographic distribution: *Leucocytozoon* spp. occur world-wide. *L. caulleryi* and *L. sabrazesi* were reported from chickens and guinea fowls in Asia.

Symptoms: Some strains of *L. caulleryi* are non-pathogenic and others are highly pathogenic, killing a high percentage of chickens in a flock. Affected chickens are anaemic, listless, diarrhoeic, have pallid combs and wattles and have marked haemorrhage in their lungs, livers and kidneys. There may be gross haemorrhage from the kidney lesions into the peritoneal cavity. *L. sabrazesi* causes a disease in chickens characterized by anaemia, pyrexia, diarrhoea, paralysis of the legs and a ropy discharge from the mouth.

Significance: *Leucocytozoon* spp. may cause considerable losses in infected flocks.

Diagnosis: In stained bloodsmears (Giemsa or Brilliant-cresyl blue) gamonts may be seen. The shape of the gamont varies with the species, some are elongated while others are rounded. There are no pigment granules. Haemorrhages, splenomegaly and hepatomegaly are seen at necropsy. Grossly visible white dots in many organs are the meronts.

Therapy: Treatment usually is not effective. Pyrimethamine (1 ppm) and sulfadimethoxine (10 ppm) administered simultaneously may prevent but not cure infections of *L. caulleryi*. Pyrimethamine (25 ppm) was recommended for therapy. Furazolidone (150 ppm, po.) may be used to avoid clinical symptoms.

Prophylaxis: Control requires eliminating the arthropod vector from the environment of the host. Insecticidal sprays and repellents sprayed within houses may be used to reduce the insect populations.

(Figure 675)

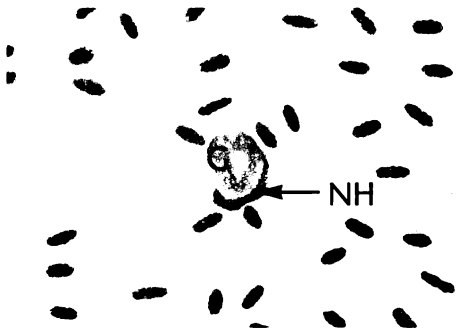

Fig. 675 *Leucocytozoon* sp.; gamont (G) in an erythrocyte (1000 ×). The nucleus of the host cell (NH) is peripheral; stained bloodsmear [4]

RICKETTSIALES

Aegyptianella spp. (*A. pullorum* and *A. moshkovskii*)

Location: The pathogens occur as initial bodies followed by development forms and marginal bodies in the cytoplasm of erythrocytes ("signet-ring").

Hosts: Chicken, turkey, duck, goose and other birds

Species description: The organisms are transmitted by *Argas persicus* and probably other tick species. Transmission by blood inoculation is possible. The incubation period is 12–15 days. The early trophozoites or initial bodies occur in erythrocytes, are small (0.5–1.0 µm) and round to oval. Spherical bodies up to 4 µm may occur, containing up to 25 small granules. The clinical condition is often complicated by fowl spirochaetosis which is also transmitted by *Argas persicus*.

Geographic distribution: North and South Africa, Asia, southern Europe, UIS

Symptoms: Ruffled feathers, anorexia, droopiness, diarrhoea and hyperthermia may be found. Anaemia, jaundice, enlargement of the liver and the spleen, yellow-green kidneys and punctiform haemorrhage of the serosa may be seen at necropsy.

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Significance: Indigenous poultry rarely suffer the acute disease, but freshly introduced stock may die within a few days.

Diagnosis: This is based on the demonstration of organisms in Giemsa-stained blood-smears. Intraerythrocytic forms (marginal bodies) and extraerythrocytic forms may be seen in leucocytes, lymphocytes, monocytes but also in the plasma.

Therapy: Tetracyclines (oxytetracycline, chlortetracycline, 15–30 mg/kg, po.) are effective.

Prophylaxis: ☞ POULTRY, ■ 5.1 TICK CONTROL (Figure 676)

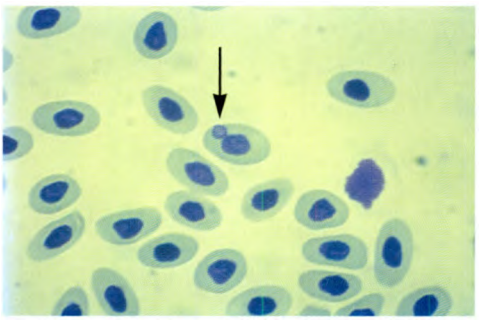

Fig. 676 *Aegyptianella pullorum*; the organism forms a marginal body in the cytoplasm of erythrocytes (arrow) (1000 ×) [13]

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3 Stages in the urogenital system

HELMINTHS

- Trematoda found in the urogenital system

***Prosthogonimus* spp. (*P. pellucidus* (syn. *cuneatus*), *P. ovatus* and other *Prosthogonimus* sp.) Oviduct fluke**

Location: Bursa of Fabricius, oviduct, posterior intestine

Hosts: Chicken, duck, goose and many species of wild birds

Species description: The adult flukes are 8–12 mm long and bear two suckers. The eggs measure 22–27 × 13–18 µm, are operculated and bear a small spine opposite to the operculum. They are passed in the faeces. The first intermediate hosts are aquatic snails and the second are the nymphal stage of various species of dragonflies. Metacercariae may persist in the insect until the final host is infected by eating either the adult dragonfly or the nymphal stage. In the final host the liberated immature trematodes migrate to the cloaca and the bursa of Fabricius where they become adult. In the mature chicken the parasites enter the oviduct.

Geographic distribution: World-wide

Symptoms: The disease usually occurs seasonally (spring and summer in temperate areas). Several hens begin to lay eggs with soft shells or no shells at all. There is marked discharge from the cloaca, consisting chiefly of lime and the feathers around the cloaca are soiled with albumen which may contain the parasites. The birds become listless, the abdomen is pendulous and the legs are held widely apart during walking. Laying is suspended and the birds are obviously ill. If peritonitis develops, the comb and wattles become cyanotic and the birds die. The trematodes may also occur within the deposited eggs.

Significance: *Prosthogonimus* spp. are among the most pathogenic trematodes of poultry. Chickens are mainly affected but occasionally also ducks. The fluke causes marked inflammation of the oviduct, the production of abnormal eggs and discharges of albumen from the cloaca. Peritonitis often develops by retroperistaltic movements of the oviduct.

Diagnosis: Large numbers of parasite eggs may be found in the discharges from the cloaca. Sometimes the parasites may be found in the abdominal cavity at necropsy.

Therapy: Praziquantel (5–10 mg/kg, po.) and mebendazole (10–50 mg/kg, po.) may be used to control *Prosthogonimus* infections.

Prophylaxis: Affected flocks should be kept away of the intermediate hosts. Fencing in chicken flocks prevents access to lake shores where chickens may acquire infection by eating dragonfly naiads.

(Figures 630, 677, 678, 679)

Fig. 677 *Prosthogonimus cuneatus* (syn. *P. pellucidus*); stained [4]

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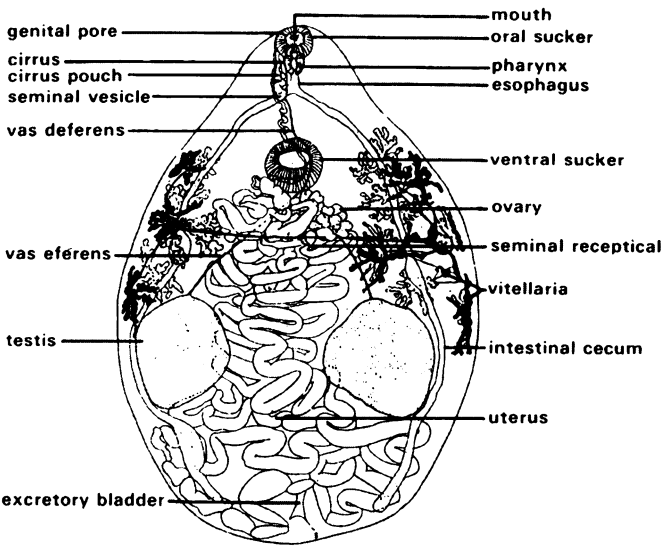

Fig. 678
Prosthogonimus macroorchis (8–12 mm long) [56]

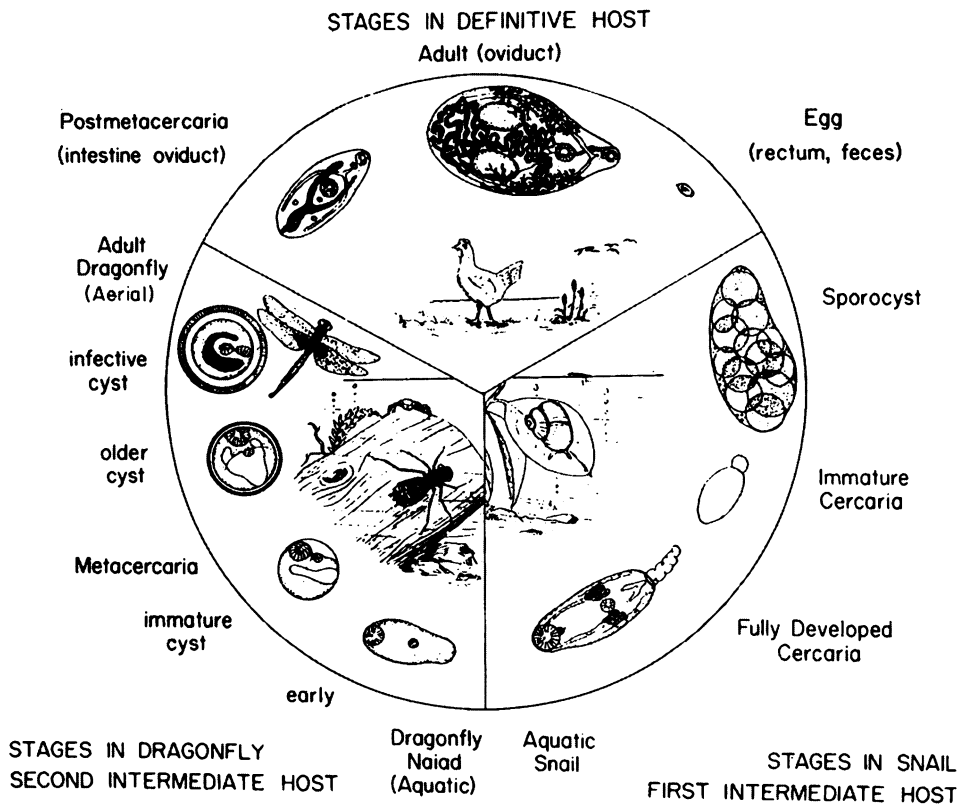

Fig. 679 Life cycle of *Prosthogonimus* sp. [56]

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4 Stages in internal organs

4.1 Locomotory system

4.1.1 Muscles

4.2 Liver

4.3 Respiratory system

4.1 Locomotory system

4.1.1 Muscles

PROTOZOA

Toxoplasma gondii

Location: Intracellular cysts with a diameter of 50 µm occur in muscles and several other tissues of chickens.

Hosts: Cats and other members of the Felidae are definitive hosts. Many mammals including all domestic animals act as intermediate hosts (☞ SWINE, ■ 4.1).

Species description: Infection is acquired orally by ingestion of sporulated oocysts from faeces of the final host (☞ SWINE, ■ 4.1).

Geographic distribution: World-wide

Symptoms: Natural infections are often clinically inapparent. The chicken is quite resistant to *T. gondii* but disease has been observed from both natural and experimental infections. Clinical signs in chickens include anorexia, paleness, emaciation, reduced egg production, diarrhoea, incoordination, ataxia, trembling, opisthotonus, torticollis and blindness. Mortality may be as high as 50%.

Significance: In chickens only sporadic outbreaks of *T. gondii* have been reported.

Diagnosis: This is usually difficult and based on finding the cysts in the muscle using the artificial digestion method. It is also possible to employ serological tests (complement fixation, haemagglutination or indirect immunofluorescent antibody tests) but the most convincing diagnosis is the isolation of the parasite by inoculation of suspect material into mice. Impression smears of peritoneal fluids or tissues stained with Giemsa, or tissue sections of brain, liver, spleen, lungs and lymph nodes may reveal the organisms by direct microscopic examination.

Therapy: This is generally not indicated.

Prophylaxis: Prevention of avian toxoplasmosis requires management practices that eliminate the source of infected tachyzoites and oocysts, by preventing exposure to cats and coprophageous arthropods. The destruction of oocysts which are disseminated throughout the premises is impossible.

Sarcocystis spp. (*S. horvathi, Sarcocystis* sp. and *S. rileyi*) Sarcosporidiosis

Location: Muscles of the breast, thigh, neck and oesophagus

Hosts: Chicken, duck and many other domestic and wild birds

Species description: Two species occur in chickens: *S. horvathi* and *Sarcocystis* sp. *S. rileyi* mainly occurs in ducks but it has also been reported from chickens. Sarcocysts of *S. rileyi* are elongate, with their long axis parallel to the muscle fibres. They are whitish and appear spindle-shaped when removed from the musculature. Sarcocysts are divided into compartments, each of which contains numerous banana-shaped cystozoites (bradyzoites). Cysto-

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zoites are 8–15 µm long and 2–3 µm wide. The life cycle is not completely known but dogs and cats may act as final hosts. Chickens are infected by ingesting oocysts or sporocysts via faeces of the final hosts (cats or dogs).
Sarcocystis spp. found in chickens: *S. horvathi*: sarcocyst: 1–10 mm, cystozoites: 9–12 × 2.5–3 µm and final host: unknown; *Sarcocystis* sp. sarcocyst: 1–10 mm, cystozoites: 14–17.5 × 2–2.5 µm and final host: dog and cat; *S. rileyi* sarcocyst: 3–5 mm, cystozoites: 8–15 × 2–3 µm and final host: unknown.

Geographic distribution: World-wide

Symptoms: Infections with *Sarcocystis* spp. in chickens are generally inapparent. Muscular dystrophy may occasionally occur.

Significance: Sarcosporidiosis may be prevalent in traditional poultry rearing systems. The pathogenicity and significance of this parasite seems to be low.

Diagnosis: This is based on demonstrating the Sarcocysts or cystozoites in artificially digested muscles and other organs at necropsy.

Therapy: Unknown and not indicated

Prophylaxis: This is difficult in extensive husbandry systems with free ranging. Intensive poultry rearing avoids the ingestion of oocysts and sporocysts of the final hosts (possibly dogs, cats and other carnivores).

4.2 Liver

PROTOZOA

Histomonas meleagridis "Blackhead"

Remarks: Extensive liver lesions may occur. These are circular, yellowish green, and characteristically depressed. In turkeys they may be up to 4 cm in diameter. The liver and caecal lesions together are pathognomonic. However, the liver lesions must be differentiated from those of tuberculosis, leukosis, avian trichomonosis and mycosis, which are raised and greyish or grey-yellow (☞ POULTRY, ■ 1).

4.3 Respiratory system

PROTOZOA

Remarks: The following parasites may also be found in the tissue of the lungs:
Trichomonas gallinae (☞ POULTRY, ■ 1)
Toxoplasma gondii (☞ SWINE, ■ 4)
Cryptosporidium spp. (☞ POULTRY, ■ 1)
Schizonts of the following Protozoa:
Sarcocystis spp. (☞ POULTRY, ■ 4.1)
Haemoproteus spp. (☞ POULTRY, ■ 2)
Leucocytozoon spp. (☞ POULTRY, ■ 2)
Plasmodium spp. (☞ POULTRY, ■ 2)

HELMINTHS

- **Nematoda found in the respiratory system**

Syngamus trachea "Gapeworm"

Location: Trachea and lungs

Hosts: Chicken and many other domestic and wild birds

Species description: The parasite is bright red in colour and found permanently in copulation. The male is 2–6 mm long and the female 5–20 mm. The mouth opening is wide, without leaf-crowns, and the buccal capsule is cup-shaped. The eggs measure 70–100 × 43–46 µm and have a thickened operculum at either pole. Infection may occur directly by ingestion of infective eggs or larvae. However, it is well established that severe field infections are associated with the ingestion of transport hosts such as earthworms, snails, slugs, and arthropods (e.g. flies). Gapeworm larvae may encyst and survive for years within an invertebrate host. Range infections occur seasonally, e.g. when great numbers of earthworms appear on the surface by rains in spring or at the onset of the rainy season, etc. *Cyathostoma bronchiale* is the causative agent of the disease in geese and ducks.

Geographic distribution: World-wide

Symptoms: Sudden death and verminous pneumonia characterize early outbreaks. Signs of gasping, choking, shaking of the head, inanition, emaciation and suffocation may follow.

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Significance: This may be of great economic significance in range-reared chickens. Young birds are affected most severely. Gapeworms are not a problem in confinement-reared poultry but cause serious losses in range-reared chickens, turkeys and other birds.

Diagnosis: This is based on the demonstration of the typical eggs in the faeces and the clinical signs. At necropsy adult gapeworms may be found to obstruct the lumina of the trachea and bronchi. The blood-red female gapeworm is usually found in copulation with a much smaller, paler male with its head embedded deep in the host tissue. The joined pair has a "Y"-shaped appearance.

Therapy: Febantel (60 ppm via feed over 6 days or 15 mg/kg, po. over 2 days), fenbendazole (100 ppm, over 4 days), flubendazole (30 ppm, over 7 days) and mebendazole (60 ppm, over 7 days) are effective against gapeworms in chickens. Ivermectin may be used orally at a dosage of 200 µg/kg. Thiabendazole (300 mg/kg, po., 1 ×) is also effective.

Prophylaxis: The control of gapeworm infections in range-reared chickens is almost impossible since the transport hosts are difficult to be eliminated. To prevent wild birds from introducing infection, pens should be isolated by overhead and lateral screening. The control of the transport hosts by soil treatments with chemicals is unsafe and not justifiable.

(Figures 680, 681, 682, 683)

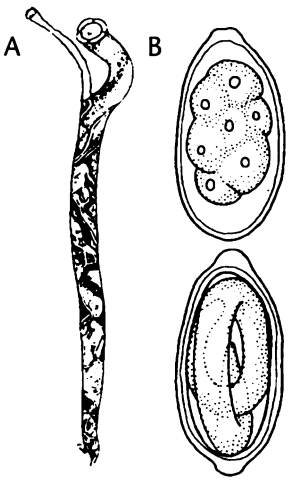

Fig. 681 *Syngamus trachea*; male and female worms in permanent copulation (A); eggs (B) [56]

Fig. 682 *Syngamus trachea*; a joined pair with a Y-shaped appearance [8]

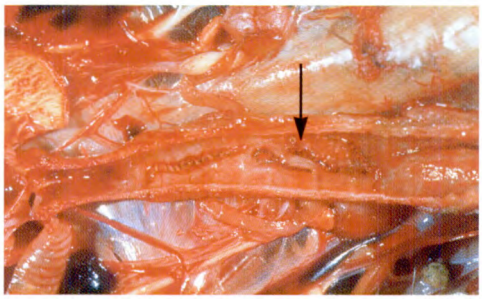

Fig. 680 *Syngamus trachea*; adult parasites in the trachea. Males are 2–6 mm and females are 15–30 mm long [53]

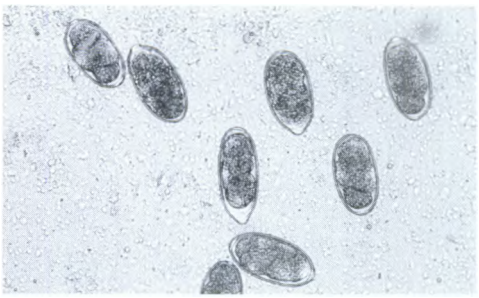

Fig. 683 Eggs of *Syngamus trachea* (74–95 × 39–44 µm)

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ARTHROPODS

- Arachnida found in the respiratory system

Cytodites nudus The "air sac mite"

Location: Respiratory passages (air sacs)
Hosts: Chicken, pigeon and other birds
Species description: The "air sac mite" lives in the respiratory passages and air sacs and sometimes also in other organs (abdominal cavity, e.g. serosa side of liver and kidneys). The males are 450–575 µm long and 280–340 µm wide, and the females are 480–600 µm long and 315–400 µm wide. Little is known about the life cycle but larvae (not eggs!) are produced by the female. Parasites are spread by respiratory mucus. The parasite has a moderate pathogenicity. It occurs mainly in range-reared small holder poultry systems and does not occur in intensive rearing systems. The mites are easily transmitted between the birds.
Geographic distribution: World-wide
Symptoms: Light infections are often inapparent. Heavy infections are accompanied by cough, dyspnoea (open beak), pneumonia, bronchitis, peritonitis.
Significance: The significance of this parasite is still in debate. Heavy infestations may predispose to secondary respiratory disorders.
Diagnosis: This is based on the demonstration of the mites on smears taken from the respiratory system at necropsy. The mites may be seen macroscopically as little white dots on the surface of the air bags.
Therapy: The elimination of affected or exposed birds has been the recommended method of control. Ivermectin was effective in golden pheasants only at very high doses (50 mg/kg, sc.).
Prophylaxis: Unknown
(Figures 684, 685, 686)

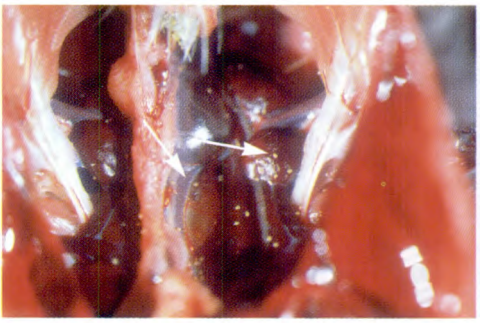

Fig. 684 *Cytodites nudus*; adult mites (450–600 µm) found in the air sacs (arrows) [53]

Fig. 685 *Cytodites nudus*; adult mite [53]

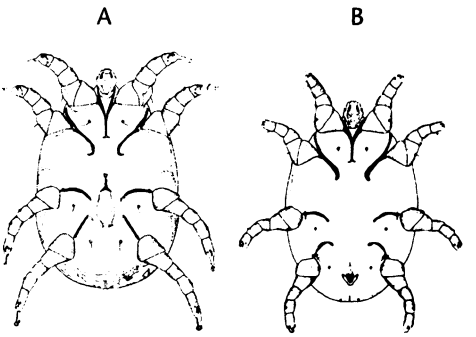

Fig. 686 *Cytodites nudus*; ventral view of adult female (A) and male (B) [29]

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5 Stages on the body surface

5.1. Skin and feathers

5.2 Eyes

5.1 Skin and feathers

HELMINTHS

- Trematoda found in the subcutaneous tissue

Collyriclum faba

Location: These trematodes appear anywhere on the body as subcutaneous cysts.

Hosts: Chicken, turkey and other bird species

Species description: The subcutaneous cysts are 4–6 mm in diameter, usually containing two adults and occur most frequently near the vent of the host. The worms measure 3–5 × 4.5–5.5 mm. They are flattened ventrally, convex dorsally and have a spiny tegument. The eggs measure 19–21 × 9–11 µm. The cysts ooze exudate which attracts flies and predisposes to bacterial infections. The life cycle is unknown but probably involves snails and insects such as dragonflies and mayflies as intermediate hosts. Only birds with access to marshy places become infected.

Geographic distribution: Asia, Europe, the Americas, Africa

Symptoms: Young birds show locomotory difficulties, inappetence, anaemia, emaciation and even death.

Significance: Infections with this trematode may be frequent in flocks which have access to marshy places. The presence of the cysts decreases the value of the carcass.

Diagnosis: Typical cysts are found around the cloacal opening and also along the abdomen and thorax. Each cyst has a central opening and contains a pair of worms. The cysts also contain a black fluid and the eggs which are discharged through the pore.

Therapy: Chemotherapy is unknown. The parasites should be removed surgically followed by an antiseptic treatment of the wound.

Prophylaxis: Prevention requires restricting birds from areas frequented by aquatic insects.

ARTHROPODS

The ectoparasites (Arthropods) are divided into two main groups:

Arachnida (arachnids) including:
- *Ticks*
- *Mites*

Insecta including:
- *Lice*
- *Heteroptera*
- *Fleas*
- *Dipterida*

- Arachnida found in/on the skin

– Ticks

Losses caused by ticks are threefold: 1) loss of blood (anaemia), 2) transmission of other diseases (avian spirochaetosis, rickettsial diseases, viruses, etc.) and 3) tick toxicosis (e.g. paralysis)(☞ CATTLE, 1).

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IXODIDAE ("hard ticks")

Hard ticks are not a primary problem in chickens. Birds are preferred hosts of larvae and nymphs of some species of *Hyalomma* and *Amblyomma* ticks that are common in the adult stage on mammals. *Haemaphysalis cinnabarina*, *H. leporispalustris*, *H. chordeilis*, *Amblyomma haebraeum* and other hard ticks of many species will feed on poultry as well as wild ground birds. Hard ticks are likely to be found only on birds that have access to range (bushy pastures and pens are preferred sites of hard tick stages). Mainly young birds are affected. Extreme emaciation, progressive weakness, restlessness and swollen eyelids may be seen in young chickens. Control is based on the use of contact-acaricides, (☞ *Argas persicus*, below) and preventingchickens from entering breeding areas of the ticks.

ARGASIDAE ("soft-ticks")

Argas persicus, A. walkerae, A. reflexus hermanni and other *Argas* spp. Fowl ticks

Location: Skin

Hosts: Chicken, turkey, pigeon, duck, goose and many other domestic and wild birds; man may also be affected.

Species description: *Argas persicus* is the most important poultry ectoparasite in many tropical and subtropical countries, being a limiting factor in successful rearing of standard breeds of poultry.

Unfed ticks are relatively easily recognized by their flattened ovoid shape and the reddish-brown colour.

The fowl tick is capable of transmitting *Borrelia anserina*, *Aegyptianella pullorum*, fowl cholera (*Pasteurella multocida*) and possibly other diseases. These ticks are particularly active in poultry houses during warm and dry weather. All stages may be found hiding in cracks and crevices during the day. Larvae can be found on the birds since they remain attached and feed for 2–7 days. Nymphs and adults feed at night in 15–30 min. Nymphs feed and moult several times before reaching the adult stage. Adults feed repeatedly and the females lay 50–100 eggs after each feeding. Adult females may live for more than 4 years without a blood meal. *A. persicus* and *A. reflexus hermanni* are found in chickens in West Africa. *A. reflexus hermanni* in Egypt is suspected of transmitting West Nile and *Chenuda* virus and the *Quaranfil* virus group.

Geographic distribution: *A. persicus* occurs world-wide in tropical and subtropical countries. *A. walkerae* occurs in sub-Saharan Africa.

Symptoms: In addition to transmitting avian spirochaetosis, aegyptianellosis and fowl cholera, *Argas* spp. produce anaemia (most important). Loss of blood may reach proportions of fatal anaemia. Low numbers may cause emaciation, weakness, slow growth and lowered production. Since the ticks are nocturnal, the birds may show some uneasiness when roosting. *A. persicus* and *A. walkerae* may cause tick paralysis, a flaccid, afebrile motor paralysis. A specific paralytic toxin is suspected to be transmitted by the sucking tick.

Significance: Soft-bodied ticks of the genus *Argas* are the most important ticks of poultry.

Diagnosis: Red spots may be seen on the skin where the ticks have fed. Larvae may be found in the feathers. The adult parasites may be found in cracks of the woodwork or in the walls of the fowl house.

Therapy and Prophylaxis: This requires treatment of premises because adult and nymphal ticks are on their hosts for only a short time and then hide in the surroundings. After houses are cleaned, walls, ceilings, cracks, and crevices should be treated thoroughly with carbaryl, coumaphos, malathion, stirofos or a mixture of stirofos and dichlorvos. The efficiency may be increased by using high pressure sprayer (☞ Table 27, p. 389).

(Figures 687, 688, 689, 690)

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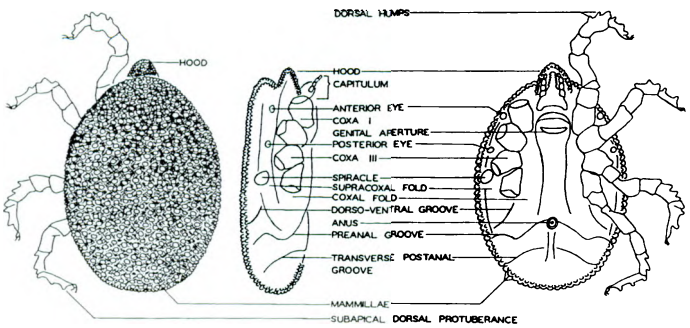

Fig. 687 *Argas* sp.; dorsal (left), lateral (middle) and ventral view (right) [24]

Fig, 688 *Argas reflexus*; adult female (5.5–11 mm long) and eggs [8]

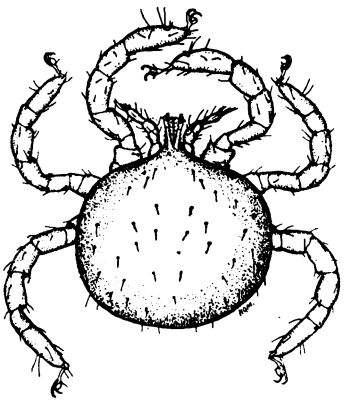

Fig. 690 *Argas persicus*; larva [3]

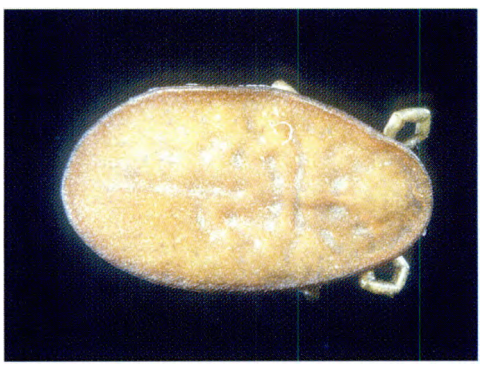

Fig. 689 *Argas persicus*; dorsal view (males are 4–5 mm and females are 7–10 mm long) [8]

Ornithodoros moubata The eyeless tampan of Africa

Remarks: *O. moubata* is widely distributed throughout East Africa and northern South Africa, extending into drier parts of Central Africa. *O. moubata* and *O. savignyi* have become associated with man and his domestic animals. These ticks live in the native huts and in the sand under trees where animals and human beings frequently seek shelter. The significance is based on the irritation of the host and transmission of *Borrelia anserina* and *Aegyptianella pullorum* to fowl. *O. moubata* is also the vector of *Borrelia duttoni* (relapsing fever of man) and African Swine Fever (☞ SHEEP AND GOATS, ■ 5.1).

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Ornithodoros savignyi The eyed tampan, the sand tampan

Remarks: It buries itself in sandy and loose clayey soils, under trees, near wells and shady spots frequented by domestic stock, particularly camels, cattle, mules, chickens and man on which it feeds. It does not occur in huts. The numbers of *O. savignyi* in infested localities reach plague proportions. The biology is very similar to that of *O. moubata*, but is has a much wider geographic distribution occurring in the arid parts of Africa, the Near East, India and Ceylon. *O. savignyi* is not known to transmit any pathogen but camels and cattle suffer greatly and may even be killed by the volume of blood lost.

– Mites

Dermanyssus gallinae Red mite, roost mite, poultry or chicken mite

Location: Mites may be found in the chicken houses during the day, particularly in cracks or where roost poles touch supports or on birds during the night.

Hosts: Chicken, turkey and other domestic and wild birds

Species description: The nymphs and adults periodically visit the host to suck blood (preferably during the night) and between the meals hide in cracks and crevices in the quarters of the birds. Under favourable conditions the mites reproduce rapidly and may become a serious pest causing irritation and anaemia due to loss of blood. A house may remain infested for 6 months after birds are removed. Mites may be transmitted by mite dispersion (egg flats, crates, persons going from farm to farm) or by direct bird contact. The chicken mite can be identified by the shape of the dorsal plate and by the long whip-like chelicerae that appear to be stylets. The adult females measure 0.7 × 0.4 mm, varying in colour from gray to deep red, depending on their blood content.

Geographic distribution: World-wide

Symptoms: Birds become listless, egg production decreases and deaths may occur because of loss of blood. *D. gallinae* may occur as a temporary parasite on humans, causing skin lesions.

Significance: Chicken mites may be found at pest level in traditional, small farm flocks and are rare in modern commercial cage-layer systems. Populations may build up very quickly in warm areas. Losses may be enormous due to irritation, anaemia and markedly reduced performance.

Diagnosis: This is based on finding the mites in the environment of the birds (nest, litter, crevices). The mites can be seen with the naked eye, especially when they are red after a recent blood meal. Masses of mites may be found in the naso-pharyngeal system of dead birds.

Therapy and Prophylaxis: ☞ CONTROL OF CHICKEN MITES AND TROPICAL-FOWL MITES INFESTATIONS, p. 382

(Figures 691, 692, 693, 694)

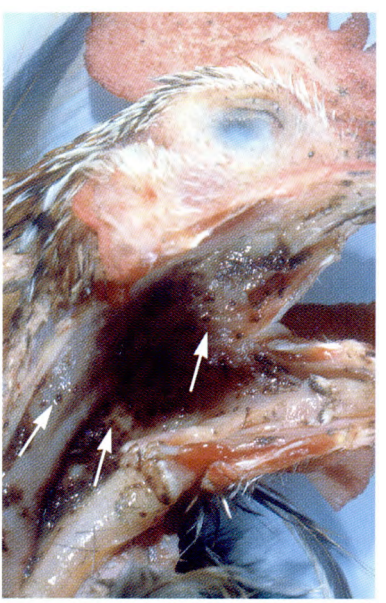

Fig. 691 *Dermanyssus gallinae*; masses of mites are often found in the naso-pharyngeal system of dead birds (arrows) [53]

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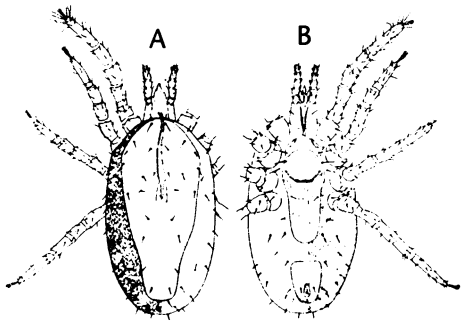

Fig. 692 *Dermanyssus gallinae*; dorsal view (A)

Fig. 693 *Dermanyssus gallinae*; adult mite (600–1000 µm long) [4]

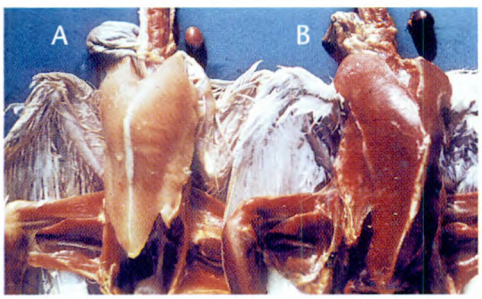

Fig. 694 Anaemia due to heavy *Dermanyssus gallinae* infestation (A); not-infested control animal (B) [54]

Ornithonyssus (syn. *Bdellonyssus*, *Liponyssus*) ***bursa*** Tropical fowl mite

Location: *O. bursa* occurs either on the bird or in its nest.

Hosts: Chicken, turkey, duck, pigeon and other birds

Species description: The tropical fowl mites are found on the fluff of the feathers, especially on those around the vent. These mites can pass their entire life cycle on chickens. They tend to be present on few feathers, giving them a dirty appearance. The life cycle is not yet completely known. *O. bursa* can also affect man but only temporarily because it is unable to survive away from its bird host. Only the adults, the protonymphs and the deutonymphs feed on the hosts blood. *O. bursa* replaces *O. sylviarum* (northern fowl mite) in warmer parts of the world.

Geographic distribution: Tropical and subtropical regions

Symptoms: Irritation, loss of weight, reduction in egg production, anaemia and death

Significance: Apart from irritating the birds and causing anaemia these two mites may transmit infections (e.g. fowlpox, Newcastle disease, *Pasteurella* spp., etc.). This might be of fundamental importance in intensified poultry rearing systems.

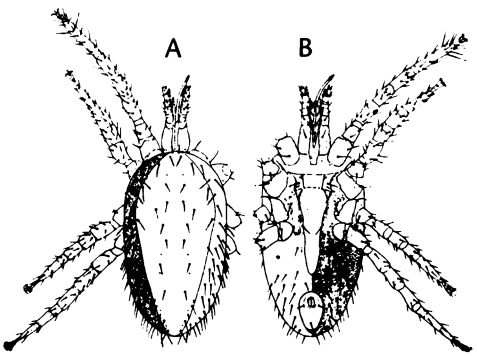

Fig. 695 *Ornithonyssus bursa*; dorsal view (A) and ventral view (B) [56]

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Fig. 696 *Ornithonyssus bursa*; adult (450–650 µm long) [53]

Diagnosis: This is determined by finding them on the feathers of affected birds. The distribution of the mites on the birds may be patchy, hundreds being found in small areas.

Therapy and Prophylaxis: ☞ CONTROL OF CHICKEN MITES AND TROPICAL-FOWL MITES INFESTATIONS.

(Figures 695, 696)

- **Control of chicken mites and tropical-fowl mites infestations**

Control of chickens infested with the tropical fowl mite (and also with *Ornithonyssus sylviarum*, the northern fowl mite) may be achieved by spraying the birds and litter with an acaricide such as carbaryl, coumaphos, malathion, stirofos or a pyrethroid compound. The acaricide spray treatments must be applied with sufficient force to penetrate the feathers in the vent area. For chicken mite control, in addition to treating the birds, the inside of the house and all hiding places for the mite, such as roosts, behind the nest boxes, cracks and crevices must be treated thoroughly using a high-pressure sprayer. Dimethoate and fenthion, permethrine, flumethrine and amitraz may be used as residual house sprays when poultry is not present (☞ Table 27, p. 389). (Figure 697)

Fig. 697 Heavy infestation with unfed *Dermanyssus gallinae*

Cnemidocoptes mutans Scaly leg mites

Location: These mites are usually found under the scales of the legs.

Hosts: Chicken and turkey

Species description: *C. mutans* is a small spherical, sarcoptic mite that usually burrows into the tissue under the scales of the legs. It is rare in modern poultry enterprises. It usually occurs on older birds on which the irritation and exudation cause the legs to become thickened, encrusted and deformed. This is the result of a massive hypertrophy of the stratum corneum (dermatitis hypertrophicans). The mites reach the feet of the birds from the ground. The lesions usually develop from the toes upwards. The parasites pierce the skin underneath the scales, causing an inflammation with exudate that hardens on the surface and displaces the scales. The entire life cycle is spent on the skin and mites are transmitted by direct contact.

Geographic distribution: World-wide

Symptoms: Thickened scaly skin leading to lameness and malformation of the feet

Significance: This may be a serious problem in traditionally reared chickens.

Diagnosis: This is based on the typical deformation of the legs (scaly legs) and the demonstration of mites in skin scrapings.

Therapy: If individual birds are to be treated, the affected legs may be dipped twice (10-

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day interval) in kerosene, mineral oil or linseed oil, or coated with vaseline. It is important that the crusts are carefully removed or soaked with a detergent prior to acaricidal treatment. The legs may also be dipped in acaricides.

Prophylaxis: Affected birds should be culled or isolated and houses cleaned and sprayed as recommended for chicken mite infestations.

(Figures 698, 699)

Fig. 698 Deformed legs due to *Cnemidocoptes mutans* infestation [53]

Fig. 699 *Cnemidocoptes mutans*, the scaly leg mite; males are 170–250 µm and females are 370–495 µm long [4]

Cnemidocoptes gallinae (syn. *Mesocnemidocoptes laevis gallinae*) Depluming mite or depluming itch

Location: The mites burrow into the skin alongside the shafts of the feathers on the back, head, neck, around the vent and on the breast and tights.

Hosts: Chicken, pheasant, pigeon and goose

Species description: These mites resemble the scaly leg mite in general structure although they are smaller, the adult females measuring about 0.3 mm in diameter. The mites occur more frequently in summer when infestations are spread rapidly by contact. Infestations cause inflammation and itching. The feathers break off and they are pulled out by the birds.

Geographic distribution: World-wide

Symptoms: The intense irritation induces the host to pull out body feathers. Infested birds show reduced weight gains and lowered production.

Significance: Heavy infestations are associated with great irritation and reduced performance of affected flocks. This infection is rare in modern poultry-rearing systems.

Diagnosis: Demonstration of the mites at the base of the feathers. This can be made by pulling out a few feathers at the edge of the lesions and searching for mites. (10% KOH-solution). The legs and pedicels are stumpy. All legs of the male possess suckers, while none of the female do so.

Therapy and Prophylaxis: Affected birds should be isolated and dipped in an acaricide (malathion, carbaryl, permethrin, stirofos). Disinfection of houses as recommended for chicken mites is important (☞ Table 27, p. 389).

Laminosioptes cysticola Cyst mite, subcutaneous mite

Location: Subcutaneous tissue

Hosts: Chicken and turkey

Species description: The parasites are found in the subcutaneous tissue where they cause calcified nodules around dead parasites.

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The life cycle is unknown.

Geographic distribution: World-wide

Symptoms: Infestations are usually inapparent.

Significance: These mites are non-pathogenic but large numbers of nodules may reduce the value of the carcass.

Diagnosis: Examination of the skin and subcutis under a dissecting microscope usually reveals the mites.

Therapy and Prophylaxis: This is unknown. Destroying affected birds is indicated to reduce the infestations within the flocks.

(Figures 700, 701)

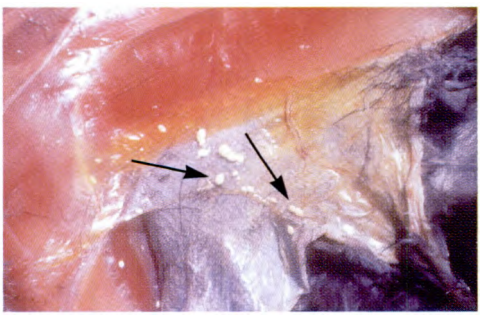

Fig. 700 *Laminosioptes cysticola*; calcified nodules in the subcutaneous tissue (arrows) [53]

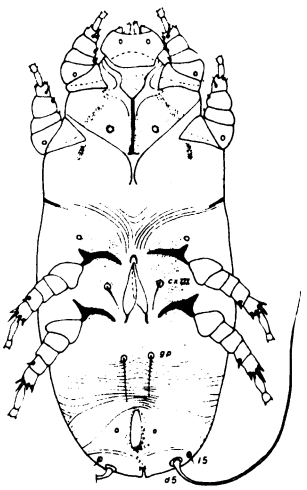

Fig. 701 *Laminosioptes cysticola*; ventral view of a female (250–270 µm long) [29]

Fig. 702 *Epidermoptes bilobatus*; skin mite (160–270 µm long) [56]

Epidermoptes bilobatus Skin mite

Location: These mites are found on the skin surface. Predilection sites are head, neck, breast, back and sometimes the whole body surface.

Hosts: Chicken

Species description: These mites are very small, the females measuring 0.17–0.22 mm. This is a common skin parasite of chicken and can cause a scaly skin disease. It has been described as a cause of pityriasis. It may produce lesions such as a fine scaly dermatitis, followed by a formation of thick, brownish, sharply edged scabs. Concomitant fungus infection by *Lophophyton gallinae* may aggravate the disease. Birds with *E. bilobatus* infection often have depluming mites at the same time. Bacterial infections may develop after the primary skin lesions.

Geographic distribution: World-wide

Symptoms: Pruritus, featherlessness, emaciation, even death

Significance: Losses in heavily infested flocks may be great.

Diagnosis: This is based on the demonstration of mites in skin scrapings.

Therapy: The following acaricides may be used to dip or spray infested chickens: Pyrethroids (fenvalerate, flumethrine, permethrine), organophosphates (trichlorfon, cyth-

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ionate) and carbamates (carbaryl, propoxur) (☞ Table 27, p. 389).

Prophylaxis: All the animals of a flock must be treated at the same time and the chicken quarters should also be disinfected.

(Figure 702)

Hypodectes propus Nest-inhabiting mites

Remarks: This is a free living nest-inhabiting mite which produces eggs which develop directly into hypopodes. These stages invade transiently the subcutaneous tissue of birds and grow markedly in size. By the time the hypopodes leave the host they have increased tenfold in length. After leaving the host the hypopodes moult directly into the adult stage.

Symptoms: Itching, feather loss, restlessness

Diagnosis: By finding the adults in the nest or the nymphes II in alterated tissue

Therapy and Prophylaxis: Nests should be treated with acaricides and infested birds should be eliminated.

(Figure 703)

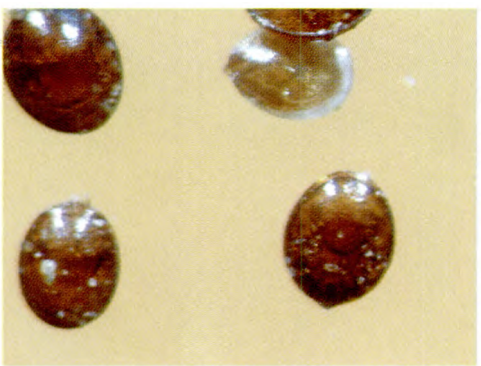

Fig. 703 *Hypodectes propus*; environmental stage of nest-inhabiting mites

- **Insecta found on the skin**

– **Lice**

MALLOPHAGA Chewing lice, chicken lice

Location: Skin and feathers

Hosts: Chicken

Species description: All chicken lice belong to the order Mallophaga, the chewing lice. More than 40 species of Mallophaga have been reported from domestic chickens. Chewing lice are characterized by possession of broad, chewing-type mandibles located ventrally on the head, incomplete metamorphosis, no wings, dorsoventrally flattened body and short antennae with 3–5 segments. Birds frequently harbour several species at the same time. All the various lice species are controlled by the same methods. Cross contamination is possible, e.g. lice from guinea fowl may occur on chicken and turkeys if these birds have physical contact. Pigeon lice are frequently found on domestic fowl if the pigeons nest above the fowl. Lice spend the entire life cycle on the birds. Eggs are attached to the feathers, often in clusters, and require 4–7 days to hatch. The more common louse species of chickens are listed in Table 26. Lice normally eat feather products but *Eomenacanthus stramineus* may consume blood and is regarded as the most common and most destructive louse of birds, world-wide. It is up to 3.5 mm long and deposits its eggs in masses at the base of feathers, especially around the vent. As a rule, lice are not very pathogenic to mature birds but louse-infested chicks may die. Lousiness frequently accompanies manifestations of poor health such as internal parasitism, infectious disease, malnutrition as well as poor sanitation.

Geographic distribution: World-wide

Symptoms: Restlessness, emaciation, feather damage, anaemia, markedly reduced performance

Significance: Lice infestation in chickens is a very important cause of production losses in traditional and intensive poultry rearing systems.

Diagnosis: Lousiness (pediculosis) is diagnosed by finding the straw-coloured lice on skin or feathers of birds. Lice of domestic birds vary in size from less than 1 mm to over 6 mm. Masses of eggs may be found attached to the bases of the feathers.

Therapy: Lice are best controlled by spraying with

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pyrethroids, carbaryl, coumaphos, dichlorvos, malathion and stirofos (☞ Table 27, p. 389).

Prophylaxis: To avoid cross-contamination, lice must be controlled on all the birds in the environment of chickens at the same time. Wild galliform or birds should never be allowed to contact poultry flocks. If treatment is required, the birds should be treated 2 times on a 10-day interval. Only the mature forms will be controlled as most of the available insecticides do not kill the eggs. Spraying of the birds is generally the most efficient way of controlling lice. Dipping is also possible when the flocks are small. Egg-laden feathers will remain a source of reinfestation and therefore thorough clean-up of all the facilities and the environment should be carried out. (Figures 704, 705)

Fig. 705 Eggs of *Eomenacanthus stramineus* at the base of feathers (arrows) [53]

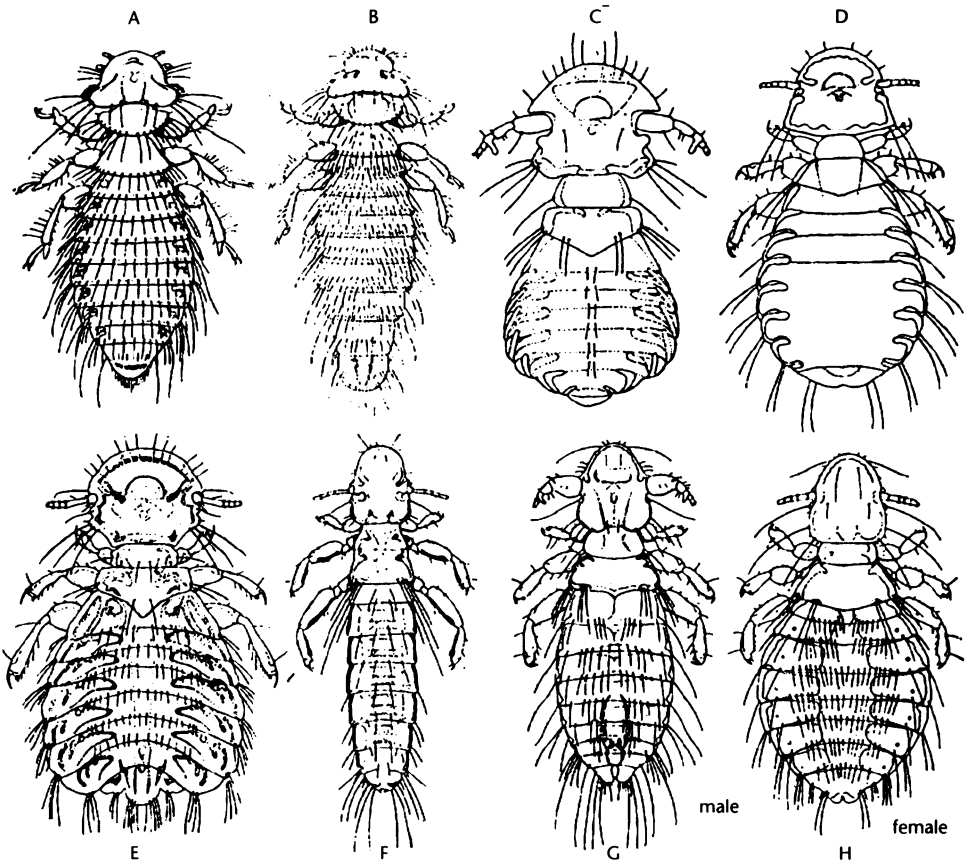

Fig. 704 Chewing lice (Mallophaga) of chicken; *Menopon gallinae* (A), *Eomenacanthus stramineus* (B), *Goniodes* (syn. *Oulocrepis*) *dissimilis*, the brown chicken louse (C), *Goniocotes gallinae* (D), *Goniodes* (syn. *Stenocrotaphus*) *gigas* (E), *Lipeurus caponis* (F) and *Cuclotogaster heterographus* (G and H) [35]

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Table 26 Mallophaga of chickens, guinea fowls and pigeons

Scientific name	Common name	Host
Menoponidae		
Eomenacanthus stramineus	Body louse	Chickens
Menopon gallinae	Shaft louse	Chickens
Lipeurus caponis	Wing louse	Chickens
Numidilipeurus tropicalis	Tropical wing louse	Chickens
Philopteridae		
Cuclotogaster heterographus	Head louse	Chickens
Goniocotes gallinae	Fluff louse	Chickens
Goniodes dissimilis	Brown chicken louse	Chickens
Goniodes gigas	Tropical brown chicken	Louse
Goniodes numidae	Feather louse	Guinea fowl
Lipeurus numidae	Slender louse	Guinea fowl
Campanulotes bidentatus	Small pigeon louse	Pigeons
Columbicola columbae	Slender pigeon louse	Pigeons

(Figures 706, 707, 708, 709, 710, 711, 712)

Fig. 707 Mallophaga found in a feather

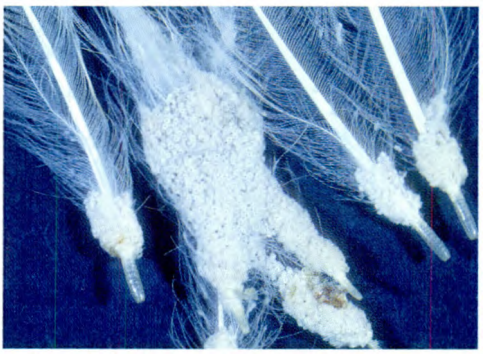

Fig. 708 Masses of eggs of *Menopon gallinae* at the base of feathers

Fig. 706 *Eomenacanthus stramineus*, the body louse (2.9–3.2 mm long) [4]

Fig. 709 *Menopon gallinae*, the shaft louse (1.5–1.9 mm long) [8]

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Fig. 710 *Lipeurus caponis*, the wing louse (2.0–2.5 mm long) [8]

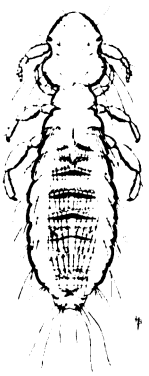

Fig. 711 *Cuclotogaster heterographus*, the head louse (2.4–2.5 mm long) [56]

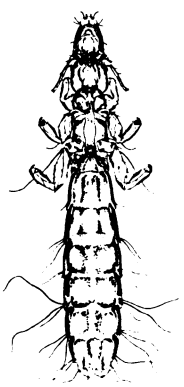

Fig. 712 *Columbicola columbae*, the slender pigeon louse (2.0–2.8 mm long) [56]

– Heteroptera

Cimex lectularius Common bedbug

Remarks: The bedbug is a common blood-sucking parasite in temperate and subtropical climates that attacks poultry, most mammals, including man. Feeding usually occurs at night. The bugs become engorged within 10 minutes, then hide in cracks and crevices. If attacked by large numbers of bugs, birds may become anaemic and performance is severely reduced (decreased egg production, high feed consumption). Heavily infested nests are abandoned. Bites are usually followed by swelling and itching. Bedbugs are rare in modern laying operations, but traditional poultry systems are often heavily affected. Control is based on thorough cleaning of the houses and quarters and the reduction of hiding places for the bugs. A thorough high-pressure spraying of the houses as for the control of fowl ticks is effective. Pyrethroids, organophosphates, carbamates and combinations are highly effective against Heteroptera (☞ Table 27 p. 389).
(Figure 713)

Fig. 713 *Cimex lectularius*, the common bed-bug (3–6 × 3 mm) [4]

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Table 27 Some insecticides for external parasite control on poultry

Lice, mites, fleas	
Dusts	
Malathion (4–5%)	Ready-to-use
Permethrin (0.25%)	Ready-to-use
Rabon	Ready-to-use
Carbaryl (5%)	Ready-to-use
Sprays	
Malathion	0.5% spray
Permethrin	0.05% spray
Tetrachlorvinphos*	0.5% spray
Stirofos (23%) and dichlorvos (5.7%)	0.5% spray
Fowl ticks, bedbugs	
Sprays	
Malathion	0.3% spray
Tetrachlorvinphos*	1.0% spray
Carbaryl	0.2% spray

* not directly on poultry

– Fleas

SIPHONAPTERA

Fleas are parasites in the adult stage but free-living as larvae. Adults vary in size from 1.5–5 mm, and possess a tough, laterally compressed body, piercing-sucking mouthparts and long legs for leaping. They are cosmopolitan in distribution but more abundant in temperate and warm climates. Females develop several eggs per day which roll off the host into surrounding litter where they incubate. Dampness and warmth is essential for further development. Larvae develop within 1–2 weeks which feed on organic matter. The larvae then pupate and the inactive pupal stage varies from 1 week to several months.
(Figure 714 and p. 332 ff.)

Fig. 714 *Ceratophyllus gallinae* in situ

Echidnophaga gallinacea Sticktight flea

Remarks: The sticktight flea is common on chickens and many other bird and mammal species in tropical and subtropical areas of the world. Adults usually attach to the skin of the head for days or weeks. The sessile adults often occur in clusters of 100 or more. The adults forcibly eject their eggs so that they reach surrounding litter. The larvae best develop in sandy, well-drained litter. Irritation and blood loss cause anaemia, reduced performance and death, particularly in young birds.
(Figure 715)

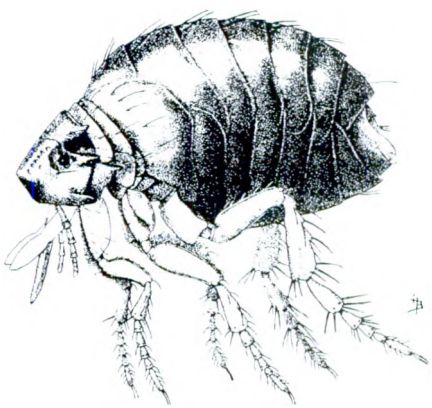

Fig. 715 *Echidnophaga gallinacea*, the sticktight flea (about 1 mm long) [56]

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Ceratophyllus gallinae Common chicken flea, European chicken flea

Remarks: This is the common chicken flea with a world-wide distribution. It occurs on chickens, many other bird species, rodents, dog and man. It breeds in nests and litter, and only goes on the birds to feed.
(Figure 716)

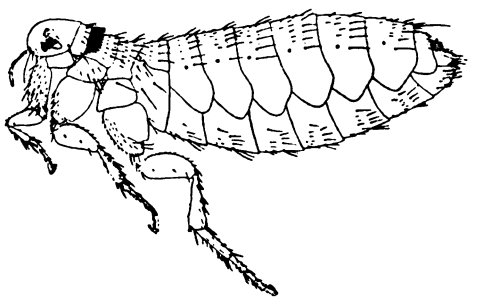

Fig. 716 *Ceratophyllus gallinae*, the common chicken flea (2.0–3.5 mm long) [56]

Ceratophyllus columbae

Remarks: This flea occurs on domestic pigeon and chickens.

Ctenocephalides felis

Remarks: It has been found in poultry houses at pest levels. Most of these houses are using cats as rodent control and it appears that the cats introduce the fleas in the house and these then move to the birds. Pulex irritans, the human flea, may occasionally also attack chickens.

- **Control of fleas**

The most important control measures are removing infested litter and dusting the litter surface with carbaryl, coumaphos or malathion to kill immature fleas.

– Dipterida

The order Dipterida includes several families whose members annoy or suck blood from birds (Table 28).

CULICIDAE Mosquitoes

Aedes spp., *Anopheles* spp. *and Culex* spp.

Remarks: A great number of mosquitoes is known to feed on poultry. Only females suck blood. Adults are most active towards the evening and at night. Poultry production facilities that utilize lagoons can have problems with mosquitoes breeding in the lagoon. Masses of mosquitoes may attack poultry and numerous deaths within a flock may occur. Several viruses are known to be transmitted by mosquitoes, e.g. fowl pox virus by *Aedes* spp. The best control is prevention of mosquito development in the environment of the chicken. Mosquitoes breed in swamps, ponds, stagnant pools and water-filled containers of all types. Such breeding grounds should be covered or eliminated (e.g. draining swampy areas). For housed poultry residual wall sprays and fogging within poultry houses may reduce the mosquitoes markedly. Screening to prevent mosquito entry may aid in control. Poultry in open houses or on range are most difficult to protect from mosquitoes (☞ CATTLE, ■ 5.1; Table 28).

SIMULIIDAE

Simulium spp. Blackflies, buffalo gnats

Remarks: Blackflies are bloodsuckers and transmit leucocytozoonosis to duck, turkey and other birds. Many species occur in tropical areas. They are similar in size to mosquitoes but are dark, short, and humpbacked with short legs. They often attack in swarms and cause great irritation, anaemia and death of birds either directly or through disease transmission. Blackfly control is extremely difficult since imma-

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ture stages are restricted to running, well-aerated water, which is often some distance from the poultry farm. Screening may aid to control attacks. Measures recommended for mosquito control are applicable to blackfly control (☞ CATTLE, ■ 5.1; Table 28).

CERATOPOGENIDAE

Culicoides spp. Biting midges

Remarks: Many species of *Culicoides* attack chickens. These are extremely small although they can easily be seen as small blackish specks moving on the skin. Their bites cause marked irritation and intense itching. Some species transmit diseases (e.g. avian infectious synovitis, *Haemoproteus nettionis* to duck, possibly also fowl pox). Control is extremely difficult since the midges will pass through ordinary screen mesh, but screens treated with malathion (6%) solution have eliminated midges for 3 weeks. Fogging with mosquito or fly sprays and residual deposits applied for fly control will help to reduce the problem. Since the habitats where these species develop are so variable, it is difficult to destroy the midges on the breeding ground (☞ CATTLE, ■ 5.1; Table 28).

MUSCIDAE

Musca domestica Housefly and its relatives

Remarks: These non-biting flies are readily produced on poultry farms and are a health and sanitation problem for both the poultry producers and neighbours. These flies do not bite or attack birds directly, but their significance is associated with the transmission of diseases. Many species may be involved. So-called filth flies are a world-wide problem on poultry farms with many other species of *Musca* and indigenous blowflies (Calliphoridae) and flesh flies (Sarcophagidae) involved. Filth flies lay their eggs in manure (some sarcophagids deposit living larvae), in moist spilled feed or on dead bird carcasses. Larvae of the housefly may develop in manure and then move to drier areas for pupation. Flies are suspected to be vectors of many mammalian as well as avian gastrointestinal diseases. Newcastle disease virus was found in flies. Houseflies and maggots may act as intermediate hosts for the tapeworm *Choanotaenia infundibulum*. Common houseflies and blowflies are capable of carrying eggs of the caecal worm *Heterakis gallinae*, which may contain the protozoan agent of histomonosis of turkeys. Certain fly larvae feed on decomposing cadavers and may ingest the toxin of the bacterium *Clostridium botulinum*. If poultry eat such maggots, botulism ("Limberneck") may occur. Prompt burial, burning or the use of disposal pits for animal cadavers will prevent botulism from such sources. Houseflies may also transmit fowl cholera, tuberculosis (*Mycobacterium avium*) when ingested by chickens and turkeys. Myiasis is not as common as in mammals. The black blowfly (*Phormia regina*) can deposit eggs in wounds in chickens, turkeys and geese and the maggots may destroy living tissue. Fly control is based on preventing breeding of flies in manure. Flies cannot breed in dry manure (moisture < 60%). Sufficient air flow should be provided over the manure. Water leaks may support fly breeding and should therefore be avoided. Insecticides may be used in several ways: as space sprays, fogs and mists (temporary control of adult flies), surface sprays (residual effect of insecticides on surfaces), baits (insecticide granules combined with fly attractants, e.g. muscamone are placed in protected areas and are highly effective). The use of larvicidal insecticides on manure is unsafe and detrimental to predators and other organisms in the manure. Insect growth regulators (cyromazine) are highly effective and may circumvent this problem (☞ CATTLE, ■ 5.1; Table 28).

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Stomoxys calcitrans Stable fly

Remarks: The stable fly attacks mammals and birds. This fly is similar in size and appearance to the common housefly, but possesses a piercing beak. Both sexes suck blood and cause intense irritation and anaemia when present in high numbers. It develops in manure with high fibre content or in wet crop refuse such as straw left in the field or other crop residues. Control is by the same measures used against houseflies. Prevention requires clean-up of crop residues and proper manure management to prevent mixing of moist manure with spilled feed. The stable fly acts as intermediate host of *Hymenolepis carioca* (☞ CATTLE, ■ 5.1).

Table 28 Fly control in poultry facilities

Residual and bait sprays	
Permetrin	0.25% spray
Stirofos	1% spray
Tetrachlorvinphos*	1% spray
Contact sprays	
Dibrom	0.25%
Pyrethrin (0.75%) and piperonylbutoxide (0.5–3.75%)	Ready-to-use oil spray
Dichlorvos	0.5% spray
Dry sugar baits	
Dibrom	Ready-to-use bait
Trichlorfon (1%)	Ready-to-use bait
Dichlorvos (1%)	Ready-to-use bait
Methomyl (Golden malrin, 1%)	Ready-to-use bait
Bomyl (True Grit Blue, 1%)	Ready-to-use bait

* not directly on poultry

HIPPOBOSCIDAE

Pseudolynchia canariensis The pigeon fly

Remarks: This is an important parasite of pigeons in warm or tropical areas. It may transmit *Haemoproteus columbae*, which causes pigeon malaria. Larvae mature inside the female and pupate immediately upon being ejected. The adult fly is dark brown and about 6 mm in length. The adult fly moves rapidly through the feather and sucks blood. The fly may also bite humans, inflicting a painful skin wound that persists for several days. Infested birds suffer from blood loss and irritation.
(Figure 717)

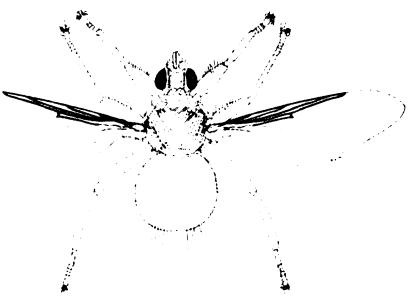

Fig. 717 *Pseudolynchia canariensis*, the pigeon fly (up to 6 mm long) [3]

5.2 Eyes

HELMINTHS

- Nematoda found in the eyes

Oxyspirura spp. (*O. mansoni, O. parvorum and O. petrowi*) Manson's eyeworm

Location: This worm occurs under the nictitating membrane and in the conjunctival sacs and nasolacrimal ducts.
Hosts: Chicken and guineafowl
Species description: This slender nematode is 12–18 mm long. The eggs pass down the lacrimal ducts and out in the faeces of the birds. The intermediate stages develop in the cockroach (*Phycnoscelus surinamensis*). The fowls acquire the parasite by ingesting infected cockroaches. Larvae migrate along the oesophagus, pharynx, and lacrimal ducts to the eye.
Geographic distribution: Tropical and subtropical regions
Symptoms: Infected birds show a peculiar oph-

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thalmia. They appear uneasy and continuously scratch at the eyes, which are usually watery and show severe inflammation. The nictitating membrane becomes swollen, projects slightly beyond the eyelids at the corners of the eyes. It is usually kept in continual motion. The eyelids are sometimes stocked together and a white cheesy material collects beneath them. Severe ophthalmia may develop and the eyeball may be destroyed. The worms are seldom found in severely affected eyes.

Significance: *Oxyspirura mansoni* is rarely found but may severely affect individual birds.

Diagnosis: Embryonated eggs (50–65 × 45 µm) are passed in the droppings. Adult worms may be found under the nictitating membrane and in the conjunctival sacs.

Therapy: Some drops of a 10% solution of levamisole (applied topically) kill the parasites as does levamisole (30 mg/kg, po.). Topical application of ivermectin (0.05 mg/kg) may also be effective against the parasites.

Prophylaxis: Strict sanitary measures, including the use of insecticides on cockroach-infested premises, provide efficient control. (Figure 718)

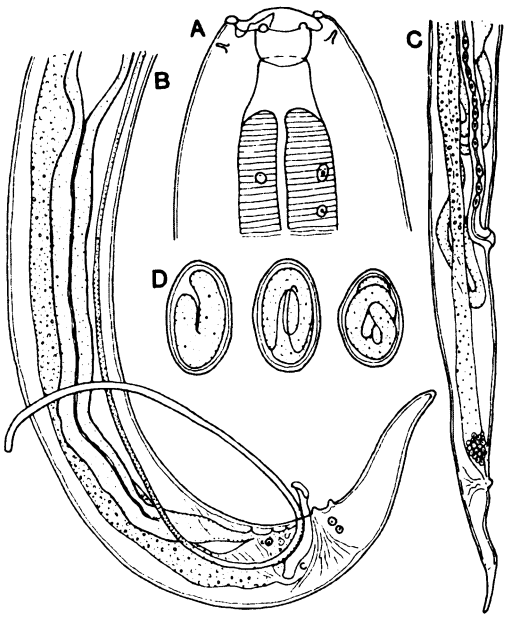

Fig. 718 *Oxyspirura mansoni*, anterior end (A), posterior end (B) of male, posterior end of female (C) and eggs (D) [56]

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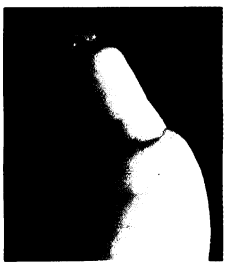
Johannes Kaufmann

Parasitic Infections of Domestic Animals



A Diagnostic Manual

This unique manual is intended as a tool for the identification and control of the wide spectrum of parasites affecting domestic animals throughout the world.

Systematic, impressively comprehensive and beautifully illustrated, the book allows the user to steer swiftly through a vast amount of information. More than 300 color and 400 black and white illustrations show the pathogens and the clinical symptoms caused by parasites, facilitating a rapid and accurate diagnosis of the species involved in any particular case. Furthermore, the profile of each and every affliction includes a list of adequate measures of therapy and prophylaxis.

Parasitic Infections of Domestic Animals is of great value for personnel in field laboratories, veterinarians and technicians, as well as for teachers and students. On another practical level, it is relevant for meat inspectors and other public health officials to identify parasites in domestic animals which are potentially harmful to humans.

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