Female Sexual Function and Dysfunction

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ISBN 978-3-319-41714-1 ISBN 978-3-319-41716-5 (eBook)
DOI 10.1007/978-3-319-41716-5

Library of Congress Control Number: 2017932279

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Foreword

I am proud and pleased to present the first edition of this book entitled *Female Sexual Dysfunctions*. The goal of this book is to disseminate the state-of-the-art, scientific, evidence-based information on the study, diagnosis, and treatment of women's sexual health concerns.

An Eastern myth tells a story about blind men and an elephant, in which a group of blind men touch an elephant to learn about it. Each one feels a different part, but only one part. They then compare notes and realize that they are in complete disagreement. Only when they stop talking and start listening, collaborating, and comparing notes are they able to see the whole elephant. The story teaches us that although one's subjective experience is true, it might not be the complete truth. So is female sexual dysfunction (FSD) the sexological elephant? The debate is not new, but opinions remain polarized. A crucial paradigm for understanding the etiology and treatment of sexual problems is to view sexuality and sexual function and dysfunction in a biopsychosocial context, meaning that we must recognize that different factors influence a woman's sexual life to get the full picture of her sexuality. The critical voices argue against the existence of female sexual dysfunctions, making accusations of disease-mongering and suggesting that the pharmaceutical industry are medicalizing female sexuality and creating a problem that does not exist. However, their opponents argue that real feminism is to give women with distressingly low sexual desire an opportunity to receive medical treatment just as men are able to do.

While the debate is going on, 30–50 % of women report low desire and 12 % report that the problem is distressing. Furthermore, the prevalence of low desire increases with age, but the level of distress decreases. For years, only one pharmacotherapeutic treatment—testosterone therapy—has been available for low sexual desire in surgically postmenopausal women. Female sexual dysfunctions have traditionally been treated with sex therapy, relationship therapy, and other psychotherapeutic approaches, which are often good treatments but are not always successful. Pharmacological treatments are, therefore, justified—especially if the underlying problem is biological. Very little research has investigated the effect of psychotherapeutic approaches, and discussions of what constitutes good end points when evaluating the effect of treatment are lacking. Is the aim to increase sexual activity, to achieve a subjective feeling of increased desire or empowerment, or to gain a deeper understanding of what promotes and prevents a good sex life? Many of these soft end points might be important for the woman but are difficult to measure in a clinical trial.

Could we imagine having the same discussion about treating male low desire with testosterone or using phosphodiesterase type 5 (PDE5) inhibitors for erectile dysfunction? What is the difference? Clinical experience with testosterone has shown us that selected women clearly benefit from medical treatment and that some women benefit from flibanserin therapy; however, the existence of a distinct placebo effect in clinical trials strongly indicates the additional importance of psychological factors. However, despite these data, some people argue that women will feel under pressure to seek unwanted medical treatment for their sexual problem, with the implication that men can be trusted to make a rational decision regarding the risk versus reward of receiving treatment, whereas women cannot. Thus, even in the twenty first century, we still approach women's and men's sexuality differently. These disparate approaches to male and female sexuality are despite substantial changes in the attitude of Western culture towards female sexuality over the past 150 years. From women's sexuality being considered something that did not exist or was wrong and immoral if it was expressed, through to the new attitudes that arose during the 1960s to 1970s when women were considered to have the same right to orgasm as men, men and women were claimed to be sexually equal, and women could control pregnancy and their own sexuality. Today's situation is an increased focus on sexual desire as something expected to be present throughout life, and the concept that sexuality is one of the cornerstones in keeping couples together. The concepts of and attitudes to female sexuality have continuously evolved, much more so than our attitudes towards men's sexuality, with men being seen as having a strong, stable, and often biologically driven sexuality that is not doubted. Furthermore, the participants are arguing like the blind men from the myth: depending on what specialty, experience, theoretical tradition, and experience we come from, our concepts of female sexuality and dysfunction are disparate, and we interpret the female sexual dysfunction elephant differently. Thus, we miss an important opportunity to see the whole elephant and address all aspects of female sexuality. Such challenges, especially when combined with the disagreement between different academic and clinical opinion leaders—all of whom claim to represent women—are harmful, as we miss the opportunity to embrace and understand all aspects of female sexuality, how it is formed and expressed, how it functions, what is important for the individual woman, and how we can best help women who seek help for a better sexual life. Once again, as we fail to work together, we are repeating the mistakes of the blind men. However, unlike an elephant, female sexual dysfunction is not a well-defined and well-delineated phenomenon, and it is experienced differently from one woman to another; in fact each woman perceives herself and her situation individually and should be consulted and treated based on her needs. To provide women with the best possible care, we need more research into all aspects of female sexuality and treatment options, and following the example of the blind men, we must finally start listening, collaborating, and comparing notes.

Vincenzo Mirone
Italian Society of Urology,
Turin, Italy

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Sociocultural Considerations

1

Donata Villari

Ἔρος δ' ἐτίναξέ μοι
φρένας, ὡς ἄνεμος κὰτ ὄρος δρύσιν ἐμπέτων.
Saffo – Ereso – 630 avanti Cristo – Leucade 570 avanti Cristo

Eros shakes my mind
like a mountain wind falling on oak trees

Paul Julius Moebius –1835–1907
Über den phisiologische Schwachsinn des Weibes 1900

Nature holds the maiden in the obscure vision of her instincts. Her repugnance for men, the repulsion that inspires her sensuality, appears to the consciousness of the virgin as absolute, enduring feelings … The better a girl is, the more firmly she is convinced that she has no desire… and that her longings should be turned towards the ideal.

"The Mental Inferiority of Woman," 1900, preface to the third edition

. .. yes and how he kissed me under the Moorish wall and I thought well as well him as another and then I asked him with my eyes to ask again yes and then he asked me would I yes to say yes my mountain flower and first I put my arms around him yes and drew him down to me so he could feel my breasts all perfume yes and his heart was going like mad and yes I said yes I will Yes.

from Molly Bloom's monologue in James Joyce, Ulysses – Paris, 1922

Since the 1970s the term "sexual medicine" has become common usage [1]. Interestingly and appropriately enough, the introduction of the 2012 *ESSM Syllabus of Sexual Medicine* [2] stresses that the use of the term "sexual", understood, however, simply as an adjective referred to sexual or gender identity, was borrowed from studies of botanical taxonomy at the beginning of the nineteenth century [3]. And even after being transferred to the context of a discipline that analyzes human sexuality, the term was at first employed exclusively in the study of reproduction.

The term "sexology" appeared for the first time in Elizabeth Osgood Goodrich Willard's 1867 work, *Sexology as the Philosophy of Life*: *Implying Social*

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_1

Organization and Government [4]. And the mathematician Karl Pearson, one of the founding fathers of modern statistics and a convinced believer in eugenics, in the 1888 inaugural lecture of the Men and Women's Club, which he had founded, entitled "The Woman's Question," stressed the need for a "real science of sexology" [5].

The Italian Paolo Mantegazza (1831–1910), physiologist and pathologist, a visionary writer who was among the first to spread Darwin's theories in Italy, published *La fisiologia dell'amore* (*The Physiology of Love*) (1873), *L'Igiene dell'amore* (The Hygiene of Love) (1877), *La fisiologia del piacere* (*The Physiology of Pleasure*) (1880), and *La fisiologia della donna* (*The Physiology of Woman*) (1893). He dedicated himself to important studies in neurophysiology and pharmacology based on animal models, treating topics absolutely in the vanguard for his time, among which were female sexuality, male and female infertility, masturbation, erectile dysfunction, and vaginismus [6].

But it was the dermatologist Iwan Bloch in his 1907 work, *Das Sexualleben unserer Zeit in seinen Beziehungen zur modernen Kultur* [7], who was the first to stress the importance of a multidisciplinary approach for those who intend to do in-depth studies of the "life of love," by integrating knowledge from diverse fields such as biology, anthropology, philosophy, psychology, sociology, ethnology, and medicine, a concept that is completely shared nowadays.

At the start of the 1900s, this "new science" seemed to be something chaotic and vague. It was in this field that Freud came onto the scene as precursor and interpreter of his times. Still, his theory of female sexuality was strongly conditioned by the ethical principles and the customs of the society of his day, despite the transgressive and groundbreaking questions his theories introduced (we need only think here of childhood sexuality).

Freud has no organic theoretical work specifically dedicated to the female psyche in the sexual sphere; what exist are mainly clinical cases or fragmentary theories. By his own admission, the origin and development of female sexuality remained an inextricable enigma, which led him, despite his charisma, to collaborate with contemporary women analysts [8].

While the myth of Oedipus remains a cornerstone in the psychosexual history of the male individual, it is attributed to the woman in its "reciprocal" form (reverse Oedipus), though with various interpretative complications and a marked asymmetry that led Freud himself to state that at the very best in the female sex "Oedipus" can never be completely overcome. Freud held that the penile substitute, the clitoris, was initially invested with strong focalization and very intense sensations but that afterward, in the so-called genital phase, these sensations were transferred to the vagina, and this represented the achievement of psychosexual maturity. In this way the clitoris was disinvested, losing its importance for orgasm, to the point that continued clitoral orgasm was interpreted as evidence of a neurosis, synonymous with fixations and regression to the pregenital phase.

Recent psychophysiological studies disclaim Freud's theory of female sexuality. Indeed, current analytical interpretation has reappraised and reformulated his hypotheses, with the consequence that the opposition between clitoral and vaginal orgasm is no longer accepted.

As we have already noted, an important contribution to the study of female psychosexual development has been given by the work of the first women analysts, the

Danish Lampl-de Groot (1895–1987) and the American Ruth Mack Brunswick (1897–1946), though in a perspective that has since been accused of "female misogyny." They went more deeply into the questions of the passive-active sex role, the conflictual mother-daughter relationship, and female castration in the process of working through the negative Oedipus [9]. Helene Deutsch (1884–1982) adopted the Freudian theory of the natural masochism of women [10], while Karen Horney (1885–1952), whose convictions were influenced by sociology and anthropology, countered with an explicative model of female development strongly influenced by social and cultural elements [11].

In 1974 Luce Irigaray identified the foundation of female sexual identity in the mother-daughter relationship [12]. This first love is banned in the patriarchal order, which relegates it to an aphasic function of identification. Irigaray went on to demand women's right to diversity, to self-love and the love of other women, without effacing themselves in the competition to win the sexual favors of men [13].

Going back to the father of the psychoanalytic approach to sexuality, in the end Freud proposed two types of femininity that are possible to find in adult women: one is altruistic, maternal, masochistic, and receptive, while the other is narcissistic, seductive, autoerotic, and not ready to love authentically – two contrasting and practically incompatible prototypes.

This dichotomy of femininity, which in a certain sense creates a gap between the generative-maternal and the erotic roles, underlies all the subsequent great battles for emancipation in industrialized countries in the twentieth century. However, in the end it has proven difficult to achieve a widespread sentiment of "interior" liberty, free from the age-old internalized identity/role models (mother/wife). Doubtlessly there has been progress in women's "rights" and in safeguarding their health and their maternity. But at the same time, the difficult climb to achieve equal opportunities in pay and positions in important social roles is still steep and uneven [14].

What is evident is that the disciplines that study sexual behavior, both male and female, have undergone a revolution in their scientific and cultural approaches in recent decades and that this is especially true as regards female sexual dysfunction (FSD). Nowadays one concept stands out clearly – female sexuality is complicated. It cannot be totally closed within a context of anatomical and physiological districts; rather, it has to be understood and contextualized in all its manifestations, which involve the biological, psychological, and sociocultural spheres. All of these have to be set in a scenario that does not underestimate economic, racial, and religious aspects, as well as differing access to resources and the presence in the world of conflicts and wars that unfortunately lead to systematic sexual violence. Indeed, even today over 135 million women worldwide have had to undergo the ritual procedure of infibulation.

The borderline between what was once considered "anomalous" and what is felt to be "normal" today is continually shifting. This is reflected in the change over the years of the system of classification of FSD that has partly revolutionized its nosography [15]. Since 1952, in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders (DSM*), many versions have been published, characterized by successive updating [16–21]. The "Consensus Development Conference on Female Sexual Dysfunction," organized by the American Foundation for Urological Disease in 1998 and followed by the one of the same name in 2003 [22–24], while maintaining some continuity with

the criteria of the DSM-IV-TR 2000 [21] and of the ICD-10 1992 [25], has introduced important novelties [22–24].

The receptive/motivational desire model introduced by Deborah Bateson in 2001, characterized by its *circularity*, repositions sexual response as the result of the modulation of interactions between the brain and the genital area through positive or negative feedback [23, 24]. The concept of "personal distress" is also introduced. By this is meant the emotional repercussions in terms of feelings of frustration felt by women due to and in relation to a sexual dysfunction. This concept represents an attempt to get beyond certain critical aspects of the previous classifications. The most important of these are the following:

- The description of male and female sexual dysfunctions as specular phenomena
- The representation of female arousal as a series of phases following a "linear model" that does not really fit it [26]
- The problematic distinction between the phases of desire and arousal
- The neglect of emotional and interpersonal aspects and of prior and current sexual experiences
- The exclusive reference to a heterosexual orientation in women living in a long-term couple relationship

Perhaps even today no effort at the classification of FSDs and the distress they cause fully encompasses such a highly complex issue. What is certain is that any future changes in definition will have to be supported by evidence-based research, though the basic domains that have already been identified (desire, arousal, orgasm, and pain) will undoubtedly continue to be cardinal fields of reference [27].

The role of sexual health as an essential prerequisite for a person's well-being, happiness, and development is universally recognized today [28, 29]. But sexual health, far beyond simply meaning the absence of disease (which, however, in many parts of the world is still a goal), requires universal awareness and promotion.

We are light-years away from the times when for primitive man the moon was the visible representation of the woman, silent and mysterious symbol of the genuine receptive essence of the feminine, in antithetical contrast with the active, bright solar essence that represented man [30]. In our day, men and women have to discover and experience a repositioning of their roles, including their sexual roles, from a perspective of mutual respect and sharing. What is more, a reconsideration of possible gender orientations also involving the sexual sphere is necessary.

References

1. Riley A. The birth and development of sexual medicine: reflections of my personal journey. J Sex Med. 2007;4:815–21.
2. Porst H, Reisman Y, editors. The ESSM syllabus of sexual medicine 2012 written by the ESSM Educational Committee. Amsterdam: Medix Publischers. 2012.
3. Hirschfeld M. Über sexualwissenschaft. Zeeitschr Sexualwiss. 1908;1:1–19.

4. Sexology as the philosophy of life: implying social organization and government. Chicago: J. R. Walsh; 1867.
5. Walkowitz JR. Science, feminism and romance: The Men and Women's Club 1885–1889.
6. Paolo Mantegazza *La fisiologia dell'amore* (1873 – Libreria Editrice G. Brigola – Milano) *Igiene dell'amore* (1877 – Libreria Editrice G. Brigola – Milano) *La fisiologia del piacere* (1880) *La fisiologia della donna* (1893 – Treves – Milano)
7. Bloch I. Das sexualleben unserer Zeit in seinen Beziehungen zur modernen Kultur. Berlin: Marcus Verlagsbuchhandlung; 1907.
8. Sigmund F. Drei Abhandlungen zur Sexualtheorie. Lipsia e Vienna: Franz Deuticke; 1905.
9. Ogilvie MB, Harvey JD. The biographical dictionary of women in science; 2000.
10. Psychoanalysis of the sexual functions of women. Leipzig/Wien/Zürich: Internationaler Psychoanalytischer Verlag; 1925.
11. Feminine Psychology (reprints), Norton, 1922–37. 1967.
12. Irigaray L. Speculum of the other woman; 1974 (Eng. trans. 1985 by Gillian C. Gill).
13. Irigaray L. This sex which is not one; 1977 (Eng. trans. 1985).
14. The Global Gender Gap Report 2014, in http://reports.weforum.org/global-gender-gap-report-2014/
15. Leiblum S. Classification and diagnosis of female sexual disorders. In: Goldstein I, Meston C, Davis SR, Traish A, editors. Women's sexual function and dysfunction. Study, diagnosis and treatment. 1st ed. London: Taylor.
16. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. DSM-I. 1st ed. Washington, DC: American Psychiatric Press; 1952.
17. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. DSM-II. 2nd ed. Washington, DC: American Psychiatric Press; 1968.
18. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. DSM-III. 3rd ed. Washington, DC: American Psychiatric Press; 1980.
19. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. DSM-III-R. 3rd revised ed. Washington, DC: American Psychiatric Press; 1987.
20. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. DSM-IV. 4th ed. Washington, DC: American Psychiatric Press; 1994.
21. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. DMS-IV-TR. 4th revised ed. Washington, DC: American Psychiatric Press; 2000.
22. Basson R, Berman J, Burnett A, et al. Report of the international consensus development conference on female sexual dysfunction: definitions and classifications. J Urol. 2000;163(3): 888–93.
23. Basson R. Human sex-response cycles. J Sex Marital Ther. 2001;27(1):33–43.
24. Basson R, Leiblum S, Brotto L, Derogatis L, Fourcroy J, Fugl-Meyer K, et al. Definitions of women's sexual dysfunction reconsidered: advocating expansion and revision. J Psychosom Obstet Gynecol. 2003;24(4):221–9.
25. The ICD-10 classification of mental and behavioural disorders. http://www.who.int/classifications/icd/en/bluebook.pdf.
26. Master WH, Johnson VE. Human sexual response. Boston: Little Brown; 1966. Masters WH, Johnson VE. Human sexual inadequacy. Boston: Little Brown; 1970. Kaplan H. The new sex therapy. New York: Brunner Mazel; 1974.
27. Mulhall JP, Incrocci L, Goldstein I, Rosen R. Cancer and sexual health. Springer; 2011. p. 93–102.
28. World Health Organization (SUI). Definition of sexuality and sexual health [Internet]. Switzerland: The World Health Organization; 2002.
29. World Health Organization (SUI). Definition of sexuality and sexual health: The World Health Organization; 2002. Sexual Health for the Millennium. A Declaration and Technical Document World Association for Sexual Health (WAS); 2008.
30. Harding ME. Woman's mysteries. A psychological interpretation of the feminine principle as portrayed in Myth, story and dreams. New York: G.P. Putnam's Sons; 1971, C.G. Jung Foundation for Analytical Psychology.

Anatomical and Physiological Description of Women's Sexuality

2

Serena Maruccia and Angela Maurizi

The female genitalia can be subdivided into the *internal genitalia* (vagina, cervix, uterus, fallopian tubes, and ovaries) and *external genitalia* (vulva), including the mons pubis, clitoris, labia majora and minora, which are the structures surrounding the urogenital cleft (Figs. 2.1 and 2.2). In anatomy textbooks there is a separation between the embryological development of the internal and external genital organs in males and females. It is important to know this because it is

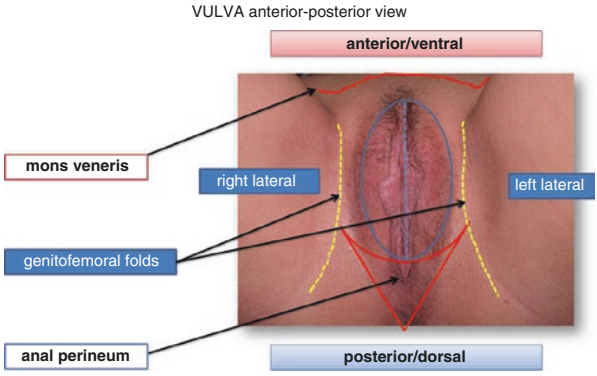

Fig. 2.1 Borders include posteriorly the *anal perineum*, laterally the medial side of the thighs, limited by *genitofemoral folds* (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_2

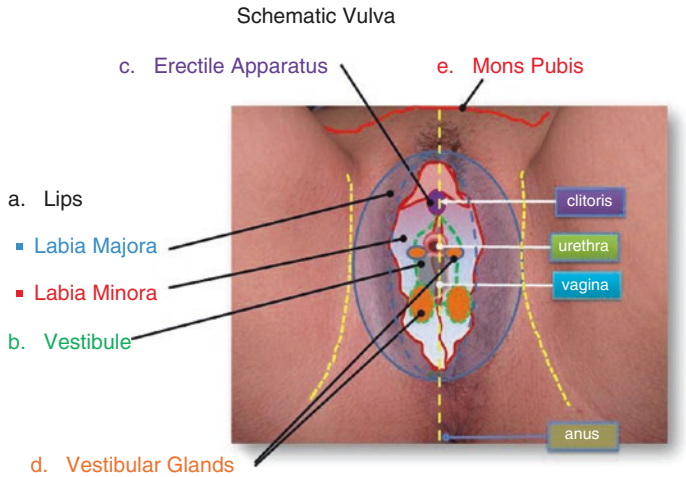

Fig. 2.2 Schematic representation of the Vulva (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

related to the functions of these organs, that are: the internal genitals have a reproductive function, while the external ones have the function of giving pleasure. This chapter describes the structures directly involved in physiological sexual response.

2.1 The Anatomy of the Organs

2.1.1 Clitoris

The clitoris is an external organ and has three erectile tissue parts, most of which lie beneath the skin: the glans, the body, and the crura; in the free part of the organ, it is composed of the body and the glans located inside of the prepuce, which is formed by the labia minora (Fig. 2.3). The size of the clitoris varies considerably. Internally, it is a triplanar complex of erectile tissue comprising a midline shaft some 20 cm long and 1–2 cm wide that divides internally into a pair of crura some 5–9 cm in length. The erectile tissue consists of trabecular smooth muscle and collagen connective tissue encircled by a thin fibrous capsule surrounded by large nerve trunks. Externally, the shaft is covered by the prepuce and is capped by a glans some 20 mm in length and 30 mm wide. Linked to the structure are two vestibular bulbs on either side of the vaginal introitus, closely applied to the urethra. The erectile compartments consist of the clitoris and the clitoral bulbs. The function of the bulbs is unclear; one speculation is that, when filled with blood, they support the vaginal wall during the thrusting of coition (Fig. 2.4).

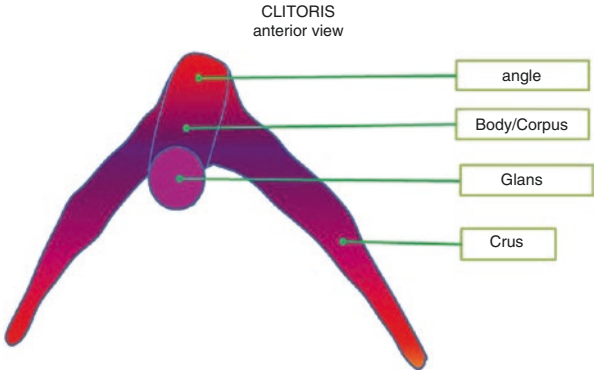

Fig. 2.3 Two portions can be distinguished: a *hidden portion*, which includes the crus and the posterior part of the body (BASE), and a *free portion*, formed by the elbow, the rod and the glans (APEX) (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

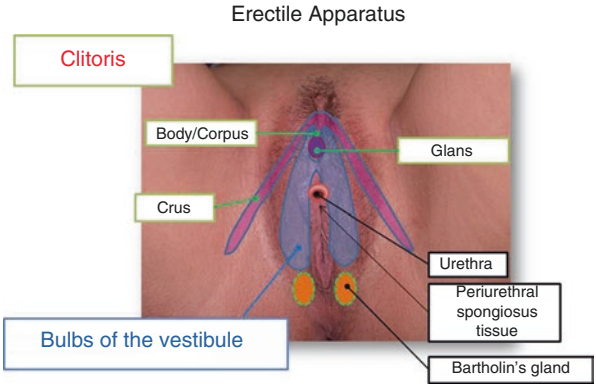

Fig. 2.4 *Erectile apparatus* comprises a median organ, the *clitoris*, and two lateral organs, the *bulbs of the vestibule* (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

2.1.2 Vagina

The vagina is a muscular tube leading from the external genitals to the cervix of the uterus.

In the unaroused state, the vagina is a potential space with its anterior and posterior walls collapsed and resting together, but they do not adhere because the walls are covered with a thin film of fluid. The average length of the vagina is 7–8 cm, and the width is 24–25 mm; this width is not uniform over the whole extension of the duct: it widens as it approaches the fornix. The vagina is located behind the bladder and the urethra, in front of the rectum, and above the vulva, into which it opens. The layered structure of the vagina consists of the luminal stratified squamous epithelium; a lamina propria layer containing connective tissue, blood vessels, nerves and receptors, collagen, and elastin fibers; and further inside a layer of smooth muscle. The vascolaritation of the vagina, stem mostly from the vaginal artery, branch of the internal iliac artery. The vaginal vessels are richly innervated by adrenergic, cholinergic, and vipergic nerves.

2.1.3 The Urogenital Triangle and Pelvic Floor Muscles

The pelvic floor muscles have the same composition in both men and women: the pubococcygeus and coccygeus muscles form the muscular diaphragm, which supports the pelvic viscera and opposes the downward thrust produced by an increase in intra-abdominal pressure.

2.1.4 Cervix

The cervix is shaped like a cylinder slightly swollen in its middle part and somewhat flattened in the direction from front to back. Its central canal is 3 mm in diameter and 2–3 cm in length and is lined with a greatly folded epithelium of columnar cells creating crypts that look like and are often mistaken for glands. The cells secrete mucus, which varies depending on the stages of the cycle, creating an optimal or hostile environment for sperm movement and survival.

2.1.5 Arterial Supply

Women's genitalia have a rich arterial blood supply. The labia are supplied from the inferior perineal and posterior labial branches of the internal pudendal artery as well as from superficial branches of the femoral artery. The clitoris is supplied by the iliohypogastric pudendal arterial bed. The middle part of the vagina is supplied by the vaginal branches of the uterine artery and the hypogastric artery; the distal part of the vagina is supplied by the middle hemorrhoidal and clitoral arteries.

2.2 Anatomy of the Distal Vagina

2.2.1 Vaginal Introitus

The distal vagina varies in appearance with age. The introitus is elastic in nature, as its mucosa is capable of permitting the release of a baby's head without tearing. The skin of the vulva is a hairless, moist and squamous epithelium (Fig. 2.5).

2.2.2 Hymen

The hymen is a thin, incomplete membrane of connective tissue, located at the edge of the vaginal canal and the vestibule. It is easily ruptured during the first

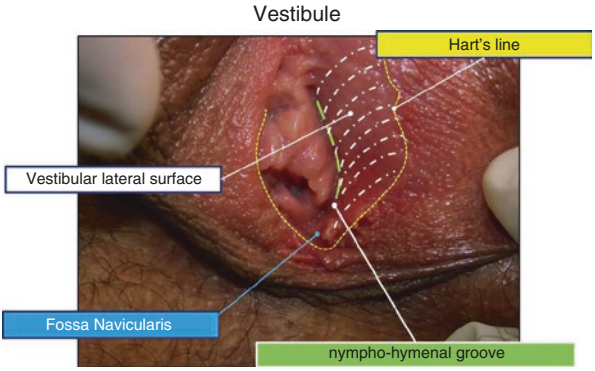

Fig. 2.5 The vulval *Vestibule* is the oval area, a cavity, between the labia minora. Labia minora form the lateral edges, extending from the upper hymenal ring and the lower Hart's line on each small lip. The vaginal opening is also more correctly called *introitus*, since the vagina is usually collapsed, with the opening closed. The introitus may be partly covered by a membranous septum, the hymen (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

sexual intercourse or regular coitus. Also routine use of tampons can cause it to rupture. So the hymen is reduced to a series of small irregular deviations around the vaginal opening termed hymenal lobules (flaps appearing after early intercourse) or carunculae myrtiformes (later less evident remains in parous introitus). Sometimes the rupture may be unnoticeable because it is minor. Occasionally the hymen may be so rigid as to cause sexual discomfort. In other rare cases, it may completely cover the vaginal opening and surgery is needed to allow menstruation. Morphological variations are very frequent (annular, semilunar, lipped, septate, cribriform, papillary, etc.)

The annular hymen has the shape of a diaphragm perforated by a central or eccentric hole. The semilunar hymen has a crescent shape which is concave anteriorly. It covers half, two-thirds, or three-quarters of the vaginal opening. The lipped or bi-lipped hymen has one or two side parts (lips), separated by a median longitudinal fissure (Fig. 2.6a-f).

2.2.3 Mons Pubis and Suspensory Ligaments

The mons pubis is a hair covered area composed of subcutaneous fat and fascial support to the clitoris and urethra. The variable shape of this area provides a sensitive anatomical marker of adult female androgenization.

Fig. 2.6 Morpological variations (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

2.2.4 Bulbs of the Vestibule

These are a double structure of erectile tissue that participate in the formation of the clitoris. The anterior edge of each bulb is combined with that of the other, under the knee of the clitoris. A venous network between bulbs and clitoris is present. The

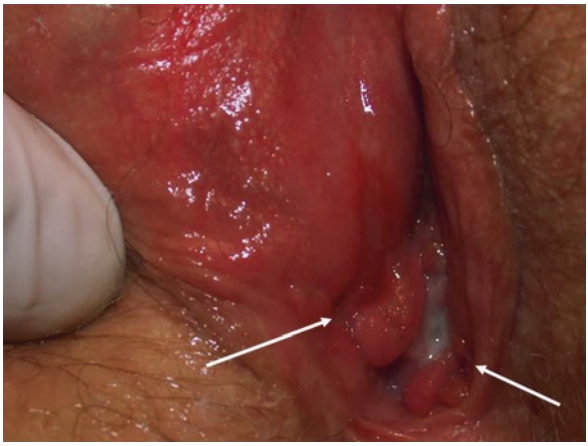

Fig. 2.7 Bartholin's glands (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

posterior edge is in contact with Bartholin's glands. Each bulb is ovoid and lies in the superficial perineal pouch, attached to the urogenital diaphragm and covered by the bulbocavernosus muscles, which are adjacent to the lateral wall of the vagina and embrace it.

2.2.5 Vestibule and the Root of the Clitoris

The vestibule comprises the area of hairless skin that extends between the medial aspect of the two labia minus, and from the frenulum of the glans anteriorly to the introitus posteriorly. Underlying the vestibule is the root of the clitoris. This zone is highly responsive to direct stimulation.

2.2.6 Vestibular Glands

They include the greater vestibular glands (Bartholin's glands), minor vestibular glands, and paraurethral Skene's glands.

Bartholin's glands are tubuloalveolar glands producing a stringy, viscous, colorless liquid. They are situated deeply in the posterior part of the big lips, just inferior to the bulbocavernosus muscles. The glands are included between the skin with the underlying Colles' fascia (fascia lata) and the inferior fascia of the urogenital diaphragm (Fig. 2.7). Excretory ducts, 1–2 cm long, open within the nympho-hymenal groove and are localized to the 5 and 7 o'clock (Fig. 2.8). Minor vestibular glands are present in more than half of the women and are located around the fourchette (it is the skin of the midline of the posterior margin of the vaginal introitus). Their number varies from 2 to 10 in each subject and the structure is similar to that of the greater glands.

Fig. 2.8 Excretory ducts (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

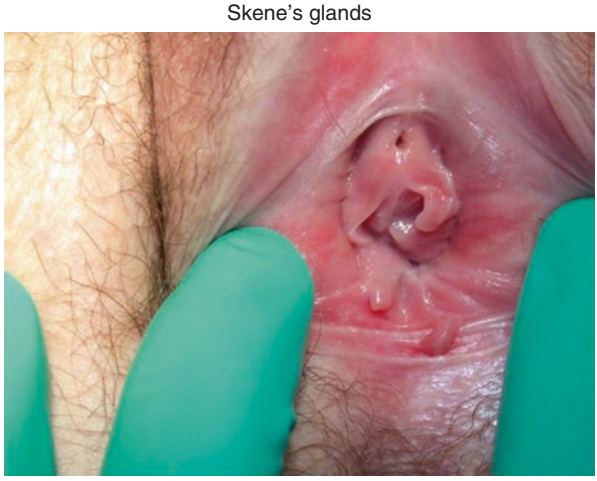

Fig. 2.9 *Paraurethral Skene's glands the so called "female prostate"*, derived from endodermic eversions of the urethra (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

Paraurethral Skene's glands open near lower half of the urethral external meatus (Fig. 2.9). They are greater than the periurethral glands.

2.2.7 Labia Minora

The labia minora, or nymphs are, at rest, approximated together; in males they correspond to the ventral wall of the cavernosa urethra and of the corpus spongiosum of the urethra. Anteriorly, the labia minora divide into lateral and medial parts. The lateral parts extend form the prepuce of the clitoris. The medial parts unite on the undersurface of the clitoris to form its frenulum. Posteriorly,

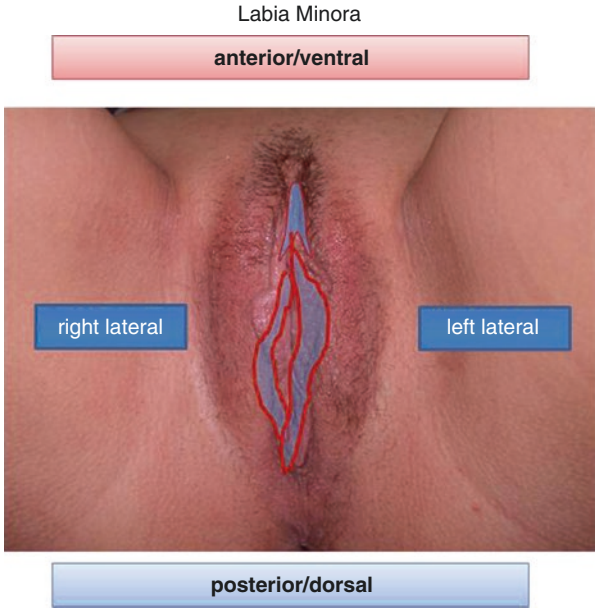

Fig. 2.10 The *labia minora* or *small lips* are two soft skin folds, flattened transversely, placed within the *labia majora* and surrounding the vestibule of the vagina (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

they form the frenulum of the labia minora but they can be also separated (Fig. 2.10). There is great variation in the size and morphology of the labia minora. They may be almost unrecognizable or may protrude from the labia majora ("hypertrophic" labia minora should not be considered a malformation). In addition, they can be asymmetrical or doubled on one or both sides. In some cultures, the labia minora can be very large because of the practice of stretching them. For example, in some African populations, they can be as large as 20 cm and are known as a "Hottentot apron." Today, elongation of the labia minora is classified in the type IV female genital mutilation. The labia minora have a notable sensibility; in fact, like the vaginal vestibule and the glans clitoris, they have a considerable number of free nervous endings and sensory receptors. The genital corpuscles are most important for the perception of erogenous sensibility, but Pacinian and Meissner's corpuscles are also present [27]. The exact functions of innervation are still under study, but the free nerve endings and Pacinian corpuscles detect pressure and vibration (Fig. 2.6).

2.2.8 Labia Majora

Covered to a variable extent by pubic hair, the labia majora are vascular fatty mounds that lie laterally to the labia minora (Fig. 2.11).

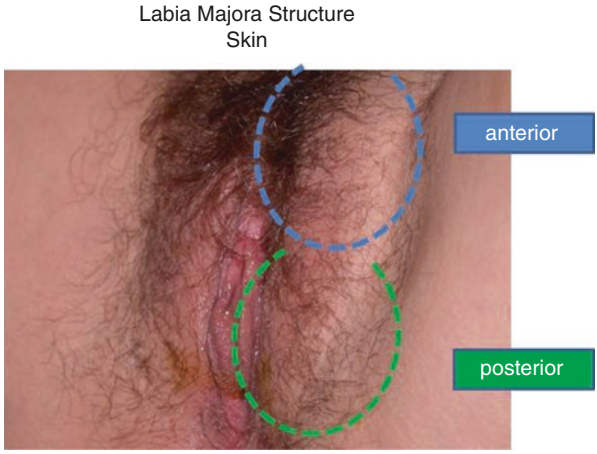

Fig. 2.11 Labia Majora Surface: Skin (Courtesy of Dr. Cosimo Oliva, Chapter 15, Atlas of Vulvar Dermatosis and Dermatitis)

2.2.9 Vascular Supply to Vulva

Arterial vascularization of the vulva mainly depends on the internal pudendal artery, a branch of the hypogastric artery.

It gives three branches:

- The inferior hemorrhoidal artery: it supplies the anus, the perianal region and the anal canal.
- The perineal artery: it supplies the perineal body, the ischiocavernosus muscles, the bulbocavernosus muscles, the superficial transverse muscle, and the vulva (posterior labial branches) with the erectile tissue of the vestibule.
- The artery of the clitoris: it is deeper and gives four further branches to supply erectile tissue in the superficial perineal pouch and the body of the clitoris.

2.3 Stimulation of the Female Sexual Act: Biological Principles of Sexual Behavior

There are two main sexual organs in the female: the clitoris and the brain. As in the male sexual act, successful performance of the female sexual act depends on both psychic and local stimulation. Thinking sexual thoughts can trigger sexual desire in the female, and this aids greatly in the performance of the female sexual act.

Such desire is based largely on a woman's background training as well as on her physiological drive, although sexual desire does increase in proportion to the level of sex hormones secreted.

A normal sexual response requires the anatomic and functional integrity of the brain's entire limbic system which is essential in both sexes for the initiation of sexual desire and related sexual phenomena.

Its function activates sexual fantasies, erotic dreams, mental sexual arousal, and the initiation of the cascade of neurovascular events triggering all of the somatic and genital responses of sexual function.

The most important areas shown to be activated during arousal are the hypothalamus, the extrastriate visual cortex, the orbitofrontal cortex, the anterior cingulate cortex, the ventral striatum, and the amygdala.

However, only the ventral striatum can be convincingly linked to female sexual arousal: overall visual sexual stimulation (VSS) produces greater neural activations in men than in women in both the hypothalamus and the amygdala. One explanation is that in men, visual stimuli have fast access to primordial systems underlying sexual response, and this reflects men's higher propensity to identify cues for sexual opportunity, especially in the visual domain. In both the amygdala and hypothalamus, women showed significant activation only with longer VSS samples, providing further support for the idea that men have a more instinctive sexual response, while women take a more thoughtful approach with regard to sexual encounters [13].

For example, the most important sexual cues in women for increasing mental arousal typically involve verbal intimacy, such as having her partner's receptive and attentive ear or having affectionate or erotic words spoken to her [14].

The somatotopical representation of the clitoris is on the dorsal convexity of the postcentral gyrus as it was in men. The secondary somatosensory cortex is readily activated by sensory stimulation in the pelvic region. In addition, VSS activated the inferior parietal lobule, which contains secondary somatosensory cortex, in both men and women, possibly as a result of the sensory input caused by genital arousal [13].

Local sexual stimulation in women occurs in more or less the same manner as in men because massage and other types of stimulation of the vulva, vagina, and other perineal regions can create sexual sensations.

The glans of the clitoris is especially sensitive for initiating sexual sensations.

As in the male, the sexual sensory signals are transmitted to the sacral segment of the spinal cord through the pudendal nerve and sacral plexus. Once these signals have entered the spinal cord, they are transmitted to the cerebrum. Also, local reflexes integrated in the sacral and lumbar spinal cord are at least partly responsible for some of the reactions in the female sexual organs.

In men and women, the sexual response is coordinated by the same neurotransmitters such as monoamines (dopamine, serotonin, and norepinephrine) and neurotrophins (including the nerve growth factor, NGF, which increases in the brain and peripheral blood when people fall in love) [14]. Male sexual behavior is typically stable over the entire adult male lifespan; this may be potentially explained by a typical male's lifelong production of testosterone at a relatively tonic, constant rate.

In contrast the physiology of the female sexuality is highly discontinuous, both during the regular menstrual cycle as well as during major reproductive life events such as pregnancy, puerperium, abortion, and menopause [17].

Hormonal fluctuations during the menstrual cycle influence mood, cognition, memory, and arousal along with sexual interest. VSS induced more brain responses in women during their midluteal phase than during their menses [13].

Desire also changes during the monthly sexual cycle, reaching a peak near the time of ovulation, probably because of the high levels of estrogen secretion during the preovulatory period.

It has also been shown that while receptivity to pheromones remains stable over life in men, there is a peak in pheromone receptivity during ovulation in women. Pheromones may be responsible for mediating interactions in the mid-cycle variations which may turn to be triggered by the ovulatory androgen peak in sexual desire, arousability, and receptivity.

It is known that male pheromones (androstenol and androstenone from male sweat) have a direct impact on female sexual desire. The smell of androstadienone also leads to a constant higher level of the cortisol hormone in women. Likewise, female vaginal pheromone (copulins) influences the male perception of the female and might induce hormonal changes in males [18].

It is clear now that sexual attraction between the human genders is modulated by sexual pheromones. However these chemical signals can modulate sexual desire between men and women. Thus, homosexual men prefer seats sprayed with androstenone and so do heterosexual women.

Moreover, the natural compounds collected from lactating women and their breastfeeding infants can increase sexual motivation of other women [15].

Sexual pheromones not only modulated sexual desire but also interfere with sexual hormones and increase sexual arousal. Sexual hormones maintain the physical and functional sexual tonus and, in turn, contribute to increasing the level of sexual desire.

Human sexual pheromones are actually derivative compounds that form sexual hormones. This interaction/synergy between sexual desire and sexual arousal occurs frequently during a normal sexual response, generating difficulty on discriminating these two phenomena [19].

The cerebral dual representation of sexual function would be possible through a dual hormonal (arousal) and pheromonal (libido) neuromodulation. Sexual arousal increases the body's need for sex, while libido augments the desire for a partner [15].

Sex steroids play a crucial role in maintaining the anatomical and functional integrity of all the structures involved in women's sexual function. Despite the growing interest in treatment of sexual dysfunction with androgens in clinical practice, no normal range of testosterone has been agreed upon, due in part to the difficulties with the sensitivity of assays for total and free testosterone in women and the fluctuations during the menstrual cycle and menopausal status [25]. The biologically active androgen is testosterone, which circulates bound tightly to sex-hormone-binding globulin (SHBG) and loosely to albumin and transcortin. The fraction of testosterone that remains unbound to SHBG is deemed bioavailable. Thus, plasma levels of total

testosterone and free testosterone as well as SHBG need to be determined clinically. Despite the lack of sensitivity of the assays and limited controlled clinical studies, an increasing body of evidence is emerging suggesting that women with signs and symptoms of androgen insufficiency respond well to androgen therapy without significant side effects. A recent systematic review including all randomized and placebo-controlled trials of treatment for FSD in postmenopausal women concluded that many treatments that are used in practice are not supported by adequate evidence [26].

2.4 Female Erection and Lubrification

During sexual stimulation, the female sexual arousal response is elicited by sensory stimulation as well as central nervous activation resulting in increased blood flow to the genitals. This culminates in a series of vasocongestive as well as neuromuscular events leading to physiological changes.

There are six vascular compartments, comprising the external female genitalia: the clitoris, clitoral bulbs, labia minora, urethra, and vestibule/vagina. These compartments are composed of erectile as well as nonerectile tissues with erectile tissue demonstrating the greatest volume change with engorgement during sexual arousal.

The erectile tissue is controlled by the parasympathetic system which induces dilation of the affluent arteries, thus probably secondary to release of acetylcholine, nitric oxide (NO), and vasoactive intestinal polypeptide (VIP).

The female urethra has venous sinuses that are filled with blood; in the lining of the urethra are triangular-shaped paracrine cells that are thought to have mechanoreceptor properties and contain serotonin [13].

Stretching of the urethra usually occurs during coitus or during digital stimulation of the anterior wall, and this may activate these mechanoreceptor cells to release serotonin, thus sensitizing the nerve endings in the urethra, creating pleasurable sensations, and converting a urinary structure into a sexual one [20].

At the beginning of sexual arousal, the blood supply to the vagina is minimal due to the high sympathetic vasomotor tone. Following the beginning of the sexual stimulus (time lapse of a few seconds), the central sympathetic tone is reduced, and the arterial supply is enhanced through VIP and/or NO release; vasomotion thus decreases as more capillaries are recruited.

Rapidly the recruitment of the capillaries becomes maximal, and the vagina, clitoris, and labia become fully congested. These events create a reactive desire to dissipate the congestion by the induction of the orgasm [13].

Parasympathetic signals pass to the bilateral Bartholin's glands located beneath the labia minora and cause them to secrete mucus immediately inside the introitus.

This mucus is responsible for much of the lubrication during sexual intercourse, although much is also provided by mucus secreted by the vaginal epithelium and a small amount from the male urethral glands.

The increased hydrostatic pressure inside the capillaries of microcirculation forces out a plasma transudate into the interstitial space around the blood vessels. Continued formation of this transudate fills up the interstitial space and then passes

through and between the cells of vaginal epithelium to leak onto the surface wall of the vagina.

In the sexually unstimulated state, the vaginal fluid has a higher K^+ and lower Na^+ concentration throughout the phases of the menstrual cycle. In contrast, the arousal lubrication fluid has a much higher Na^+ concentration than the basal fluid approaching that of plasma. On cessation of the sexual arousal, the vaginal Na^+ is transferred back to the blood, thus resetting the vagina to the basal just moist condition [21, 22].

This lubrication is necessary during intercourse to establish a satisfactory massaging sensation rather than an irritating sensation, which may be provoked by a dry vagina. A massaging sensation constitutes the optimal stimulus for evoking the appropriate reflexes that culminate in both the male and female climaxes.

The clitoris is the homologue of the male's glans and corpora cavernosa. It is an external organ and has three erectile tissue parts: the glans, the body, and the crura. In the free part of the organ, it is composed of the body and the glans, located inside the prepuce, which is formed by the labia minora. A longer distance between the clitoral complex and the vaginal lumen correlates with poorer sexual function [16].

The clitoris has a unique function: sexual pleasure. During sexual arousal it is shown in its erection and an increase in size (especially the diameter). During the plateau phase, it is observed that at the height of the arousal and orgasm, there is a retraction of the glans into the prepuce [6].

This event happens in every woman, regardless of the type of stimulation, coital position, degree of clitoral tumescence, or the initial clitoral size.

This occurs because in adult women, the size of the glans is much smaller than in males and does not grow during puberty. In fact, it does not enlarge during sexual arousal. In addition, during sexual arousal, the female prepuce does not retract as it does in males, because it is continuous with the labia minora. So with erection of the body of the clitoris, there is the apparent disappearance of the glans within the prepuce [6].

The erectile cycle has been extensively studied in the penis and to a lesser extent in the clitoris and other female erectile organs. Erection from flaccid state is reached in three phases: latent, tumescence, and rigid or muscular [6].

Erection is due to a neurovascular mechanism: an inflow of arterial blood and an obstruction of venous return. Tumescence is due to a reduction in the alpha-sympathetic tonus of the cavernous tissue permitting influx of the arterial blood and a decreased venous flow from compression of the subalbugineal venous network against the tunica albuginea of the corpus cavernosum. Rigidity is due to an increase in intracavernous arterial pressure together with contraction of the perineal muscles under the somatic control of the pudendal nerve [6].

Contraction of the ischiocavernosus muscle produces the male rigid-erection phase.

In sexual arousal the role of both the cervix and the uterus has not been definitely settled. It has been reported that the uterus contracts during high levels of sexual excitement and at orgasm [13].

While genital stimulation is very effective in creating female sexual arousal, the stimulation of the female nipple or breast can significantly enhance sexual arousal, but very few studies are available [13].

2.5 Female Orgasm

When local sexual stimulation reaches maximum intensity, and especially when the local sensations are supported by appropriate psychic conditioning signals from the cerebrum, reflexes are initiated that cause the female orgasm or climax. Orgasm may be defined as "a variable, transient peak sensation of intense pleasure, creating an altered state of consciousness, usually accompanied by involuntary, rhythmic contractions of the pelvic striated circumvaginal musculature, with concomitant uterine and anal contractions and myotonia that resolves the sexually induced vasocongestion (sometimes only partially), usually with an induction of well-being and contentment" [24]. The female orgasm is analogous to emission and ejaculation in the male, and it may help promote fertilization of the ovum.

Indeed, the human female is known to be somewhat more fertile when inseminated by normal sexual intercourse rather than by artificial methods, thus indicating an important function of the female orgasm.

Possible reasons for this are as follows. First, during the orgasm, the perineal muscles of the female contract rhythmically, which results from spinal cord reflexes similar to those that cause ejaculation in the male. It is possible that these reflexes increase uterine and fallopian tube motility during the orgasm, thus helping to transport the sperm upward through the uterus toward the ovum; also, the orgasm seems to cause dilatation of the cervical canal for up to 30 min, thus allowing easy transport of the sperm.

Second, in many lower animals, copulation causes the posterior pituitary gland to secrete oxytocin; this effect is probably mediated through the brain amygdaloid nuclei and then through the hypothalamus to the pituitary.

The oxytocin causes increased rhythmical contractions of the uterus, which have been postulated to cause increased transport of the sperm.

In addition to the possible effects of the orgasm in fertilization, the intense sexual sensations that develop during the orgasm also pass to the cerebrum and cause intense muscle tension throughout the body.

But after culmination of the sexual act, this gives way to a sense of satisfaction characterized by relaxed peacefulness, an effect called resolution.

Even today, female sexuality is considered in terms of reproduction, instead of pleasure. Also women have to reach orgasm during vaginal intercourse to be considered true women, even if the female orgasm is caused by female erectile organs and not by the vagina.

Vaginal orgasm doesn't have any scientific basis, but it was invented by Freud in 1905 [23].

The vagina has no anatomical structure that can cause orgasm, and up by now, the presence of vagus nerve terminations in the vagina has not been demonstrated.

In fact, in women with vaginal agenesis, the sexual responses of the artificial vagina are identical to those of the normal vagina.

When sign of impending orgasm occurs, presuming that effective sexual stimulation is continued, orgasm is sure to follow in women with an artificial vagina, just as it does in women with the normally constituted vagina. The characteristic physiological expression of orgasm in both artificial and normal vaginas is the onset of regularly recurring contractions of the bulbocavernosus muscle and of the perineal muscle [23].

The variation in the distance between a woman's glans clitoris and her urethra predicts the likelihood that she will experience orgasm during intercourse, and it was proposed that if the distance was less than 2.5 cm, a woman was very likely to have orgasm only from sexual intercourse.

However vaginal orgasm does not exist: the "vaginal" orgasm that some women report is always caused by the surrounding erectile organs. It is confirmed by the fact that the duration of the penile-vaginal intercourse is not important for the woman's orgasm [23].

In fact it is known that penile-vaginal intercourse actually stimulates the clitoris through thrusting traction on its attached ligaments via the anterior vaginal wall, but orgasm with a finger in the vagina is possible by moving the hand in circle to stimulate all the female erectile organs.

The anterior vaginal wall is an active organ, transmitting, during intercourse, the effect of penile thrusting in the vagina to the clitoris, by stretching the two ligaments that insert around its base.

In 1950, Grafenberg described a distinct erotogenic zone on the anterior wall of the vagina, which was referred to as Grafenberg spot (G-spot). As the result, the G-spot has become a central topic of popular speculation and multimillion dollar business and refers to an erotically sensitive spot.

The G-spot represents the part of the urethra that contains the periglandular of paraurethral tissue, but studies by Puppo and Gruenwald stated that the G-spot of the anterior vaginal wall is located in Pawlik's triangle, a region that corresponds to Lieutaud's triangle in the bladder.

The existence of a specific anatomical structure known as the G-spot has not been proven by any relevant scientific studies.

Many men think that long intercourse is the key to having orgasms during intercourse, but longer intercourse is not helpful to women, and some females may be grateful to get it over quickly [23].

For example, premature ejaculation does not exist if both partners agree that the quality of their sexual encounters is not influenced by the time of ejaculation. Noncoital sexual acts after male ejaculation can be used to produce orgasm in women.

In the vaginal vestibule, the external orifice of the urethra is seen with the paraurethral ducts opening on both sides. They are found in women, with the intraurethral glans considered as the female prostate. The secretion of these glands is expelled through the urethral meatus or through the orifices of the paraurethral ducts into the vaginal vestibule, which corresponds to the dorsal wall of the male

cavernosa urethra. Female prostate secretion during orgasm corresponds to the emission phase female ejaculation [6].

From a physiological point of view, the term "female emission" is more accurate than female ejaculation. In a low percentage of women, powerful emission is reported. In the male this corresponds to the emission of seminal fluid into the prostatic urethra. At times this emission may be mistaken for coital incontinence, but the liquid composition is obviously not urine.

The lack of the ejaculation phase in the female could explain why women do not have refractory period and are able to have multiple orgasms [23].

Orgasm is an intense sensation of pleasure achieved by stimulation of erogenous zones (clitoris first) that have a heightened sensitivity.

Physiologically, it is a brief episode of physical release from the vasocongestive and myotonic increment developed in response to sexual stimuli. All female orgasms follow the same reflex response pattern, no matter what the source of sexual stimulation.

An orgasm that comes from rubbing the clitoris cannot be distinguished physiologically from intercourse or breast stimulation alone.

Distinguishing between clitoral and vaginal orgasm is not correct from a physiological point of view. The high sensitivity of the female external genitals is not only due to the clitoris but also to the thin epithelial lining covering the labia minora and the vestibule.

The stimulation of the labia minora and the vestibule of the vagina would facilitate the achievement of orgasm in women, with feelings even higher than clitoral stimulation alone, with a more global stimulation of the external genitalia, which include the whole female erectile apparatus.

Clitoris responds to equal facility to both somatogenic and psychogenic forms of stimulation.

Women have the physical capability of being multiorgasmic, that is, they can have one or more additional orgasms within a short time without dropping below the plateau level of sexual arousal.

Being multiorgasmic depends on both continued effective sexual stimulation and sexual interest. Multiple orgasms in females seem to occur more frequently during masturbation [23].

In the matter of physical capability, females have an almost unlimited orgasmic potential, while men, because of the refractory period, are unable to have a rapid series of ejaculations.

Women don't have a true refractory period, and orgasmic potential is undoubtedly restricted by fatigue.

A woman's capability to have multiple orgasms is dependent on a combination of developmental, psychological conditions.

Some women may experience a status orgasmus, which is a single, long-continued orgasmic episode or a series of rapidly recurrent orgasmic experiences between which no recordable plateau-phase intervals can be demonstrated, which can last up to 1 min [6].

Menopause results in a decline in circulating estrogen levels, which results in atrophy of urogenital tissues, vaginal shortening and thinning, decrease in vaginal elasticity, and lubrication.

For all these reasons, sexual intercourse may become uncomfortable [6].

These alterations are related to the reproductive organs and do not affect the clitoris.

Female orgasm and clitoral sexual response in fact are not affected by aging, and for this reason, women have the physical capability of being orgasmic in all ages.

Female orgasm is possible in all women, always with effective stimulation of the female erective organs, for example, the female penis, during masturbation, cunnilingus, partner masturbation, and also during vaginal/anal intercourse simply by stimulating the clitoris with a finger.

It is important for partners the development of specific coital techniques to facilitate clitoral stimulation and that the female superior coital position allows direct stimulation of the clitoris to be achieved easily with the fingers [6].

Sexuality is an important part of health and quality of life.

Women's sexual rights are fundamental, and sexual pleasure, including autoerotism, is a source of physical and psychological well-being that contributes to human happiness (World Association for Sexual Health 2008).

The knowledge of anatomy and physiology of female sexual organs is essential to women's health.

References

1. O'Connell HE, DeLancey JO. Clitoral anatomy in nulliparous, healthy, premenopausal volunteers using unenhanced magnetic resonance imaging. J Urol. 2005;173:2060–3.
2. O'Connell HE, Hutson JM, Anderson CR, Plenter RJ. Anatomical relationship between urethra and clitoris. J Urol. 1998;159:1892–7.
3. O'Connell HE, Anderson CR, Plenter RJ, Hutson JM. The clitoris: a unified structure. Histology of the clitoral glans, body, crura and bulbs. Urodinamica. 2004;14:127–32.
4. O'Connell HE, Sanjeevan KV, Hutson JM. Anatomy of the clitoris. J Urol. 2005;174:1189–95.
5. O'Connell HE, Eizenberg N, Rahman M, Cleeve J. The anatomy of the distal vagina: towards unity. J Sex Med. 2008;5:1883.
6. Puppo V. Anatomy and physiology of the clitoris vestibular bulbs, and labia minora with a review of the female orgasm and the prevention of female sexual dysfunction. Clin Anat. 2013;26:134–52.
7. The ESSM Syllabus of Sexual Medicine 2013.
8. Anglana F et al. editors. Trattato di Patologia Vulvare. Vol. I. SEE-Firenze; 2003.
9. Neill SM, Lewis F. Basics of vulvar embriology, anatomy and physiology. In: Neill SM, Lewin F, editors Ridley's the vulva. 2nd ed. Singapore: Wiley-Blackwell; 2009.
10. Williams PL, Bannister LH, Berry MM, et al. In: Gray's anatomy. 38th ed. New York: Churchill Livingstone; 1995.
11. Testut L, Latarjet A. Traite´ d'Anatomie Humaine. neuvie`me e´ dition. Paris: G. Doin & Cie; 1972.
12. Verkauf BS, Von Thron J, O'Brian WF. Clitoral size in normal women. Obstet Gynecol. 1992;80:41–4.
13. Salonia A, Giraldi A. Physiology of women's sexual function: basic knowledge and new findings. J Sex Med. 2010;7:2637–60.

14. Graziottin A. Giraldi A Anatomy and physiology of Women's Sexual Function in: Porst H. Buvat J. (Eds), ISSM (International Society of Sexual Medicine) Standard Committee Book, Standard practice in SexualMedicine. Blackwell, Oxford: UK; 2006. p. 289–304.
15. Motofei I. A dual physiological character for sexual function: libido and sexual pheromones. BJUI. 2009;104:1702–8.
16. Vaccaro C. The use of magnetic resonance imaging for studying female sexual function. Clin Anat. 2015;28:324–30.
17. Graziottin A. Similarities and differences between male and female sexual dysfunctions. In: Kandeel F, Lue T, Pryor J, Swerdloff R, editors. Male sexual dysfunction: pathophysiology and treatment. New York: Marcel Dekker; 2006.
18. Gammer K, Jutte A. Battle of odors: significance of pheromones for human reproduction. Gynakol Gerburtshilfliche Rundsch. 1997;37:150–3.
19. Ortigue S, Bianchi-Demicheli F. Interaction between human sexual arousal and sexual desire. a challenge for social neuroscience. Rev Med Suisse. 2007;3:809–13.
20. Riley A, Lees W, et al. An ultrasound study of human coitus. In: Bezemer W et al., editors. Sex masters. Amsterdam: Excerpta Medica; 1992. p. 29–32.
21. Wagner G. Vaginal transudation. In: Beller F, Shumacher G, editors. The biology of the fluids in the female genital tract. Amsterdam: Elsevier North Holland Inc; 1979. p. 25–34.
22. Wagner G. Electrolytes in vaginal fluid during the menstrual cycle of coitally active and inactive women. J Reprod Fertil. 1980;60:17–27.
23. Puppo V, Puppo G. Anatomy of sex: revision of the new anatomical terms used for the clitoris and the female orgasm by sexologists. Clin Anat. 2015;28:293–304.
24. Meston CM, Hull E, Levin RJ, Sipski M. Disorders of orgasm in women. J Sex Med. 2004;1:66–8.
25. Nappi RE, Domoney C. Pharmacogenomics and sexuality: a vision. Climacteric. 2013;16 Suppl 1:25–30. doi:10.3109/13697137.2013.806402. Epub 2013 Jun 12.
26. Davis SR. When to suspect androgen deficiency other than at menopause. Fertil Steril. 2002;77:S68–71.
27. Schober J, Aardsma N, Martin Alguacil N. Terminal innervation of female genitalia, cutaneous sensory receptors of the epithelium of the labia minor. Clin Anat. 2015.

Female Sexuality: A State of Mind

3

Linda Vignozzi

Female sexuality encompasses a broad range of behaviors and processes, including female sexual identity and sexual behavior and the physiological, psychological, social, cultural, political, and spiritual or religious aspects of sexual activity.

Sexuality varies across the cultures and regions of the world, and has continually changed throughout history, and this also applies to female sexuality. Aspects of female sexuality include issues pertaining to biological sex, body image, self-esteem, personality, sexual orientation, values and attitudes, gender roles, relationships, activity options, and communication.

In particular, three major dimensions seem to interact on women's sexual health: female sexual identity, sexual function, and sexual relationship [23, 24, 26].

Healthy sexual function and sexual dysfunction were studied for the first time by Masters and Johnson [39] and later elaborated on by Kaplan [32]. In the 1960s, they described four phases of the human sexual response cycle: excitement, plateau, orgasm, and resolution.

Sexual excitement refers to a subjective feeling of sexual pleasure and accompanying physiological changes. This phase includes vaginal lubrication in females. Plateau phase is a heightened state of excitement attained with continued stimulation. There is marked sexual tension in this phase, which sets the stage for the orgasm. The third stage, orgasm or climax, is defined as the peak of sexual pleasure. The final phase is resolution, during which a general sense of relaxation and well-being is experienced [39].

Today it is known that this linear model more accurately depicts the male than the female sexual cycle [31].

Recently, a more female-specific intimacy-based model of sexual response has been proposed. [4, 6]. This model describes a circular relationship between

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_3

sexuality and satisfaction. In 2002, Basson described a "sexual response cycle" that incorporates psychological and social aspects into female sexual function, such as emotional intimacy and emotional satisfaction as well as sexual desire and physical satisfaction [4, 6].

This model definitively recognizes that sexual function and response are different in men and women. For example, in contrast to men, women's desire does not always precede sexual arousal, with many women participating in sexual activity out of love and affection for their partners. Once engaged in sexual activity, women may then become aroused and then experience desire. For women, the sexual response cycle is intimately intertwined with the overall relationship that they are in and incorporates the societal and psychological milieu.

For men, sexual function and response centers on the ability to achieve and maintain an erection. For women, however, sexual response is much more complex, involving social, psychological, neurologic, vascular, and hormonal processes and includes complex interaction of sexual stimulation, the central nervous system, the peripheral neurovascular system, and hormonal influences [7, 15, 48].

Therefore, it seems clear that women's sexuality is multifactorial, rooted in biologic, psychosexual, and context-related factors [2, 3, 8, 16–19, 23, 24, 35, 36, 45, 49]. The latter include couple dynamics, family and sociocultural issues, and developmental factors, including sexual abuse [5, 13, 34].

Several recent studies analyzed the role of interpersonal and affective factors in the development and maintenance of sexual problems [9, 10, 21, 25, 46], because this is especially true for female sexuality [12]. In her revised model of female sexual response, Rosemary Basson, for example, highlights the importance of factors such as self-image, emotional intimacy, and relationship satisfaction [4]. The development of this model has led to a shift in research emphasis with a stronger focus on psycho-affective and interpersonal factors [9, 10, 27, 43].

One such psycho-affective aspect is emotional intelligence (EI). Mayer and colleagues describe EI as some sort of intelligence in the sense of a cognitive ability, representing individual differences in processing affective information [40].

In spite of the awareness of the importance of psycho-affective factors in the development of sexual problems and the remarkable number of studies investigating the role of EI in a vast number of human behaviors and disorders [28], so far, only one study has investigated the possible relationship between EI and sexual functioning [9]. In the study conducted by Burri and colleagues, the authors reported a link between EI and female orgasmic frequency, with women showing higher EI levels also reporting more frequent orgasms [9].

Several constructs closely related to EI have also been associated with sexual functioning, including personality, alexithymia, and differentiation of self [10, 27, 33, 41]. In a recent study, EI was significantly negatively correlated with female sexual desire [51].

A further important aspect to consider is the role of interpersonal factors in determining of sexual problems, even in the context of organic diseases [1, 42, 47]. The relational component is not easy to evaluate and quantify, since its relative weight may vary according to age, partnership dynamics such as length and stage of

partnership, and reproductive status, and it can be significantly influenced by cultural gender schemes. It has been reported that, with aging, women emphasize the importance of sexual intercourse less and place a greater value on intimacy, companionship, and affection [29]. The quality of a relationship has been highlighted as a main determinant of sexual interest and behavior in older married Greek women [44]. In a sample of Portuguese women from general population (mean age 35 years), relationship length predicted lower sexual desire [11]. A survey in middle-aged women in Hong Kong found that divorce history is a risk factor for lack of sexual interest and inability to achieve orgasm [52]. A survey of more than 900 female hospital employees in Taiwan (mean age 36 years) found that a poor relationship with the partner and perception of partner's sexual dysfunction were major risk factors for low desire, low arousal, low orgasmic function, and low satisfaction [30].

The recognition of the negative impact of male sexual dysfunctions on partner's sexual life is well established. In particular, the adverse effects of erectile dysfunction (ED) on the partner's sexual experience have been widely investigated [20]. It has been suggested that, in women, a satisfying penile-vaginal intercourse, for which an adequate erection is a prerequisite, can provide higher levels of sexual satisfaction, resulting in lower rates of depression as compared with other types of sexual activity [14]. Other authors suggested that orgasm during penile-vaginal intercourse and simultaneous orgasms in the couple are specifically associated with women's satisfaction, whereas other elicitors of orgasm are not [50].

The impact of another common male sexual dysfunction, premature ejaculation (PE), on female partners has also been studied in detail [37]. It has been reported that PE can lead the partner to inadequate central and genital arousal, vaginal dryness, and inability to climax [22].

Finally, Maseroli and colleagues recently provided evidence that women's sexuality seems to be mostly impaired by the perceived reduction of their partner's sexual interest [38]. In a sample of 156 heterosexual women consulting for sexual problems, results showed that sexual functioning (as derived by Female Sexual Function Index, FSFI) decreased as a function of partner's age, conflicts within the couple, a relationship without cohabitation, and the habit of engaging in intercourse to please the partner; FSFI total score increased as a function of frequency of intercourse, attempts to conceive, and fertility-focused intercourse. FSFI total score showed a negative, stepwise correlation with partner's perceived hypoactive sexual desire (HSD), whereas no significant correlation was found between FSFI and ED, PE, or DE. In an age-adjusted model, partner's HSD was negatively related to FSFI total score and arousal, lubrication, orgasm, satisfaction, and pain domains. Partner's HSD was also significantly associated with somatized anxiety, low frequency of intercourse, low partner's care for the patient's sexual pleasure, and with a higher frequency of masturbation, even after adjusting for age. In patients not reporting any HSD, FSFI total score was significantly lower when their partner's libido was low; the correlation disappeared if the patient also experienced HSD.

In conclusion, these findings highlight the relevance of psycho-affective and relational factors in the development of female sexual problems.

References

1. Aerts L, Enzlin P, Verhaeghe J, Poppe W, Vergote I, Amant F. Long-term sexual functioning in women after surgical treatment of cervical cancer stages IA to IB: a prospective controlled study. Int J Gynecol Cancer. 2014;24:1527–34.
2. Basson R, Berman J, Burnett A, Derogatis L, Ferguson D, Fourcroy J, Goldstein I, Graziottin A, Heiman J, Laan E, Leiblum S, Padma-Nathan H, Rosen R, Segraves K, Segraves RT, Shabsigh R, Sipski M, Wagner G, Whipple B. Report of the International Consensus Development Conference on female sexual dysfunction: definitions and classification. J Urol. 2000;163:888–93.
3. Basson R, Leiblum S, Brotto L. Revised definitions of women's sexual dysfunction. J Sex Med. 2004;1:40–8.
4. Basson R. Are the complexities of women's sexual function reflected in the new consensus definitions of dysfunction? J Sex Marital Ther. 2001;27:105–12.
5. Basson R. Introduction to special issue on women's sexuality and outline of assessment of sexual problems. Menopause. 2004;11:709–13.
6. Basson R. Using a different model for female sexual response to address women's. Problematic low sexual desire. J Sex Marital Ther. 2001;27:395–403.
7. Berman JR, Bassuk J. Physiology and pathophysiology of female sexual function and dysfunction. World J Urol. 2002;20:111–8.
8. Binik YM, Reissing E, Pukall C, Flory N, Payne KA, Khalife S. The female sexual pain disorders: genital pain or sexual dysfunction? Arch Sex Behav. 2002;31:425–9.
9. Burri AV, Cherkas LM, Spector TD. Emotional intelligence and its association with orgasmic frequency in women. J Sex Med. 2009;6:1930–7.
10. Burri AV, Schweitzer R, O'Brien J. Correlates of female sexual functioning: adult attachment and differentiation of self. J Sex Med. 2014;11:2188–95.
11. Carvalho J, Nobre P. Sexual desire in women: an integrative approach regarding psychological, medical, and relationship dimensions. J Sex Med. 2010;7:1807–15.
12. Clayton AH. Epidemiology and neurobiology of female sexual dysfunction. J Sex Med. 2007;4:260–8.
13. Clulow C. Adult attachment and couple psychotherapy. Hove: Brunner-Routledge; 2001.
14. Costa R, Brody S. Sexual satisfaction, relationship satisfaction, and health are associated with greater frequency of penile–vaginal intercourse. Arch Sex Behav. 2012;41:9–10.
15. Davis SR, Guay AT, Shifren JL, et al. Endocrine aspects of female sexual dysfunction. J Sex Med. 2004;1:82–6.
16. Dennerstein G. Dyspareunia and DSM: a gynecologist's opinion. Arch Sex Behav. 2005;34(28):57–61; author reply 63–67.
17. Dennerstein G. Vaginal yeast colonization in nonpregnant women: a longitudinal study. Obstet Gynecol. 2005;105:1493; author reply 1494.
18. Dennerstein L, Lehert P, Burger H. The relative effects of hormones and relationship factors on sexual function of women through the natural menopausal transition. Fertil Steril. 2005;84:174–80.
19. Dennerstein L, Lehert P. Modeling mid-aged women's sexual functioning: a prospective, population-based study. J Sex Marital Ther. 2004;30:173–83.
20. Fisher WA, Eardley I, McCabe M, Sand M. Erectile dysfunction (ED) is a shared sexual concern of couples I: couple conceptions of ED. J Sex Med. 2009;6:2746–60.
21. Frohlich P, Meston C. Sexual functioning and self-reported depressive symptoms among college women. J Sex Res. 2002;39:321–5.
22. Graziottin A, Althof S. What does premature ejaculation mean to the man, the woman, and the couple? J Sex Med. 2011;8:S304–9.
23. Graziottin A, Basson R. Sexual dysfunction in women with premature menopause. Menopause. 2004;11:766–77.

24. Graziottin A, Brotto LA. Vulvar vestibulitis syndrome: a clinical approach. J Sex Marital Ther. 2004;30:125–39.
25. Graziottin A, Dennerstein L, Alexander JL, Giraldi A, Whipple B. Classification, etiology, and key issues in female sexual disorders. In: Porst H, Buvat J, The Standards Committee of the International Society for Sexual Medicine, editors. Standard practice in sexual medicine. Malden: Blackwell Publishing; 2006. p. 305–14.
26. Graziottin A. Libido: the biological scenario. Maturitas. 2000;34 suppl 1:S9–16.
27. Harris JM, Cherkas LF, Kato BS, Heiman JR, Spector TD. Normal variations in personality are associated with coital orgasmic infrequency in heterosexual women: a population based study. J Sex Med. 2008;5:1177–83.
28. Hertel J, Schütz A, Lammers CH. Emotional intelligence and mental disorder. J Clin Psychol. 2009;65:942–54.
29. Hurd CL. Older women and sexuality: experiences in marital relationships across the life course. Can J Aging. 2006;25:129–40.
30. Jiann B-P, Su C-C, Yu C-C, Wu TT, Huang J-K. Risk factors for individual domains of female sexual function. J Sex Med. 2009;6:3364–75.
31. Kammerer-Doak D, Rogers RG. Female sexual function and dysfunction. Obstet Gynecol Clin North Am. 2008;35(2):169–83.
32. Kaplan HS. Disorders of sexual desire. New York: Brunner/Mazel; 1979.
33. Kirsch LG, Becker JV. Emotional deficits in psychopathy and sexual sadism: implications for violent and sadistic behavior. Clin Psychol Rev. 2007;27:904–22.
34. Klausmann D. Sexual motivation and the duration of the relationship. Arch Sex Behav. 2002;31:275–87.
35. Leiblum S, Rosen R. Principles and practice of sex therapy. New York: Guilford; 2000.
36. Levin R. The physiology of sexual arousal in the human female: a recreational and procreational synthesis. Arch Sex Behav. 2002;31:405–11.
37. Limoncin E, Tomassetti M, Gravina GL, Ciocca G, Carosa E, Di Sante S, Gentile V, Mirone V, Montorsi F, Lenzi A, Jannini EA. Premature ejaculation results in female sexual distress: standardization and validation of a new diagnostic tool for sexual distress. J Urol. 2013;189:1830–5.
38. Maseroli E, Fanni E, Mannucci E, Fambrini M, Jannini EA, Maggi M, Vignozzi L. Which are the male factors associated with female sexual dysfunction (FSD)? Andrology. 2016;4:911–20.
39. Masters WH, Johnson VE. Human sexual response. Boston: Little, Brown & Co.; 1966.
40. Mayer JD, Salovey P, Caruso DR. Models of emotional intelligence. In: Sternberg R, editor. Handbook of intelligence. Cambridge: Cambridge University Press; 2000. p. 396–420.
41. Michetti PM, Rossi R, Bonanno D, Tiesi A, Simonelli C. Male sexuality and regulation of emotions: a study on the association between alexithymia and erectile dysfunction (ED). Inter J Impot Res. 2006;18:170–4.
42. Miner M, Esposito K, Guay A, Montorsi P, Goldstein I. Cardiometabolic risk and female sexual health: the Princeton III summary. J Sex Med. 2012;9:641–51.
43. Palacios S, Castañoa R, Graziottin A. Epidemiology of female sexual dysfunction. Maturitas. 2009;63:119–23.
44. Papaharitou S, Nakopoulou E, Kirana P, Giaglis G, Moraitou M, Hatzichristou D. Factors associated with sexuality in later life: an exploratory study in a group of Greek married older adults. Arch Gerontol Geriatr. 2008;46:191–201.
45. Plaut M, Graziottin A, Heaton J. Sexual dysfunction. Oxford: Health Press; 2004.
46. Qureshi S, Ara Z, Qureshi VF, Al-Rejaie SS, Aleisa AM, Bakheet SA, Al-Bekairi AM. Sexual dysfunction in women: an overview of psychological/psychosocial, pathophysiological, etiological aspects and treatment strategies. PhcogRev. 2007;1:41–8.
47. Rosen NO, Bergeron S, Sadikaj G, Glowacka M, Baxter ML, Delisle I. Relationship satisfaction moderates the associations between male partner responses and depression in women with vulvodynia: a dyadic daily experience study. Pain. 2014;155:1374–83.

48. Salonia A, Munarriz RM, Naspro R, et al. Women's sexual dysfunction: a pathophysiological review. BJU Int. 2004;93:1156–64.
49. Segraves R, Balon R. Sexual pharmacology: fast facts. New York: WW Norton & Co; 2003.
50. Weiss P, Brody S. International Index of Erectile Function (IIEF) scores generated by men or female partners correlate equally well with own satisfaction (sexual, partnership, life, and mental health). J Sex Med. 2011;8:1404–10.
51. Willi J, Burri A. Emotional intelligence and sexual functioning in a sample of Swiss men and women. J Sex Med. 2015;12(10):2051–60.
52. Zhang H, Yip PSF. Female sexual dysfunction among young and middle-aged women in Hong Kong: prevalence and risk factors. J Sex Med. 2012;9:2911–8.

Evaluation Systems of Female Sexual Function

4

Maria Teresa Filocamo and Nadine Di Donato

Abbreviations

ALLOW	Ask, Legitimize, Limitations, Open up, Work together
BISF-W	Brief Index of Sexual Functioning for Women
CSFQ	Changes in Sexual Functioning Questionnaire
DSFI	Derogatis Sexual Functioning Inventory
DSM	Diagnostic and Statistical Manual of Mental Disorders
FSD	Female sexual dysfunction
FSDS	Female Sexual Distress Scale-Revised
FSDS-R	Female Sexual Distress Scale-Revised
FSFI	Female Sexual Function Index
HSDD	Hypoactive sexual desire disorder
MFSQ	McCoy Female Sexuality Questionnaire
PISQ-12	Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire
PLISSIT	Permission, Limited Information, Specific Suggestions, Intensive Therapy
SFQ	Sexual function questionnaire
SHOW-Q	Sexual Health Outcomes in Women Questionnaire
SHOW-Q	Sexual Health Outcomes in Women Questionnaire
SIDI-F	Sexual Interest and Desire Inventory-Female
SQOL-F	Sexual Quality of Life-Female
SSRIs	Selective serotonin reuptake inhibitors
SSS-W	Sexual Satisfaction Scale for Women
WSID-SF	Women's Sexual Interest Diagnostic Interview-Short Form

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_4

Sexuality is a complex process, coordinated by the neurologic, vascular, and endocrine systems.

Sexuality includes family, societal, and religious beliefs and goes sour by aging, health status, and personal experiences. In addition, sexual activity embodies interpersonal relationships; each partner conveys unique attitudes, needs, and responses into the coupling. A breakdown in any of these areas may lead to sexual dysfunction.

Female sexual dysfunction (FSD) is a continuum of psychological and organic disorders focused on sexual desire with interrelated problems of arousal, orgasm, and sexual pain that impairs quality of life for many women [1]. FSD can afflict women of any age, and its severity worsens with the endocrinology of advancing years. Impact is often subtle. FSD may express as apparently unrelated emotional manifestations that could degrade quality of life and family relationships, in social sphere and in the workplace.

Female sexual dysfunction is defined as any problem that may be encountered in the sexual response cycle that deviates from a woman's normal range of functioning [2]. Defining female sexual dysfunction is not as absolute for women because of the qualitative nature of female sexual function. What may be abnormal for one woman may be normal for another woman [3]. Sexual dysfunction falls on a continuum with female sexual disorder [3].

In sexual dysfunction, there is a break in normal sexual functioning at one or many points in the sexual response cycle. In comparison, a sexual disorder consists of both the sexual dysfunction element in addition to persistent distress [4]. An abnormality in one's sex life can exist but may not justify further evaluation except when the woman experiences a certain degree of distress over it. When investigating female sexual function versus dysfunction and disorder, distress has to be included and is perhaps the most important variable because of the large range of what can otherwise be normal for women [5]. The distress must be experienced by the woman herself, and that which bothers her partner alone is not then a sexual dysfunction of the woman but rather of her partner [6]. Female sexual dysfunction and disorder must be debated in the context of each individual woman's life, culture, social, individual experiences, relationship, and health in order to extricate the distress element.

Sexual problems are highly prevalent in women. In the USA, approximately 40 % of women have sexual concerns, and 12 % report distressing sexual problems [7].

Female sexual dysfunction appears in different forms, including lack of sexual desire, impaired arousal, inability to achieve orgasm, or pain with sexual activity.

Female sexual dysfunction can be subdivided into desire, arousal, orgasmic, and sexual pain disorders. Sexual pain disorders include dyspareunia and vaginismus.

The diagnosis of female sexual dysfunction requires the physician to obtain a detailed patient history that defines the dysfunction, identifies causative or confounding medical or gynecologic conditions, and evokes psychosocial informations.

The sexual dysfunction should be defined in terms of onset and duration and situational versus global manifestations. A situational dysfunction occurs with a specific partner, in a certain setting or in a definable circumstance.

The presence of more than one dysfunction should be assessed, because considerable interdependence may exist. A patient complaining about decreased desire might have an orgasmic disorder from insufficient stimulation, with decreased desire developing secondarily as a result of unsatisfying sexual encounters.

4.1 Female Sexual Dysfunction: Revised Classification of Sexual Dysfunction (DSM-V)

The Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria proved to be in a constant of evolution [8]. The first edition of the DSM, in 1952, cataloged 60 categories of abnormal behavior. By 1994, the fourth edition (DSM-IV) listed 297 separate disorders and over 400 specific psychiatric diagnoses [9]. As with other disorders, DSM criteria for sexual dysfunctions reflect the prevailing psychiatric thinking of the time of publication; they have thus evolved throughout the years, reflecting advancements in the understanding of sexual disorders. Diagnostic categories of female sexual interest as described in the DSM-IV 1994 (American Psychiatric Association 1984) were based on the model of human sexual response proposed by Masters and Johnson [10] and further developed by Kaplan [11]. However, recent research has put into question the validity of that model; both the strict distinction between different phases of arousal and the linear model of sexual response were found to inadequately explain sexual behavior, particularly in women [12, 13]. This has in turn led to several proposed changes in sexual dysfunction diagnostic criteria [14]. The DSM-V, published in May of 2013, presents changes in the sexual dysfunction chapter in an attempt to correct, expand, and clarify the different diagnoses and their respective criteria. Although many of the changes are subtle, some are noteworthy: gender-specific sexual dysfunctions were added, and female disorders of desire and arousal were amalgamated into a single diagnosis called "female sexual interest/arousal disorder" [15]. The classification of sexual dysfunctions was simplified. There are now only three female dysfunctions as opposed to five in the DSM-IV. Female hypoactive desire dysfunction and female arousal dysfunction were merged into a single syndrome called sexual interest/arousal disorder. Similarly, the formerly separate dyspareunia and vaginismus are now called genito-pelvic pain/penetration disorder. Female orgasmic disorder remains in place. Unlike its predecessor, the DSM-V includes the requirement of experiencing the disorder 75–100 % of the time to make any diagnosis of sexual disorder, with the notable exception of substance or medication-induced disorders. Moreover, there is now a required minimum duration of approximately 6 months. Finally, in order to make a diagnosis, the disorder must be deemed to have caused significant distress (the DSM-IV requirement of "interpersonal difficulty" was removed). A new group of criteria called "associated features" was also introduced. It is subdivided into five categories: (1) partner factors (e.g., partner sexual problem, partner health status); (2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); (3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric

comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); (4) cultural or religious factors (e.g., inhibitions related to prohibitions against sexual activity or pleasure, attitudes toward sexuality); and finally (5) medical factors relevant to prognosis, course, or treatment. The criteria of the newly introduced female disorder of sexual interest/arousal are based on those of hypoactive desire disorder. Three out of six criteria are required for diagnosis. As for the diagnosis of female orgasmic disorder, one or both of the following should be present 75–100 % of the time: the absence, infrequency or delay of orgasm, and/or reduced intensity of said orgasm. Regarding the new genito-pelvic pain/penetration disorder, one of the following should occur persistently or recurrently to establish a diagnosis: difficulty in vaginal penetration; marked vulvovaginal or pelvic pain during penetration or attempt at penetration; fear or anxiety about pain in anticipation of, during, or after penetration; and tightening or tensing of pelvic floor muscles during attempted penetration. In conclusion, the DSM-V seeks to remedy some of the inconsistencies of the previous edition. One of the major changes that the DSM-V introduces to the classification of sexual dysfunctions is the merger of sexual disorders of desire and arousal in females. Researchers who advocated this amalgamation [16] based their recommendations on a large body of research suggesting that the separation may have been artificial. Another important change was the fusion of the diagnoses of dyspareunia and vaginismus into a single entry named genito-pelvic pain/penetration disorder. This decision was based on the conclusion that the two disorders could not be reliably differentiated, for two main reasons. Firstly, the diagnostic formulation of vaginismus as "vaginal muscle spasm" was not supported by empirical evidence [17]. Secondly, fear of pain or fear of penetration is commonplace in clinical descriptions of vaginismus [17]. Kaplan even describes it as "phobic avoidance" [11]. Carvalho et al., after testing five alternative models of female sexual function, concluded that the diagnoses vaginismus and dyspareunia overlapped to a great degree [18]. The new edition with the aim to increase the validity and clinical usefulness of DSM introduced also duration and frequency requirements for sexual disorders. All diagnoses except substance- and medication-induced sexual dysfunction now require a minimum duration of approximately 6 months as well as the presence of symptoms 75–100 % of the time. This development corrects what was seen as a flaw in sexual dysfunction diagnostic criteria, especially when compared to other DSM-IV diagnoses which did have duration requirements.

Dyspareunia is defined as recurrent or persistent genital pain associated with sexual intercourse that is not caused exclusively by lack of lubrication or by vaginismus [19]. Diagnosis of dyspareunia is made when the pain causes marked distress or interpersonal difficulty. Dyspareunia can involve pain on entry or deep pain. Painful entry is typically reflective of vulvodynia, inadequate lubrication, or vaginismus. Dyspareunia, which is frequently linked with hypoactive sexual desire disorder (HSDD), has the same situational and psychosocial causes and thus resolves in response to treatment of HSDD. In other cases it is linked with gynecologic disorders, such as endometriosis or vestibulitis, chronic medical conditions, or drugs [20, 21].

4.2 Assessing of Women with Sexual Disorders

Normal versus abnormal sexual functioning in women is poorly understood, although the concept of normal female sexual function continues to develop. A complete history combined with a detailed physical examination is necessary for the evaluation of any women with sexual complaints or concerns. Although laboratory evaluation is rarely helpful in diagnosis or treatment, it may be indicated in women with abnormal physical examination findings or suspected comorbidities. Female sexual dysfunction has been linked to hypertension and its treatment, coronary artery disease, diabetes mellitus, other endocrinological disorders (hyperprolactinemia), and chronic renal failure. The same vascular aberrations that cause erectile dysfunction in men associated with cardiovascular risk factors can cause arousal difficulties in women. Effects of pelvic surgery and hormonal changes associated with menopause are known.

Physicians are often not accustomed with and poorly educated about obtaining a complete sexual history [22] even though this is an important component of primary health care [23]. There are a number of validated self-report and interview-based tools for assessing female sexual dysfunction, but they are mainly used in research settings [24]. The brief sexual symptom checklist is a self-report tool that may be useful in the primary care setting as in addition to a complete sexual history [25]. The checklist includes four basic questions to determine the patient's satisfaction with her sexual function, details about specific sexual problems, and the willingness of the patient to discuss these problems with the physician [25].

Discussions about sexuality should begin with open-ended questions. If a sexual concern is identified, a detailed history that includes menstrual, obstetric, reproductive, and sexual histories should be done; status of current relationships and sexual activity should be required, so as family and personal beliefs about sexuality, and history of sexual trauma or abuse [25]. Additional elements of the history include medical and surgical history; medication use, including herbal supplements; alcohol, tobacco, and illicit drug use; family history; and birth control method. Several medical conditions and medications are associated with sexual dysfunction.

The Permission, Limited Information, Specific Suggestions, Intensive Therapy (PLISSIT) or Ask, Legitimize, Limitations, Open up, Work together (ALLOW) method can be used to facilitate discussions about sexual concerns and initiation of treatment [25–27] (Table 4.1).

Although physical examination findings are often normal, a complete inspection, including a focused pelvic check, can identify pathology and provide patient education about normal anatomy and reassurance that no abnormality is present. The pelvic examination can detect evidence of low hormone levels, infection, hypo- or hypertonicity of pelvic floor muscles, adhesions, and tenderness. The remaining physical examination focuses on mental status, blood pressure, musculoskeletal, thyroid, breast, and neurologic abnormalities. Abnormal findings are more likely in older women, in women with known gynecologic pathology or chronic systemic disease [28].

Table 4.1 Models for initiating discussion and treatment of female sexual dysfunction

ALLOW
Ask the patient about sexual function and activity
Legitimize problems, and acknowledge that dysfunction is a clinical issue
Identify limitations to the evaluation of sexual dysfunction
Open up the discussion, including potential referral
Work with the patient to develop goals and a management plan
PLISSIT
Obtain permission from the patient to discuss sexuality (e.g., "I ask all my patients about their sexuality, is that okay to do with you now?")
Give limited information (e.g., inform the patient about normal sexual functioning)
Give specific suggestions about the patient's particular complaint (e.g., advise the patient to practice self-massage to discover what feels good to her)
Consider intensive therapy with a sexual health subspecialist

ALLOW Ask, Legitimize, Limitations, Open up, Work together; *PLISSIT* Permission, Limited Information, Specific Suggestions, Intensive Therapy

Laboratory evaluation is rarely helpful; however, a focused evaluation is appropriate, particularly if the history or examination suggests a medical condition. Although some experts advocate testing hormone levels in postmenopausal women or in women with decreased desire or arousal, there is no reliable correlation between hormone levels and sexual function [28].

Sexual dysfunction may be the manifestation of psychiatric illness or an adverse effect of psychotropic medication use [29]. If a woman has sexual complaints while taking a psychotropic medication, a detailed history is necessary to identify the etiology [29]. The use of selective serotonin reuptake inhibitors (SSRIs) is a common cause of medication-induced female sexual dysfunction, although all antidepressant classes can cause dysfunction [30]. SSRIs most commonly cause delayed or absent orgasm and decreased libido [30]. The incidence of SSRI-induced sexual dysfunction is estimated to be 30–50 % [31].

4.3 Instrument for the Assessment of Sexual Function

Different types of questionnaires have been used in the last few years to investigate sexual function and dysfunction [21, 32, 33]. Studies of human sexuality are inclined to bias and have varied confounding factors because of the large cultural framework as well as psychosocial factors that distinguish this aspect of human behavior.

Due to its subjective and multifactorial nature and relationship with emotional processes, sexual function needs adequate instruments for assessment, like self-report questionnaires, investigating different aspects of sexual life with high reliability and validity. Thus, to provide a multidimensional assessment of sexual function, a number of self-administered questionnaires have been developed in the recent past years. Several of these measures have demonstrated adequate psychometric properties, especially test–retest reliability, internal consistency, and

discriminant validity. Different types of questionnaires were used to investigate sexual dysfunction of women, such as the Derogatis Sexual Functioning Inventory (DSFI), the sexual function questionnaire (SFQ), the Female Sexual Function Index (FSFI), the Female Sexual Distress Scale-Revised (FSDS-R), and the Sexual Health Outcomes in Women Questionnaire (SHOW-Q).

All these instruments have been developed during the recent past, with the majority having been validated during the past decade. The questionnaires vary mainly in terms of their levels of comprehensiveness; however, all have developed well against established psychometric criteria and have appeared empirical evidence of reliability and validity. A good standardized instrument should evaluate and measure multiple domains, should have internal consistency (α) of at least 0.70 for all domains, and should demonstrate test–retest reliability into an interval of 2–4 weeks of at least 0.50 for items that should display stability over time (Table 4.2).

The Brief Index of Sexual Functioning for Women (BISF-W) was developed in response to the lack of a brief, standardized self-report measure of overall sexual function in women [34]. Previous self-report measures have been either excessively

Table 4.2 Resume questionnaires on sexual function present in scientific literature

Inventory	Published norms	Interpretive levels
BISF-W	Functional women/surgically menopausal women	22 questions, seven domains, total score
CSFQ	Depressed/nondepressed women	5 domains; total score
DISF	Community sample (T scores)	5 domains; total score
FSFI	FSAD/HSDD/FOD/dyspareunia/functional women	5 domains; 19 items; total score
SFQ	Functional/dysfunctional women	7 domains; 26 item; total score
MFSQ *	Functional/dysfunctional women	2 domains; 14 items; total score
FSDF	Functional/dysfunctional women	13 items; total score
PISQ-12	UI/POP	12 items; total score
SIDI-F	HSDD/FOD/functional women	13 items; total score
WSID-SF	HSDD/postmenopausal women	9 items; total score
SQOL-F	Functional/dysfunctional women, spinal cord-injured women/quality of life	18 items; total score
SSS-W	Functional/dysfunctional women	5 domains; total score
SHOW-Q	Functional/dysfunctional women, same-sex relationship	12 items; total score

limiting or inappropriate for the use in large-scale clinical trials. The BISF-W consists of 22 items, assessing the major dimensions of sexual desire, arousal, orgasm, and satisfaction [34]. Several items were adapted from the CSFQ, particularly those assessing frequency of sexual behavior, fantasy, masturbation, and sexual preference. Additional items were included to address specific issues supposed to concern women's sexual functioning and satisfaction, such as body image, partner satisfaction, and sexual anxiety. Several items were designed to evaluate sexual performance difficulties in women, such as diminished arousal or lubrication, pain or tightness during intercourse, and difficulties in reaching orgasm. Items assessing the impact of health problems on sexual functioning are also included [34].

The Changes in Sexual Functioning Questionnaire (CSFQ) was developed with specific versions for females and males to assess sexual functioning in all the domains of the sexual response cycle [35]. It was developed to be used in both clinical and research settings. CSFQ-W is a 35-item instrument identifying five scales of sexual functioning. The original CSFQ items were tested and revised on the basis of conceptual content to ensure that five aspects of sexual functioning (i.e., sexual desire, sexual frequency, sexual satisfaction, sexual arousal, and sexual completion) were evaluated [35]. The CSFQ was used clinically and had the particularity to include a section identifying the sexual pattern of the individual, which permitted information about how much sexual change someone experienced over time [35]. In addition, information on drugs use was collected.

Information about changes could be correlated to the five domains of sexual functioning, so that the clinician could better focus on strategically targeted treatment for the cause of the problem, which could be related to medication, illness, relationship problems, or a combination of difficulties [35]. In addition, the CSFQ addressed the need for an assessment instrument that could differentiate current sexual dysfunction from previous "normal" sexual function and/or lifelong sexual dysfunction [35].

The Derogatis Interview for Sexual Functioning (DISF/DISF-SR) is a coordinated set of brief matched instruments designed to provide an estimate of the quality of an individual's current sexual functioning [36]. The DISF is semi-structured interview comprised of 25 items and reflects quality of sexual functioning in a multi-domain format. The DISF-SR is a matching self-report inventory designed to achieve the same goal in a patient self-report mode. All instruments in the DISF series are designed to be interpreted at three distinct levels: discrete items, functional domains, and aggregate summary (total) score [36].

DISF items are arranged into five primary domains of sexual functioning: sexual cognition/fantasy, sexual arousal, sexual behavior/experience, sexual orgasm, and sexual drive/relationship. In addition, an aggregate DISF total score is computed which summarizes quality of sexual functioning across the five primary DISF domains [36]. Both the DISF and the DISF-SR take approximately 12–15 min to administer. Internal consistency reliabilities for measures of the DISF-SR are well within acceptable ranges, as are test–retest temporal stability coefficients. The DISF/DISF-SR has demonstrated good discriminative validity and sensitivity to treatment-induced changes and is currently available in 12 foreign languages [36].

Female Sexual Function Index (FSFI) is made up of 19 items encompassing the six domains: desire (items 1–2), arousal (items 3–6), lubrication (items 7–10), orgasm (items 11–13), satisfaction (items 14–16), and pain (items 17–19). The total FSFI score is the sum of all points, and the higher the score, the better the sexuality. Sexual dysfunction was defined as an FSFI score < 26.55, based on the published validation studies [37, 38]. A very good discriminate validity and ability to predict the prevalence of sexual problems have been reported [39, 40].

The sexual function questionnaire (SFQ) is a self-report questionnaire designed to measure female sexual function [41]. It is comprised of 28 items reflecting all aspects of the sexual response cycle—desire, arousal, and orgasm—as well as dyspareunia. Factor analysis produced seven domains of female sexual function: desire, physical arousal–sensation, physical arousal–lubrication, enjoyment, orgasm, dyspareunia, and partner relationship [41]. The item content of the SFQ was reviewed by an external panel of clinicians with expertise in psychology, physiology, gynecology, physical medicine, and the treatment of FSD. Internal consistency of the domains ranged from 0.79 to 0.91 for all domains except partner relationship, which was 0.65, and test–retest reliability is in the acceptable range [41].

The McCoy Female Sexuality Questionnaire (MFSQ) was developed from the questionnaire used in a longitudinal study of the menopausal transition and designed to measure aspects of female sexuality likely to be affected by changing sex hormone levels [42]. The original questionnaire was revised to insure that questions were easy to understand and that labels for the Likert scales described a continuum. The revised MFSQ contains 19 questions, 18 items using 7-point Likert scales with labels at the center and endpoints, and one item requesting a frequency of activity [42]. Seven studies involving both clinical and convenience samples and two with double-blind randomized controlled trials used 7, 9, 10, or 17 MFSQ items and demonstrated acceptable reliability, internal consistency, apparent face, and content validity as well as considerable evidence of construct validity [43]. Results showed selected MFSQ item ratings decreased as women progressed through the menopausal transition, varied positively with endogenous estradiol and androgen levels, were higher in postmenopausal women receiving hormone replacement therapy (HRT), and differentiated between different types of oral contraceptives and the presence or absence of ovaries [44].

The Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire (PISQ-12) is a validated and reliable short form that evaluates sexual function in heterosexual women with urinary incontinence and/or pelvic organ prolapse and predicts long-form scores [45]. The PISQ-12 was able to distinguish between women with low or high sexual functioning scores as measured by the SHF-12, a validated sexual function questionnaire that serves as a gold standard. Short forms are useful in the clinical setting because they reduce the time and burden to the patient and provide the clinician with objective means of evaluating functional outcomes of either medical or surgical interventions. In the research setting, a short form is useful when quality-of-life analysis is part of the armamentarium used to evaluate outcomes and compare results [45].

The Sexual Interest and Desire Inventory-Female (SIDI-F) is a clinician-administered instrument that was developed to measure severity and change in response to treatment of HSDD [46]. Seventeen items were included in a preliminary version of the SIDI-F, including 10 items related to desire and seven items related to possible comorbid factors (e.g., other kinds of sexual dysfunction, general relationship satisfaction, mood, and fatigue) [46]. It is a brief, clinician-administered rating scale designed to assess severity of HSDD symptoms in women. Analyses show that majority of the items of the SIDI-F function well in discriminating individual differences in HSDD severity [47]. The validity of the SIDI-F as a measure of HSDD severity was confirmed by a number of observations. Women with a clinical diagnosis of HSDD had significantly lower SIDI-F scores than women not meeting diagnostic criteria for any subtype of female sexual dysfunction and women diagnosed with female orgasmic disorder [48]. There was a high correlation between scores on the SIDI-F and scores on the FSFI and an interactive voice response version of the CSFQ. A cutoff score of 33 was proved to indicate the presence of HSDD [48].

Another questionnaire focused on HSDD is the Women's Sexual Interest Diagnostic Interview-Short Form (WSID-SF) structured and validated by Derogatis et al. in 2008 to identify hypoactive desire disorders [49]. The authors used this tool on 629 postmenopausal women, and WSID-SF was demonstrated to have good specificity and sensitivity (discriminant validity) in identifying HSDD when compared with FSFI, MFSQ, and FSDS. The intraclass correlation coefficient was 0.80 for women with HSDD and 0.84 for women without HSDD [50].

The Sexual Quality of Life-Female (SQOL-F) questionnaire is a short instrument that specifically assesses the relationship between female sexual dysfunction and quality of life. Symonds and coworkers developed the questionnaire in 2005 [51]. The basis for the generation of the SQOL-F questionnaire was Spitzer's Quality of Life (QOL) model that involved physical, emotional, psychological, and social components. Validity of the SQOL-F questionnaire first was assessed in the UK and the USA. In the UK setting, studying a sample of 1296 women aged 18–65 years, internal consistency was found to be 0.95, and the questionnaire discriminated well between depressed and not depressed women [51]. In the USA setting, studying three groups of women (women with spinal cord injury, women with sexual dysfunction, and a sample of healthy women), the SQOL-F was lower among women with sexual dysfunction as expected lending support to its discriminate validity. In addition, intraclass correlation coefficient was reported to be 0.85, which showed an appropriate stability for the questionnaire [51].

The Sexual Satisfaction Scale for Women (SSS-W) represents a brief, 30-item, multifaceted measure of women's sexual satisfaction [52]. It exhibits sound psychometric properties and has a demonstrated ability to discriminate between clinical and nonclinical populations. The final SSS-W consists of five domains (two relational, three personal) of six items each: communication, compatibility, contentment, relational concern, and personal concern. Items in the communication, compatibility, and contentment domains were written to reflect themes relating to sexual satisfaction noted in prior literature. The SSS-W was developed to provide a

comprehensive measure of sexual satisfaction and sexual distress that would benefit researchers and clinicians interested in further understanding what constitutes sexual satisfaction in women and how it relates to levels of sexual functioning [52].

The standardized Female Sexual Distress Scale-Revised (FSDS-R) is a screening instrument consisting of 13 items for measuring sexually related personal distress. The fixed choice response format offered the five increments: "never," "rarely," "occasionally," "often," and "always." Sexual distress was defined as a FSDS-R score > 11, based on the published validation studies. The higher the score, the greater the distress [53]. A very good discriminate validity and ability to predict the prevalence of sexual problems have been shown for this instrument [54].

Sexual Health Outcomes in Women Questionnaire (SHOW-Q) is formed by 12 items organized conceptually to include 2–3 items per domain: satisfaction with sex, orgasm frequency, sexual desire, and pelvic problem interference with sex. Factor analysis demonstrated a 12-item scale with high internal consistency reliability (Cronbach's α=0.86) and four reliable subscales (α=0.73 to 0.84). SHOW-Q involves women of diverse sociodemographic and clinical background, including women in same-sex relationships and women who are sexually active without a partner as well as sexually inactive women. SHOW-Q investigates also different aspects of sexual life and pelvic problem interference with sex [55].

Although self-administered questionnaires offer a valid and user-friendly means of assessing sexual function, several limitations should be noted.

First, these measures provide information only on current level of sexual function and cannot substitute for a detailed sexual, psychological, or medical history. Furthermore, the current questionnaires do not provide information on specific background or etiology or the role of comorbid medical or psychiatric conditions. Additionally, some patients may experience discomfort or embarrassment while completing questionnaires or symptom scales or may have difficulty with comprehension. Steps should always be taken to ensure privacy and confidentiality and to assist the patient with comprehension when indicated. Finally, questionnaire or symptom scales should not be used as an alternative to or substitute for direct inquiry or face-to-face clinical interaction with the clinician.

References

1. Buster JE. Sex and the 50 something woman: strategies for restoring satisfaction. Contemp Ob Gyn. 2012;8:32–9.
2. Sobczak JA. Female sexual dysfunction: knowledge development and practice implications. Perspect Psychiatr Care. 2009;45:161–72.
3. Basson R. Women's sexual dysfunction: revised and expanded definitions. CMAJ. 2005;172:1327–33.
4. Clayton AH. Epidemiology and neurobiology of female sexual dysfunction. J Sex Med. 2007;4 suppl 4:260–8.
5. Domoney C. Sexual function in women: what is normal? Int Urogynecol J Pelvic Floor Dysfunct. 2009;20 suppl 1:s9–17.
6. Kammerer-Doak D, Rogers RG. Female sexual function and dysfunction. Obstet Gynecol Clin North Am. 2008;35:169–83.

7. Stuckey BG. Female sexual function and dysfunction in the reproductive years: the influence of endogenous and exogenous sex hormones. J Sex Med. 2008;5:2282–90.
8. Balon R. The DSM criteria of sexual dysfunction: need for a change. J Sex Marital Ther. 2008;34:186–97.
9. Ratey JJ, Johnson C. Shadow syndromes. New York: Pantheon Books; 1997.
10. Masters WH, Johnson VE. Human sexual response. New York: Bantam; 1966.
11. Kaplan HS. The new sex therapy. New York: Brunner/Mazel; 1974.
12. Graham CA, Sanders SA, Milhausen RR, McBride KR. Turning on and turning off: a focus group study of the factors that affect women's sexual arousal. Arch Sex Behav. 2004;33:527–38.
13. Janssen E, McBride KR, Yarber W, Hill BJ, Butler SM. Factors that influence sexual arousal in men: a focus group study. Arch Sex Behav. 2008;37:252–65.
14. Basson R, Leiblum S, Brotto L, Derogatis L, Fourcroy J, et al. Revised definitions of women's sexual dysfunction. J Sex Med. 2004;1:40–8.
15. American Psychiatric Association. DSM-5: diagnostic and statistical manual for mental disorders. 5th ed. New York: American Psychiatric Press; 2013.
16. Graham CA. The DSM diagnostic criteria for female sexual arousal disorder. Arch Sex Behav. 2010;39:240–55.
17. Binik YM. The DSM diagnostic criteria for vaginismus. Arch Sex Behav. 2010;39:278–91.
18. Carvalho J, Vieira AL, Nobre P. Latent structures of female sexual functioning. Arch Sex Behav. 2012;41:907–17.
19. Basson R, Berman J, Burnett A, et al. Report of the International consensus development conference on female sexual dysfunction: definitions and classifications. J Urol. 2000;163:888–93.
20. Boyer SC, Goldfinger C, Thibault-Gagnon S, et al. Management of female sexual pain disorders. Adv Psychosom Med. 2011;31:83–104.
21. Fritzer N, Haas D, Oppelt P, et al. More than just bad sex: sexual dysfunction and distress in patients with endometriosis. Eur J Obstet Gynecol Reprod Biol. 2013;169:392–6.
22. Clayton AH. Sexual function and dysfunction in women. Psychiatr Clin North Am. 2003;26(23):673–82.
23. Nusbaum MRH, Hamilton CD. The proactive sexual health history. Am Fam Physician. 2002;66(9):1705–12.
24. Meston CM, Derogatis LR. Validated instruments for assessing female function. J Sex Marital Ther. 2002;28 suppl 1:155–64.
25. Hatzichristou D, Rosen RC, Broderick G, et al. Clinical evaluation and management strategy for sexual dysfunction in men and women. J Sex Med. 2004;1(1):49–57.
26. Feldman J, Striepe M. Women's sexual health. Clin Fam Pract. 2004;6(499):839–61.
27. Sadovsky R. The role of the primary care clinician in the management of erectile dysfunction. Rev Urol. 2002;4 suppl 3:S54–63.
28. Berman JR. Physiology of female sexual function and dysfunction. Int J Impot Res. 2005;17 suppl 1:S44–51.
29. Bldwin D, Mayer A. Sexual side-effects of antidepressant and antipsychotic drugs. Adv Psychiatr Treat. 2003;9:202–10.
30. Werneke U, Northey S, Bhugra D. Antidepressants and sexual dysfunction. Acta Psychiatr Scand. 2006;114(6):384–97.
31. Balon R. SSR-I associated sexual dysfunction. Am J Psychiatry. 2006;163(9):1504–9.
32. Montanari G, Di Donato N, Benfenati A, et al. Women with deep infiltrating endometriosis: sexual satisfaction, desire, orgasm, and pelvic problem interference with sex. J Sex Med. 2013;10:1559–66.
33. Mabrouk M, Montanari G, Di Donato N, et al. What is the impact on sexual function of laparoscopic treatment and subsequent combined oral contraceptive therapy in women with deep infiltrating endometriosis? J Sex Med. 2012;9:770–8.
34. Taylor JF, Rosen RC, Leiblum SR. Self-report assessment of female sexual function: psychometric evaluation of the Brief Index of Sexual Functioning for Women. Arch Sex Behav. 1994;23(6):627–43.

35. Clayton AH, McGarvey EL, Clavet GJ. The changes in Sexual Functioning Questionnaire (CSFQ): development, reliability and validity. Psychophaemacol Bull. 1997;33(4):731–45.
36. Derogatis LR. The Derogatis interview for sexual functioning (DISF, DISF-SR): an introductory report. J Sex Marital Ther. 1997;23:291–304.
37. Rosen R, Brown C, Heiman J, et al. The Female Sexual Function Index (FSFI): a multidimensional self-report instrument for the assessment of female sexual function. J Sex Marital Ther. 2000;26:191–208.
38. Wiegel M, Meston C, Rosen R. The female sexual function index (FSFI): cross-validation and development of clinical cutoff scores. J Sex Marital Ther. 2005;31(1):1–20.
39. Ter Kuile MM, Brauer M, Laan E. The Female Sexual Function Index (FSFI) and the Female Sexual Distress Scale (FSDS): psychometric properties within a Dutch population. J Sex Marital Ther. 2006;32:289–304.
40. Filocamo MT, Serati M, Li Marzi V, et al. The Female Sexual Function Index (FSFI): linguistic validation of the Italian version. J Sex Med. 2014;11(2):447–53.
41. Symonds T, Abraham L, Bushmakin AG, et al. Sexual function questionnaire: further refinement and validation. J Sex Med. 2012;9(10):2609–16.
42. McCoy N. The McCoy female sexuality questionnaire. Qual Life Res. 2000;9:739–45.
43. Rellini AH, Nappi RE, Vaccaro P, et al. Validation of the McCoy female sexuality questionnaire in an Italian sample. Arch Sex Behav. 2005;34(6):641–7.
44. Nijland EA, Weijmar Schultz WC, Davis SR. Effects of Tibolone and raloxifene on health-related quality of life and sexual function. Maturitas. 2007;58(2):164–73.
45. Rogers RG, Coates KW, Kammerer-Doak D, et al. A short form of the Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire (PISQ-12). Int Urogynecol J Pelvic Floor Dysfunct. 2004;14(3):164–8.
46. Clayton AH, Segraves RT, Leiblum S, et al. Reliability and Validity of sexual Interest and Desire Inventory-Female (SIDI-F), a scale designed to measure severity of female hypoactive sexual desire disorder. J Sex Marital Ther. 2006;32(2):115–35.
47. Sills T, Wunderlich G, Pyke R, et al. The Sexual Interest and Desire Inventory- Female (SIDI-F): item response analyses of data from women diagnosed with hypoactive sexual desire disorder. J Sex Med. 2005;2(6):801–18.
48. Clayton AH, Segraves RT, Bakish D, et al. J Womens Health (Larchmt). 2012;19(12):2191–5.
49. DeRogatis LR, Allgood A, Rosen RC, et al. Development and evaluation of the Women's Sexual Interest Diagnostic Interview (WSID): a structured interview to diagnose hypoactive sexual desire disorder (HSDD) in standardized patients. J Sex Med. 2008;5(12):2827–41.
50. DeRogatis LR, Allgood A, Auerbach P, et al. Validation of a Women's Sexual Interest Diagnostic Interview-Short Form (WSID-SF) and a Daily Log of Sexual Activities (DLSA) in postmenopausal women with hypoactive sexual desire disorder. J Sex Med. 2010;7(2 pt 2):917–27.
51. Symonds T, Boolell M, Quirk F, et al. Development of a questionnaire on sexual quality of life in women. J Sex Marital Ther. 2005;31:385–97.
52. Meston C, Trpnell P. Development and validation of a five factor sexual satisfaction and distress scale for women: the Sexual Satisfaction Scale for Women (SSS-W). J Sex Med. 2005;2(1):66–81.
53. Derogatis L, Clayton A, Lewis-D'Agostino D, et al. Validation of the female sexual distress scale-revised for assessing distress in women with hypoactive sexual desire disorder. J Sex Med. 2008;5:357–64.
54. Ter Kuile MM, Weijenborg PT, Spinhoven P. Sexual functioning in women with chronic pelvic pain: the role of anxiety and depression. J Sex Med. 2007;7:1901–10.
55. Learman LA, Huang AJ, Nakagawa S, et al. Development and validation of a sexual functioning measure for use in diverse women's health outcome studies. Am J Obstet Gynecol. 2008;198:710.e1–8.

Becoming a Woman: When and Why Gender Dysphoria May Challenge the Basic Steps of Women's Sexual Identity

5

Alessandra Graziottin

5.1 Introduction

Becoming a woman is a long path. It starts even before conception, when dreaming about the child to come is shaped by parent's expectations, fantasies, needs and desires. It is shaped during the prenatal life, first of all by chromosomic, genetic and endocrine factors, modulated by complex – and yet little understood – metabolic, psychosexual and environmental factors. The *uterine black box* is indeed the secret setting of powerful biological and subtle psychosexual dynamics. It is powerfully modulated in the postnatal life by endocrine, affective, psychosexual and contextual factors.

In times when gender identity disorders (GID), namely, gender dysphoria (GD), are increasing, physicians are requested to consult in areas where little or no information and learning were offered during the standard medical training. The long path of "becoming a woman" is therefore presented here with the special perspective of the many challenges a child and an adolescent girl have to face when they are not that happy of being and feeling female. The goal is to offer insight and considerations on the process of gender identity construction in girls, rooted in current guidelines and inspired by the author's clinical experience, to empower colleagues' confidence when dealing with such a delicate and multifaceted issue [1–7].

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_5

5.2 The Challenge of Women's Sexuality

Human sexuality encompasses three major dimensions: *sexual identity*, *sexual function* and *sexual relationship* [2]. *Gender identity*, *gender role* and *gender orientation* are considered major contributors of sexual identity [2]. Encompassing the concept of sexuality, a major focus is currently devoted to the gender issues, with three readings:

- *Gender identity* (which overlaps with the concept of sexual identity)
- *Gender role* (a subspecification of the sexual identity)
- *Sexual* (gender) *orientation* [3]

Satisfaction with the gender identity outcome depends on a number of variables that deserve the highest multidisciplinary consideration [2–6].

5.3 The Body's Secrets and Gender Issues

The sense of personal identity and gender identity are rooted first in the physical appearance, as Freud stated back in 1923 [1]. The body issue of gender appearance, the way we look and the way we perceive our look is vital for each of us in the lifespan and, even more so, for children/adolescents with gender dysphoria. Early on, in 1912, Sigmund Freud wrote "anatomy is destiny" [8]: the aspect of the external genitalia at birth is the first social cornerstone of gender identity leading to the attribution of the "anagraphic sex". The description as "male" or "female" usually triggers comprehensive family and social interactions oriented first to appreciate and reinforce the self-perception of the child as either a boy or a girl and, second, his/her adherence to the gender norms of that family and cultural belonging [2, 9].

In *girls*, a rewarding *identification with the mother* (or another affectively persistent/constant significant female caregiver, such as the grandmother in many families) and a satisfying *complementation with the father* (or another affectively persistent/constant respectful male figure) further contribute to develop a solid and satisfied female gender identity [2, 3]. This positive self-perception is well expressed in the statement, "I'm happy to be a girl": feminine, joyful, tender, pretty, vital, energetic, enjoying playing, learning and dynamically interacting with age-mates, friends and adults in the process of expressing herself at best [3].

5.4 The Endocrine Priming of the Brain and Body

5.4.1 Becoming a Girl

To understand the vulnerabilities of the gender identity process, a few words should be devoted to the basic biological steps of gender differentiation. The female genital organs (and the brain) differentiate in the feminine phenotype during the embryonic

period without particular hormonal influences, whilst "becoming a boy" requires active androgens production since the early stages of embryonic development. This biological asymmetry is key for the reading of the biological contributors of gender dysphoria. Indeed the *female is the default programme* [10] exemplified in XO subjects (Turner syndrome): they are infertile, but the external genitalia are female and the behaviour is female. Not one case of gender dysphoria in Turner syndrome women is reported in the literature, in this author's knowledge.

In chromosomic girls, androgens are necessary to partially masculinise the brain, body and genitals. Androgens can be of foetal origin, such as in the adrenogenital syndrome, maternal or, rarely, exogenous. In girls, this author's working hypothesis is that psychodynamic, affective and contextual factors may contribute to a mild GD of "defensive" motivational origin, whilst more severe GD up to a frank "expressive" transsexualism requires an androgenic priming at least of the brain, if not of the genitals.

5.4.2 Becoming a Boy

During the foetal life, the production of androgens at male physiologic levels for the gestational age [10] is essential to differentiate internal and external genitalia into the "male" organs and functions. In boys, a reduced androgenic priming of the brain may lead to the emergence of the original basic female brain, with coherent feeling and behaviours. Opposite to the Bible's narrative, it is Adam that is born by androgen-driven differentiation from Eve's basic body, both in the brain and in the genitals. Inadequate androgen priming may contribute to inadequate development of external genitalia and insufficient brain androgenic priming, up to intersex ambiguous appearance.

There are three key directors of male gonad differentiation:

1. The sex-determining region Y protein (SRY) also known as testis-determining factor (TDF) [11]. It is a protein that in humans is encoded by the SRY gene located in the Y chromosome. Its expression causes the development of primary sex cords, which later develops to seminiferous tubules. These cords form in the central part of the yet-undifferentiated gonad, turning it into a testis. The now-induced Leydig cells of the testis start secreting testosterone, whilst the Sertoli cells produce anti-Mullerian hormone, which inhibits the development of the Mullerian ducts into the female inner genitalia (salpinges, uterus, vagina).
2. Androgens, secreted by the Leydig cells, further "force" the basic programme into the progressively male phenotype.
3. Anti-Mullerian hormone (AMH) produced by Sertoli cells in men and by the granulosa cells of the ovary in women further contributes to direct the process into suppressing the female code whilst enhancing the male one.

The gonadal differentiation takes place since the early gestational weeks; in the second month of the foetal life, the female phenotype depends therefore on the

absence of sexual SRY protein, androgens and anti-Mullerian hormone (AMH), leading to gonads composed of an inner medulla (ovarian stroma) and an outer cortex (parenchyma).

The biological asymmetry in the process of gender differentiation may explain why gender identity disorders are more prevalent in the male gender, although reliable epidemiological data are still lacking [12]. In terms of probability, in chromosomic XY boys, it is more likely that a biologically complex process (the androgenisation of the basic female body and brain) undergoes disruptions and inadequacies leading to a self-perception and "hard-wired" inner body image more adherent to the basic female gender programme than vice versa.

5.5 "I Want to Wear Trousers"

In children and adolescent girls, "I want to be a boy" is the alerting "tip of the iceberg" of a very heterogeneous set of psychodynamic and biological conditions that require the highest empathic clinical attention [13–16].

Indeed gender identity disorders encompass a spectrum of very different motivations, defensive and expressive, perceived first at the emotional level and then progressively at the cognitive one (Table 5.1).

5.5.1 In Flight from Femininity: The Defensive Motivation

At one extreme, clinicians recognise girls *in flight from femininity*: when the female gender is perceived as "the losing one". This is the *defensive* motivation against a female condition recognised as humiliating, restrictive, submissive and abused, in all the cultures and family contexts that still are built on limiting women's right to express themselves at their best whilst imposing a pervading social code of women's inferiority. Predisposing, precipitating and maintaining factors can be considered.

A. *Predisposing factors* – Different biological factors can interact with psychosexual ones:
   - *Biological* gender uneasiness may progress to a severe dysphoria with the contribution of:
     1. *Not yet* detected endocrine factors acting on the brain, during pregnancy, such as high level of maternal stress with an increase of adrenal androgens

Table 5.1 "I wanted to wear *only* trousers": motivations to *behave* as male in prepubertal girls

Defensive against a female condition perceived as submissive, limiting, losing, restrictive and obsolete
Expressive of desires, needs, interests, talents, vocations and passions typical of male children

Modified from Graziottin [39]

2. Subclinical level of adrenal congenital hyperplasia both in foetal life and early childhood
3. Iatrogenic drugs administered in pregnancy [2]

– *Psychosexual*

- *Inadequate identification with the mother* and lack of a meaningful female significant other. The damage for the sexual identity is more pervasive when the mother is perceived as detached, emotionally distant, neglecting, refusing and abusive from the early days/months of life. This could become a prominent cofactor in the girl's progressive refusal of the female gender identity. A parallel, stronger identification with the father, or a significant other positive male in the family, including a loved brother or grandfather, may facilitate the desire/choice of becoming a boy. In girls, when the process of identifying with the same gender parent (the mother) and complementing with the parent of the opposite gender (the father) is disrupted, a major psychodynamic contributor to a gender dysphoria is in play. *Too much father, too little mother* from the early days/months of life well describes the parental scenario contributing to a stronger identification with the father or his stable male surrogate. An androgenic priming during the foetal life may predispose to and potentiate such a shift.
- The *disappointment/delusion about the femininity* at receiving two types of role messages:
    - *Girls must not behave as such*, and similarly restrictive messages, expressed and repeated both with words and reproaching attitudes (sights, feelings, tones of the voice, verbal and physical punishments). The active and vital girl who wants to express herself may feel that her personal talents ("stereotypically" considered appropriate for boys) are literally killed. She may feel a progressive sense of rage, anger and rebellion towards the female "stereotypes". The leading thought, "Why boys can do what they want and I can do nothing?" may gradually drive her inner perception into the thought that "she must be a boy if she feels that way".
    - *Girls must help moms at home,* the early recruitment in all kinds of homework, whilst boys can still play is still active in many cultures and in families of lower socioeconomic setting. Caring of younger sibling, cleaning the house and dresses, ironing, cooking and in many cultures still serving the family men are the only allowed roles. The oppression of being a girl, repressed and humiliated by gender role female duties, may lead to sadness and depression. Girls who do not surrender to this kind of forced and stereotyped female role identity may gradually shift to desire a male gender role if not a full male gender identity.
- *Perception of sexual vulnerability.* The experience of sexual violence, within and/or outside the family, may induce serious disruptions in the growth of sexual identity and sense of self-worth. For these unfortunate children, who have been sexually harassed or abused, being a girl equals being a prey [3]. The need to avoid the perpetuation of the abusive context may induce different "self-protective" dynamics: from "I want to wear only trousers" (still a symbol of male

gender role in many cultures) perceived as an armour, a defence and a key for freedom [17] to "I want myself to become a boy".

B. *Precipitating factors* – A "collapse of awareness" about having GD may be triggered by other key events:
   - *Mediagenic transsexualism*. The increasing media visibility of gender disorder may offer to the confused child and adolescent the "magic" solutions to her inner problems, difficulties and emotional turmoil. A kind of dangerous "self-fulfilling prophecy", more insidious in children with a weak sexual identity, or with non-transsexual gender dysphoria, when they are desperately looking for a definitive, solid identity, whatever it could be. The defensive motivations presented above may lead to a clinical diagnosis of gender dysphoria: this label should be "handed with care" and possibly kept on hold (or expressed as a very mild disorder). Colette Chiland [18], a psychiatrist with an extensive experience with children and adolescent gender disorders, warns against an early use of "diagnostic etiquettes" as such, given the high plasticity of gender identity in the lifespan and maximum at adolescence. She suggests that psychotherapeutic support should be offered to the child/girl and to the family, to improve the psychological well-being *within the gender of birth*. An empathic, skilled and experienced female psychotherapist may do a good job for and with the child. The enormous psychoplasticity of young brains may ease the goal of a satisfactory mediation in a loving and respectful female therapeutic setting.

Precipitating factors include [2, 3]:

- The onset of puberty, with the appearance of breast and periods, forcing the girl to move from a "totipotent, partially undifferentiated identity" to a definite female gender: a shocking discovery for many GD girls [19]
- The perception of a disturbing/unaccepted homosexual drive
- A disturbing drive to masturbation, "unacceptable" for the girl as pleasure derives from the stimulation of the "hated" and somehow "untouchable" female genitalia
- The loss of a very significant relative, often the father or a surrogate male parent (usually the grandfather)

Maintaining factors include on one side the unaddressed persistence of predisposing and precipitating factors and on the other the lack of professional support with a careful evaluation of defensive vs expressive GD conscious and unconscious motivations.

5.5.2 The Expressive Motivation

I'm a boy trapped in a girl's body. This is the core perception of girls at the opposite end of the spectrum of gender dysphoria (with all the mixed motivations in between). The want to *become a boy expresses lifelong desire*, *vocation*, *interests and talents*, *more typical of a male child*. It is not a denial of or a flight from femininity, but the real

feeling of belonging to the male gender, with male "body image" and "body feelings" hard-wired into the brain [20]. Usually these girls are recognised as "boys" from age-mate companions since the first 2–3 years of life. Motivations and emotions to become a boy can be variably enhanced and supported by biological contributors. One of the most powerful biological contributor is brain-derived neurotrophic factors (BDNF) (with current more evidence, however, in male to female GD) [21, 22].

5.6 Emotions, Neurovegetative Pathways, Motor and Hormonal Correlates

The physical appearance is shaped by the emotions that live and express themselves in the neurovegetative physical domain. Desire, anger, fear and panic with separating distress, the four basic emotions command systems [23], immediately express themselves in the whole body (they live within and with the body) and are the first experience we all have of our being alive, loved, disregarded/neglected or hated.

Emotions are not a cloud of feelings over the head. They have very solid somatic correlates, mediated by the *neurovegetative system* (that appears to be up-regulated in persons with GD). They have an immediate *motor expression*: *moving towards* for desire, *fight or flight* for fear, *moving against* for anger and *looking for a comforting presence/hug* for panic with separation distress [23]. Emotions indeed reshape continuously the perception of human body image, further modulated by affective dynamics and mood. Cognitive issues further contribute. Emotions are continuously modulated by *hormones* and by sexual hormones in the foetus and then from puberty onwards. This is why endocrine medical issues are critical in this field. To reach and maintain a new, positive F to M identity requires more than a number of successful operations (but the quality of surgical outcome is certainly a powerful prerequisite).

5.7 Time: The Advantages of a "Pubertal Delay"

"To delay puberty or not"? This is the painful dilemma that parents and health-care providers have to face when looking for the best for a daughter and patient with GD. The appearance of breast, periods and female body shape is a major physical and emotional shock for a girl with lifelong GD. The breast growth elicits negative feelings (girls with GD usually detest it) and triggers the social comments on her "frightening" (as she perceives it) becoming "a real woman". All girls with GD use any type of constrictive and covering garments and attitudes (tight breast fasciae, posture with bent shoulders, oversize pullovers and jackets) to minimise the social perception/appreciation of the breast growth. Why not prevent/delay this growth? Menarche appearance causes a major waste of emotional and physical energy that would be better utilised on the psychosexual work and real-life issues.

Time is key: a right treatment in the wrong moment – for that individual patient – is wrong. The age at puberty determines a basic gender-related, often neglected, somatic fact: *height*. In women, the progressive estrogens' uprise leading to menarche coincides with the end of the height growth, with very few centimetres obtained afterwards in a minority of girls (the taller ones). Estrogens close the long bone cartilages that in childhood allow and modulate the lengthening of long bones. Within the large variety linked to race, family genes, food and light availability, males are usually taller than females. Puberty can be delayed for a few years with GnRH analogue (GnRHa): a kind of "puberty on hold", a "sleeping puberty" as it can be explained to young patients and parents, totally reversible when the final decision – to change sex or not – is finally and more serenely taken. Blocking pubertal development at Tanner stage 2 for prepubertal, gender nonconforming children is a relatively new but reversible and highly beneficial strategy to delay puberty, giving patients and families time to come up with a transition plan – a real life-saving choice [24].

Growth hormone (GH) administered in parallel to GnRHa seems to promote height's growth more than the analogues alone in prepuberal girls with central precocious puberty [25]. GH and GnRHa combined have been used in children with idiopathic short stature, obtaining a height increase of 1.0–1.3 SD [26].

No controlled studies with GH have been carried out so far in GD girl, in this author's knowledge. The issue needs to be explored prospectically to evaluate if the advantages theoretically considered here can be substantiated in GD patients.

As discussed above a *pubertal delay*, from 10–12 up to 14–16 years of age, would offer a number of potential *advantages* to girls with lifelong gender dysphoria (F to M).

5.8 Practical Tips for the Clinician Dealing with Gender Disorders

A few distilled suggestions, rooted from the author's clinical practice and current literature, highlight the importance of a balanced approach to the many biological and psychosexual issues elicited by GD.

– *Biological advantages* that should be discussed with the young patient and her parents include:
  - *Height*: to gain 10–15 cm or more in comparison to the height of a normal puberty would be a major advantage for an F to M girl, in terms of body image, self-perception and sense of personal beauty and worthiness. The physical appearance, and inner self-perception, would further benefit from:
  - *Regular physical exercise* that should be encouraged and recommended: its anti-inflammatory effect may have multiple advantages, physical and emotional. Reduction of neuroinflammation, thanks to a regular physical activity, is a major contributor to a better mood [3].
  - *Stronger muscle growth:* this contributes to the perception of a more solid body shape and body image [27].

- *Better mood:* is to be pursued with healthy lifestyles, physical exercise first. Sports induce a physiologic motor discharge of negative emotions (so high in patients with gender dysphoria), increase endorphins, dopamine and serotonin, thus contributing to a better psychotherapeutic emotional approach to the disturbing gender issues. Every child and adolescent should be encouraged to be active, to play and commit himself/herself to a sport of choice. Indeed *physical exercise* improves brain plasticity, behaving as a kind of *endogenous pharmacotherapy* [28]. It improves the perception of well-being: its usefulness should be explored in controlled studies in girls with GD.
- *Vitamin D level*: it should be checked periodically, and its *supplementation* when appropriate is to be recommended in pre- and postpubertal girls with GD. A prospective study indicate a very significant correlation between hypovitaminosis D and precocious puberty [29, 30], a problem that would be even more relevant in girls with GD, as all their psychosexual problems would be exasperated.
- *Calcium intake:* it should be adequate (at least 1000 mg/day) [31]. Calcium supplementation should be considered when the daily intake is inadequate and in lactase-deficient girls to contribute to an optimal bone mass even if prolonging the prepubertal amenorrhea with GnRHa and GH.
- *Dehydroepiandrosterone (DHEA):* its supplementation could be considered to further support the height and muscle growth. Unfortunately no controlled studies are available on these issues in this author's knowledge. The overall feeling is that these important biological aspects are still underconsidered in the management of lifelong or early-onset GD in girls.

– *Psychosexual advantages* of a pharmacologic (reversible) *delay of puberty*:
  - It potentiates the opportunity to *work on the many psychosexual issues* involved, whilst the girl is still in a "neutral" physical state, not yet overdetermined by the appearance of secondary sex characteristics: a still non-adequately appreciated opportunity. In the Netherlands, gender dysphoric adolescents may be eligible for puberty suppression at age 12, subsequent cross sex hormone treatment at age 16 and gender reassignment surgery at age 18. Initially, a thorough assessment is made of the gender dysphoria and vulnerabilities in functioning or circumstances. Psychological interventions and/or gender reassignment may be offered, with increase well-being in GD adolescent patients [32]. In GD girls, withholding physical medical interventions, such as the pharmacologic pubertal delay, seems currently more harmful to the well-being in both adolescence and adulthood when compared to cases where physical medical interventions were provided [33]. The Japanese guidelines revised in 2012 suggest delaying puberty with GnRHa until the age of 15, after which cross sex hormones may be given if appropriate [34].
  - It *reduces depression and emotional problems*. Ongoing studies have demonstrated that in adolescents treated with GnRH from 12 up to 16 years of

age, behavioural and emotional problems and depressive symptoms decreased, whilst general functioning improved significantly during puberty suppression. The authors conclude that puberty suppression may be considered a valuable contribution in the clinical management of GD in adolescents [35].

Key Point There is a growing consensus that treatment with gonadotropin-releasing hormone analogue and/or cross sex hormones, in collaboration with transgender-competent mental health professionals, is an intervention that appears to be appropriate in carefully selected youth with gender dysphoria [36].

The attitude of working with psychosexual support and medical cross hormonal treatment, whilst postponing surgery after the age of 18, is well defined in the GD M to F scientific literature [37].

Postponing puberty seems to be the most reasonable approach. From the studies that have been published so far, it seems that the benefits outweigh the risks. However, more systematic research in this area is needed to determine the safety of this approach [38].

The advantages include the "protection" from negative symptoms associated with periods. Menarche, a challenging experience per se in GD girls, is even more disturbing if it is associated with heavy periods; dysmenorrhea, which increases significantly with increasing biological and/or psychosexual stress [3, 39, 40]; and premenstrual symptoms (depression, irritability, aggressivity outbursts, headache, food cravings, mastodynia, abdominal bloating) [39, 41] contributing to her perception that menstruation is a curse in such a detested gender of birth. Since the appearance/diagnosis of a GD, encouraging sport, music, dancing, all the activities that improve the girl's personal skills should be encouraged with the girl and the family: they may offer a natural positive experience of the body reality, increase self-esteem and age-mates' respect, favour social connections whilst reducing the tendency to pursue the monomanic obsession of gender concerns and the tendency to isolate in a kind of virtual life. Given the complexity of factors and the importance of the therapeutic decisions, hormone treatment for pubertal suppression and subsequent gender transition needs to be always *individualised* within stringent protocols in multidisciplinary specialist units [42].

5.9 Relational Factors in Prepubertal Girls with GD

Family dynamics can be major contributors in the modulation of gender dysphoria, in the timing of the search for a professional help and in supporting the child in her desperate pursuit of a comforting and comfortable sexual identity [2, 3]. Quality of relationships with age-mates and teachers may further contribute. Relational factors play a lifelong major dynamic role in the lifelong reshaping of human sexual identity. They maintain such a role in GD girls as well. A search of identity often dreamed of, caressed and cultivated as an endless promise of happiness, which unfortunately is not the case in the majority of GD individuals.

5.10 Professional Issues in Prepubertal Girls with GD

In prepubertal girls with GD, the professional support should have at least five major goals:

- To establish a deep, meaningful, warm and empathic relationship, based on non-judgemental trust, but at the same time with concrete perspectives on real-life issues and challenges [43, 44]
- To understand, share and evaluate the conscious and unconscious motivations to sex reassignment, when dreamed of and requested [2, 3, 45]
- To consider and discuss with the girl her expectations (real and delusional, mainly on the cosmetic and sexually functional outcome) [2, 3]
- Critically important, to consider a well-tailored and timely pharmacological intervention:
  - To delay puberty and optimise growth (with GnRH analogues and possibly GH) [3, 44, 46–48]
  - To integrate vitamins (D first of all) and oligoelements (iron first, as anaemia is a major contributor to depression) when indicated [10]
  - To ease the suffering with antidepressants, anxiolytics or antipsychotics, when specifically indicated and when "giving words to the emotional pain", with a psychotherapeutic support, is not enough to facilitate their search for a more satisfying identity [14]
- To support the family with a psychodynamic intervention [3]

5.11 Which Is the Optimal Age for the Surgical Decision: Legal Aspects

Which is the optimal age for a girl with GD to decide for her future? At 16 years of age, when many countries recognise the maturity to get a driving licence and vote, a comprehensively diagnosed and supported GD F to M girl is more likely to be able to make her final choice. The outcome can be optimal only if she has been maintained in a prepubertal status by a well-tailored pharmacologic treatment: this will allow her starting the appropriate hormonal treatment and then be operated of hysteroannessiectomy, or her remaining in her gender of birth, with a variable modulation of her final goals. When the diagnosis of severe gender dysphoria is accurate, all the examined and followed-up adolescents with a pharmacologically delayed puberty have far better psychological, emotional and general life adjustments, a smoother transition to hormone treatment and sex reassignment [2, 3, 36, 46–48].

5.12 Surgical Aspects of Delaying Puberty in Severe GD

To *keep puberty on hold* has a number of practical and very relevant consequences. By avoiding the breast growth, the girl will be spared the view, touch, proprioception and perception of an organ she detests and the surgical trauma of a mastectomy

later on in life. By preventing the appearance of periods, the girl will be spared the perception of a disturbing organ and function that could later on be removed with likely far less emotional (and physical?) costs. By preventing the development of a female body shape, often with cellulitis and stretch marks, a number of cosmetic interventions may be prevented later on in life. The overall result would be a significant reduction of the number of surgeries and potential side effects, an optimal timing of surgery and a far better general cosmetic outcome and psychosexual achievement. A choice and a process more likely to be closer to a well-assisted pursuit of the best personal sexual identity than leaving the "natural" pubertal process to go on with enormous distress and more physical consequences. Finally, what is the adolescents' perception of the opportunity and meaning of delaying puberty?

A very accurate qualitative study [48] focusing on adolescent's wording and reading of a pharmacologically retarded puberty, carried out in the Netherlands, finds out three major themes:

1. The difficulty of determining what is an appropriate lower age limit for starting puberty suppression. Most adolescents found it difficult to define an appropriate age limit and saw it as a dilemma.
2. The lack of data on the long-term effects of puberty suppression. Most adolescents stated that the lack of long-term data did not and would not stop them from wanting puberty suppression.
3. The role of the social context, for which there were two subthemes: (a) increased media attention, on television and on the internet; (b) an imposed stereotype. Some adolescents were positive about the role of the social context, but others raised doubts about it.

Compared to clinicians, adolescents were often more cautious in their treatment views. It is important to give voice to gender dysphoric adolescents when discussing the use of puberty suppression in GD. Otherwise, professionals might act based on assumptions about adolescents' opinions instead of their actual considerations, a golden recommendation that holds its powerful truth in the whole lifespan. Listening carefully, empathically and competently to patients' complaints, symptoms, expectations and dreams remains the golden way to a proper diagnosis and well-tailored treatment. A treatment that may as well differ from the current guidelines when the careful listening to the young patients' need suggests to have a different approach [48].

5.13 The Challenge of Caring About Gender Issues

Last, but not least, a higher awareness about the multiple barriers that prevent the proper approach to gender issues is needed to change both our clinical attitudes and the quality of diagnostic and therapeutic setting that can be offered.

To pursue this goal, it is first key to listen to *our* inner emotions when a child or an adolescent asks for help for her uneasiness about being a woman or for a frank

gender dysphoria, either explicitly or through her behaviour. Awareness about our countertransferral dynamics, as physicians and human being, is the first step for the proper emotional setting when a gender issue is complained of by our patients. Second, a basic knowledge on the key biological and psychosexual contributors of gender identity construction and development is essential. Third, a major effort should be devoted to reduce the rigid barriers that prevent an adequate diagnosis and care of gender issues. Current studies describe barriers spanning six themes: (1) few accessible paediatric and gynaecological providers are trained in gender-affirming health care, (2) lack of consistently applied protocols, (3) inconsistent use of chosen name/pronoun, (4) uncoordinated care and gatekeeping, (5) limited/delayed access to pubertal blockers and cross sex hormones and (6) insurance exclusions [49].

Recommendations include:

1. Mandatory training on gender-affirming health care and cultural humility for providers/staff
2. Development of protocols for the care of young transgender patients, as well as roadmaps for families
3. Asking and recording of chosen name/pronoun
4. Increased number of multidisciplinary gender clinics
5. Providing cross sex hormones at an age that permits peer-congruent development
6. Designating a navigator for transgender patients in clinics

The most accurate evaluation of physical, emotional, psychoaffective, relational and contextual difficulties should be taken into account to evaluate the best options. The goal is to offer the girl/patient (and her family) real expectations about the potential outcomes prior to start a long treatment path still rich of unappreciated uncertainties and risks.

Conclusion

Becoming a woman is a long path. The need of a solid sexual identity is even more compelling today, in a world where gender differences are becoming more elusive or denied. Gender dysphoria is certainly a complex and challenging issue first for the patient, a human being that bears a tremendous need and drive to find out and determine once and for all who she/he really is. It is challenging for the family, often unaware of the intense suffering of the child and of the intrinsically complex tasks of gender reassignment pathways. It is difficult for the health-care providers who are required to have and offer a well-balanced human touch and professional skill in an extremely complex area.

The early diagnosis of severe GD in girls and the serious consideration of the many aspects it involves should induce the health-care providers to pharmacologically *delay* the onset of puberty, whilst appropriately supporting the growth by healthy lifestyles, with optimal diet; vitamins (especially vitamin D); oligoelements such as calcium, magnesium and iron when indicated; regular daily exercises and respect of the sleep hours (at least 7, better 8); and avoidance of alcohol, smoke or drugs: all aspects frequently neglected, in this author's knowledge.

Antidepressants, anxiolytics and antipsychotics should as well be considered in selected cases.

The goal is to get quality years of personal and physical growth to optimise a more matured and "digested" choice whatever it would be: either remaining in the gender of birth with appropriate, more satisfying inner adjustments or move to a final male gender identity with real expectations and a higher probability of having them fulfilled.

References

1. Freud S. Das Ich und das Es. Wien: Internationaler Psychoanalytischer Verlag; 1923.
2. Baldaro Verde J, Graziottin A. L'enigma dell'identità – Il transessualismo. Torino: Edizioni Gruppo Abele; 1991.
3. Graziottin A. The hot questions of prepubertal gender dysphoria in girls. In: Trombetta C, Liguori G, Bertolotto M, editors. Management of gender dysphoria. A multidisciplinary approach to transsexualism. Milan: Springer; 2014. p. 217–27.
4. Ercan O, Kutlug S, Uysal O, et al. Gender identity and gender role in DSD patients raised as females: a preliminary outcome study. Front Endocrinol (Lausanne). 2013;4:86–93.
5. Sandberg DE, Gardner M, Cohen-Kettenis PT. Psychological aspects of the treatment of patients with disorders of sex development. Semin Reprod Med. 2012;30(5):443–52.
6. Martin CL, DiDonato MD, Clary L, et al. Preschool children with gender normative and gender non-normative peer preferences: psychosocial and environmental correlates. Arch Sex Behav. 2012;41(4):831–47.
7. Mattila AK, Fagerholm R, Santtila P, et al. Gender identity and gender role orientation in female assigned patients with disorders of sex development. J Urol. 2012;188:1930–7.
8. Freud S. Über die allgemeinste Erniedrigung des Liebeslebens (Beiträge zur Psychologie des Liebeslebens, Bd. 2). Jahrbuch für psychoanalytische und psychopathologische Forschungen. 1912;4(1):40–50.
9. Money JW, Ehrhardt AA. Man & woman, boy & girl: gender identity from conception to maturity. Northvale: Jason Aronson; 1996.
10. Graziottin A, Gambini D. Anatomy and physiology of genital organs. Women: what is relevant for the clinical practice. In: Vodusek D, Boller F, editors. Neurology of sexual and bladder disorders (Handbook of clinical neurology, Vol. 130, 3rd Series). Amsterdam: Elsevier BV; 2015. p. 39–60.
11. Harley VR, Clarkson MJ, Argentaro A. The molecular action and regulation of the testis-determining factors, SRY (sex-determining region on the Y chromosome) and SOX9 [SRY-related high-mobility group (HMG) box 9]. Endocr Rev. 2003;24(4):466–87.
12. Shields JP, Cohen R, Glassman JR, et al. Estimating population size and demographic characteristics of lesbian, gay, bisexual, and transgender youth in middle school. Adolesc Health. 2013;52(2):248–50.
13. Levine DA, Committee On Adolescence. Office-based care for lesbian, gay, bisexual, transgender, and questioning youth. Pediatrics. 2013;132(1):e297–313.
14. Leibowitz SF, Telingator C. Assessing gender identity concerns in children and adolescents: evaluation, treatments, and outcomes. Curr Psychiatry Rep. 2012;14(2):111–20.
15. Wallien MS, Cohen-Kettenis PT. Psychosexual outcome of gender-dysphoric children. J Am Acad Child Adolesc Psychiatry. 2008;47(12):1413–23.
16. Drummond KD, Bradley SJ, Peterson-Badali M, Zucker KJ. A follow-up study of girls with gender identity disorder. Dev Psychol. 2008;44(1):34–45.
17. Cardella L. Volevo i pantaloni. Milan: Mondadori; 1989.
18. Chiland C. Enfance et transsexualisme. Psychiatr Enfant. 1988;31(2):313–73.

19. Steensma TD, Kreukels BP, de Vries AL, Cohen-Kettenis PT. Gender identity development in adolescence. Horm Behav. 2013;64(2):288–97.
20. Ramachandran VS, McGeoch PD. Occurrence of phantom genitalia after gender reassignment surgery. Med Hypotheses. 2007;69(5):1001–3.
21. Fuss J, Biedermann SV, Stalla GK, Auer MK. On the quest for a biomechanism of transsexualism: is there a role for BDNF? J Psychiatr Res. 2013;47(12):2015–7.
22. Fontanari AM, Andreazza T, Costa ÂB, et al. Serum concentrations of brain-derived neurotrophic factor in patients with gender identity disorder. J Psychiatr Res. 2013;47(10): 1546–8.
23. Pankseep J. Affective neuroscience: the foundations of human and animal emotions. New York: Oxford University Press; 1998.
24. Forcier MM, Haddad E. Health care for gender variant or gender non-conforming children. R I Med J. 2013;96(4):17–21.
25. Pasquino AM, Pucarelli I, Segni M, et al. Adult height in girls with central precocious puberty treated with gonadotropin-releasing hormone analogues and growth hormone. J Clin Endocrinol Metab. 1999;84(2):449–52.
26. Wit JM, Balen HV, Kamp GA, Oostdijk W. Benefit of postponing normal puberty for improving final height. Eur J Endocrinol. 2004;151 Suppl 1:S41–5.
27. Scerpella TA, Dowthwaite JN, Gero NM, et al. Skeletal benefits of pre-menarcheal gymnastics are retained after activity cessation. Pediatr Exerc Sci. 2010;22(1):21–33.
28. Sale A, Berardi N, Maffei L. Environment and brain plasticity: towards an endogenous pharmacotherapy. Physiol Rev. 2014;94(1):189–234.
29. Villamor E, Marin C, Mora-Plazas M, Baylin A. Vitamin D deficiency and age at menarche: a prospective study. Am J Clin Nutr. 2011;94(4):1020–5.
30. Chew A, Harris SS. Does vitamin D affect timing of menarche? Nutr Rev. 2013;71(3): 189–93.
31. Esterle L, Nguyen M, Walrant-Debray O. Adverse interaction of low-calcium diet and low 25(OH)D levels on lumbar spine mineralization in late-pubertal girls. J Bone Miner Res. 2010;25(11):2392–8.
32. Cohen-Kettenis PT, Steensma TD, de Vries AL. Treatment of adolescents with gender dysphoria in the Netherlands. Child Adolesc Psychiatr Clin N Am. 2011;20(4):689–700.
33. de Vries AL, Cohen-Kettenis PT. Clinical management of gender dysphoria in children and adolescents: the Dutch approach. J Homosex. 2012;59(3):301–20.
34. Nakatsuka M. Puberty-delaying hormone therapy in adolescents with gender identity disorder] [Article in Japanese. Seishin Shinkeigaku Zasshi. 2013;115(3):316–22.
35. de Vries AL, Steensma TD, Doreleijers TA, Cohen-Kettenis PT. Puberty suppression in adolescents with gender identity disorder: a prospective follow-up study. J Sex Med. 2011; 8(8):2276–83.
36. Khatchadourian K, Amed S, Metzger DL. Clinical management of youth with gender dysphoria in Vancouver. J Pediatr. 2013. doi:10.1016/j.jpeds.2013.10.068 [Epub ahead of print].
37. Milrod C. How young is too young: ethical concerns in genital surgery of the transgender MTF adolescent. J Sex Med. 2014;11(2):338–46.
38. Kreukels BP, Cohen-Kettenis PT. Puberty suppression in gender identity disorder: the Amsterdam experience. Nat Rev Endocrinol. 2011;7(8):466–72.
39. Graziottin A. "The shorter, the better": a review of the evidence for a shorter contraception hormone-free interval. Eur J Contracept Reprod Health Care. 2015;20:1–13.
40. Iacovides S, Avidon I, Bentley A, Baker FC. Reduced quality of life when experiencing menstrual pain in women with primary dysmenorrhea. Acta Obstet Gynecol Scand. 2013. doi:10.1111/aogs.12287 [Epub ahead of print].
41. Craner J, Sigmon S, Martinson A, McGillicuddy M. Perceptions of health and somatic sensations in women reporting premenstrual syndrome and premenstrual dysphoric disorder. J Nerv Ment Dis. 2013;201(9):780–5.
42. Hewitt JK, Paul C, Kasiannan P, et al. Hormone treatment of gender identity disorder in a cohort of children and adolescents. Med J Aust. 2012;196(9):578–81.

43. Schwartz D. Listening to children imagining gender: observing the inflation of an idea. J Homosex. 2012;59(3):460–79.
44. Zucker KJ, Wood H, Singh D, Bradley SJ. A developmental, biopsychosocial model for the treatment of children with gender identity disorder. J Homosex. 2012;59(3):369–97.
45. Ehrensaft D. From gender identity disorder to gender identity creativity: true gender self child therapy. J Homosex. 2012;59(3):337–56.
46. Hembree WC. Guidelines for pubertal suspension and gender reassignment for transgender adolescents. Child Adolesc Psychiatr Clin N Am. 2011;20(4):725–32.
47. Vrouenraets LJ, Fredriks AM, Hannema SE, Cohen-Kettenis PT, de Vries MC. Perceptions of sex, gender, and puberty suppression: a qualitative analysis of transgender youth. Arch Sex Behav. 2016. [Epub ahead of print].
48. Olshan J, Eimicke T, Belfort E. Gender incongruity in children with and without disorders of sexual differentiation. Endocrinol Metab Clin North Am. 2016;45(2):463–82.
49. Shumer DE, Spack NP. Current management of gender identity disorder in childhood and adolescence: guidelines, barriers and areas of controversy. Curr Opin Endocrinol Diabetes Obes. 2013;20(1):69–73.

Endometriosis and Sexuality

6

Nadine Di Donato and Renato Seracchioli

6.1 Endometriosis and Related Symptoms

Endometriosis is a chronic and progressive condition which affects 5–10 % of women in reproductive age [20]. It is characterized by the presence of endometrial-like tissue outside the uterus which induces a local inflammatory response [23, 57]. It is an enigmatic condition; the etiology is uncertain and contested [20, 103]. Common symptoms are chronic pelvic pain, fatigue, congestive dysmenorrhea, heavy menstrual bleeding, and deep dyspareunia [23], and it is suggested that 47 % of infertile women have endometriosis [68]. There is no cure for endometriosis and so the management focuses on symptom relief [51]. This can involve a range of interventions including analgesics, hormonal therapy, both minimally invasive and radical surgery, and, where relevant, fertility treatment, with varying rates of success [31]. It is the most frequent cause of deep dyspareunia [39], and patients with the disease have a ninefold increase in risk of experiencing this symptom when compared with the general female population [5].

An international cross-sectional survey, involving 12 centers in 10 countries, investigating the quality of life of women with surgically diagnosed endometriosis ($n = 931$) reported that 28 % had originally presented with dyspareunia as one of their primary symptoms. Some 47 % suffered with painful sex at the time of the survey. Of these, 61 % reported pain often or always, and 54 % would either interrupt or avoid intercourse all together due to their pain [22]. Another large, cross-country study suggested that 24–25 % of women with diagnosed endometriosis experienced dyspareunia [7]. Denny and Mann [24] found that for 69 % of women, dyspareunia included pain in the hours and days after sex, and Ferrero et al. [38] suggested that women with endometriosis had frequently suffered dyspareunia

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_6

during their entire sex lives [24, 38]. Dyspareunia is generally described as deep (pain experienced inside the vaginal canal, at the level of the cervix, or in the pelvic uterine abdominal region) or superficial (pain in vulvar region and in the vaginal introitus) [11]. It is associated with a negative attitude toward sexuality, lack of self-confidence, anxiety, and the consequent avoidance of sexual relations [54, 66]. Women with dyspareunia have lower frequency of intercourse and lower levels of desire and experience fewer orgasms [63]. Ferrero et al., analyzing sexual life in three groups of women with dyspareunia, evidenced that the prevalence of deep dyspareunia was significantly higher among women with endometriosis than in controls. In particular, women with uterosacral ligament endometriosis had higher pain scores, a reduced number of intercourse per week, and a less satisfying orgasm and felt less relaxed and fulfilled after sex than the other groups [38]. A principal pathogenic mechanism in dyspareunia is altered awareness of pain recurrence due to previous experiences of coital pain. Therefore, the focus during sexual intercourse turns to sensation of (possible) pain instead of enjoyment [82]. The experience of pain and the loss of pleasure are recurrently recognized and become reinforced by repeated experiences. This process creates a cognitive scheme of negative expectations that disturbs sexuality [82]. Consequently, responses to dyspareunia include starting but then stopping intercourse, changing positions, enduring intercourse because of a desire for pregnancy, and enduring intercourse because of a reluctance to allow endometriosis to affect yet another aspect of life or because of a desire for closeness with a partner [24]. They cope with pain, either because their wish for a pregnancy outweighs their experience of pain or because of the importance of sexual intimacy.

Recently, a case–control study evaluated sexual function in patients affected by deep infiltrating endometriosis compared to healthy women, and they found an overall impairment of sexual function in all aspects, satisfaction, orgasm, desire, and pelvic problem interference with sexual intercourse [27]. The pelvic pain interference with sex was frequently reported by patients with deep infiltrating endometriosis (58 %), while it affected only 1 % of healthy women. Knowing that sexual desire is profoundly influenced by emotion and it is governed by a complex of inhibitory and excitatory influences, Di Donato et al. [27] reported lack of sexual desire in 45 % of women with deep infiltrating endometriosis, in spite of 14 % of healthy women. Orgasm and satisfaction, as aspects of sexual function, are also impaired in women with endometriosis. They have a multifactorial genesis depending on psychological, anatomic, and physiological factors. Due to their awareness of feeling pain, women with endometriosis may develop an unrelaxed attitude through the intercourse, and they hardly achieve orgasms compared to healthy women. Anticipation and fear of pain may provoke decreased arousal; lack of lubrication; pelvic floor hypertonus, resulting in mechanical trauma of the vestibular mucosa and urethral meatus; as well as resistance to penetration due to restriction of vaginal entry [94].

In endometriosis patients, the mechanism of pain during coital activity can be caused by traction of the inelastic parametria, by pressure on the endometriotic nodules, by immobilization of postero-uterine pelvic structures, and by a

neuropathic mechanism related to nerve injury or inflammatory stimuli. The presence of endometriosis is associated with an increased pain perception, and women with endometriosis generally experience major exacerbation of pain when even minor pressure is exerted on nodules [3]. Hyperalgesia is the occurrence of excruciating pain when a non-painful stimulus is applied and is a foremost characteristic of neuropathic pain, which corresponds to a painful sensation that is out of proportion with the intensity of nociceptor stimulation [2]. Neuropathic pain is usually related to nerve injury or inflammatory stimuli. Both of these conditions are found in endometriosis, as it has been demonstrated that sensory nerve fibers are frequently invaded by endometriotic stromal cells [3] and that several mediators such as histamine, tryptase, tumor necrosis factor α, prostaglandins, serotonin, interleukin-1, and nerve growth factor are abnormally synthesized and released by activated macrophages and mast cells as well as leukocytes within the endometriotic lesions, around sensory nerve fibers, and in peritoneal fluid [3]. Based on the chronic inflammatory condition primed by ectopic endometrium, a sort of vicious cycle may develop that determines nociceptors, sensitization, neurotropism and local neoneurogenesis, and activation of sensory nerve fibers, with resulting hyperalgesia [50]. Moreover, there is evidence that the presence of endometriosis is associated with increased pain perception [48], due to abnormal modulation of nociceptive input with an increase in the intensity of the neural signal ascending to the cerebral cortex [50]. Therefore, pain originating in endometriotic foci appears to lead to central sensitization and generalized hypersensitivity [50]. Organic problems may cause also secondary sexual dysfunction, as factors responsible for the origination of pain may be different from the ones that are active in the perpetuation of pain [50]. Thus deep dyspareunia caused by endometriosis can be viewed as an originally visceral type of pain secondary to chronic inflammation but with several superimposed components, including hyperalgesia, abnormal cortical perception, and psychological factors [50].

6.2 Endometriosis: Types and Sexual Function

Endometriosis inducing an inflammatory reaction and infiltration of anatomic structures causes pain symptoms such as dysmenorrhea, dyspareunia, chronic pelvic pain, dysuria, and dyschezia. It is classified in different anatomical types such as superficial, ovarian, and deep infiltrating endometriosis (DIE). It is estimated that the incidence of DIE is around 20 % of women with endometriosis [34]. There is a substantial impact of endometriosis on the quality of sex life: between 33.5 % [7] and 71 % [40] of women reported that endometriosis negatively affected their sex lives. Many studies indicated that women with endometriosis have less sexual and partnership satisfaction than healthy women [9, 35, 99]. Tripoli et al., through the Golombok Rust Inventory of Sexual Satisfaction (GRISS), underlined that 40 % of women with endometriosis were sexually unsatisfied and sexual satisfaction, sexual frequency, sexual aversion, and vaginismus were all significantly affected in women with endometriosis [88]. The complexity of this disease is not just a cause of painful symptoms but also of an overall impairment of different aspects of sexuality. It was

reported that quality of sex life was affected in all stages of endometriosis both in women with minimal and severe endometriosis [17]. Vercellini et al. reported that women with endometriosis experience more frequent and severe deep dyspareunia and worse sexual functioning compared with women without endometriosis. Dyspareunia was reported in 67 % of women with rectovaginal endometriosis, 53 % of women with peritoneal and/or ovarian endometriosis, and in 26 % of controls. No differences in sexual function were found between women with diverse endometriosis forms [96].

In particular, DIE defined as a form of endometriosis which penetrates for more than 5 mm under the peritoneal surface [61] is strongly associated with a significant reduction of quality of life [40, 41, 45] and sexual function [64, 82, 88]. It involves the uterosacral ligaments, vagina, intestinal wall, rectovaginal pouch, ureteral, and urinary bladder [16, 55]. Several studies correlated dyspareunia with the presence of DIE specifically of the uterosacral ligaments [33, 74]. Similarly, the presence of a vaginal nodule may affect sexual function through its direct stimulation during intercourse [82]. In general, nociceptive input from viscera, either the vagina or uterosacral ligaments affected by endometriotic lesions, leads to a central sensitization and provokes a generalized hypersensitivity in women with this disease [38]. Other studies demonstrated that complete excision of endometriosis, including vaginal lesion resection, seems to offer a significant increase in sexual satisfaction and decrease in sexual problems [69]. Moreover, advanced stage of disease may affect several aspects of women's life including psychological areas, and it seems that the number of deep infiltrating endometriotic nodules is directly proportional to the reduction of sexual function [69]. Three potential predictors (pelvic pain intensity, DIE status, and revised American Society for Reproductive Medicine stages) of female sexual dysfunction were identified in women with histologically confirmed endometriosis. The multivariable analysis showed that moderate-to-severe pelvic pain and revised American Society for Reproductive Medicine stage III or IV were associated with increased risk of having female sexual dysfunction [52]. Subjects in the rectovaginal endometriosis group had more than three times the risk of being sexually unsatisfied or feeling little or no sexual pleasure and about two times the risk of engaging in limited or no sexual activity and of reduced capacity to reach orgasm, compared with patients without endometriosis. However, subtle differences in frequency and severity of deep dyspareunia between women with rectovaginal lesions and those with peritoneal and/or ovarian lesions were found. There was a lack of correlation between severity of deep dyspareunia and level of sexual functioning in patients with endometriosis, independently from lesion type. Probably, the relationship between endometriosis and sexual dysfunction is much more complex than can be explained by anatomic distribution of lesions. A possible explanation is that organic dyspareunia is just one among a number of determinants of sexual functioning in women with endometriosis. In other words, because female sexuality is multifactorial and comprises psychologic processes, the effect of pain at intercourse could have been buffered or diluted by other factors, thus reducing part of its relative impact. Personality traits, coping capacity, degree of couple intimacy, partner emotional support, participation, solicitousness or hostility, marital

adjustment, and even quality of medical information and care may greatly influence the level of perception, interpretation, and acceptance of such a multifaceted symptom [65]. In conclusion, independently from lesion type, women with endometriosis reported more frequent and severe deep dyspareunia and performed worse in terms of sexual functioning compared with women without endometriosis. Owing to the intimate nature of the symptom, dyspareunia should be viewed in a broader clinical perspective, considering also the potential psychologic and interpersonal consequences. In other words, dyspareunia should not be simplistically considered to be equivalent to any other organic pain symptom, but should instead be investigated within the context of relational adjustment, psychologic well-being, global sexual functioning, and overall female health [96].

6.3 Endometriosis and Psychological Impact on Relationship

Pain during intercourse is particularly upsetting because it usually occurs whenever intercourse is attempted, whereas dysmenorrhea and dyschezia typically afflict women for a limited number of days each month. Some women learn to adapt their sexual position to accommodate the pain [25, 26], as the retrocervical area is most likely to be struck during deep vaginal penetration. Nonetheless, this frustrating complaint generally results in less satisfying orgasms, reduced relaxation and fulfillment after sex, limitations in the frequency of intercourses, and eventual overall impairment in quality of sex life [38].

Several studies have found that women feel that the symptoms and experience of endometriosis more generally have had a negative impact on their relationships and have in some cases contributed to relationship breakdown [19, 25, 32]. Fagervold et al. [32] reported that 40 out of 78 (51 %) women felt that endometriosis had a negative effect on their relationship, that 12 reported serious problems with relationships, and that 6 had experienced relationship breakdown as a result. However, in some cases partners were also identified as an important source of support [25]. In a rare though small-scale study of women and their male partners, Butt and Chesla [10] found that the experience of living with endometriosis disrupted day-to-day life and intimate relatedness for couples. They identified five relationship "coping" patterns: "together but alone," "battling together," "conjoined through disability," "totalized by care giving," and "engaged in mutual care" [10]. In two separately designed studies, 50 % and 56 % of women reported that endometriosis had affected their relationships, causing a split in 10 % and 8 % of cases, respectively [22]. Partners of women with endometriosis should be intensely involved in the management of the disease from the beginning. Proper counseling of the woman and her partner on the nature of the disease and its potential consequences for their sex life could hopefully diminish the sexual burden for these couples. Qualitative research found that women were reluctant to discuss dyspareunia with healthcare professionals and that women reported that healthcare professionals did not ask about this [25, 26]. However, Bernuit et al. [7] found that nearly half (47 %) of women with diagnosed

endometriosis who reported that pain affected their quality of life had visited their doctor due to dyspareunia [7]. Interventions to enable couples to address the impact of endometriosis on sexual relations are limited, and those that are available are reported by couples to be unhelpful [10]. Recent research has identified problematic care experiences in endometriosis centers and identified the need for a more patient-centered approach to care [21]. Prospective and retrospective longitudinal cohort studies would enable to more adequately capture the changing and dynamic nature of the impact of endometriosis on people's lives. This is particularly relevant given the impact of this condition on life-course markers such as childbearing, relationships, and career progression. It is a necessary mixed method to explore the impact of endometriosis to draw on the combined strengths of qualitative and quantitative research [73, 83]. The weight of evidence from quantitative studies suggests that rates of depression, anxiety, and emotional distress are higher for women with endometriosis than control groups or the general population. While no firm conclusions can be drawn about the role of pain when comparing women with endometriosis pain and women with pain-free endometriosis, the overall findings comparing women with chronic pelvic pain related to endometriosis and women with chronic pelvic pain from other or unknown causes suggest that it is the experience of pain that is associated with mental health difficulties and emotional distress. Qualitative studies highlight the emotional distress that characterizes many women's lives, including feelings of isolation, guilt, worry, worthlessness, and hopelessness and feeling unable to cope. The mechanisms by which this occurs are unclear however, as pain, emotional distress, fatigue, and dissatisfaction with healthcare and available treatments are all interwoven within the complex experience of endometriosis. Despite calls for care and support to better focus on psychological and emotional experiences and impact, depression, anxiety, and emotional distress appear to prevail, and there are few reports of systematic interventions to address these issues.

6.4 Clinical and Demographic Factors and Sexual Function

Thirty-two percent of patients affected by endometriosis complain sexual dysfunction with pain during intercourse, reduced number of sexual intercourses per month, and feelings of guilt toward the partner. The experience of pain creates a cognitive scheme of negative expectations that disturbs sexuality [42]. The evaluation of sexual function is complex and influenced by many factors such as pain during intercourse, chronic pelvic pain, partner relationship, history of previous surgery, body mass index (BMI), and education. A prospective cohort study evaluated the impact of sociodemographic and anamnestic characteristics of women with deep infiltrating endometriosis on the quality of life and sexual function. A poor quality of life and sexual function were found which were worsened by the presence of history of previous surgery and low BMI. Probably the adipose tissue, by producing estrogen, may positively influence the sexual life through an increase of libido and sexual desire [28]. In a previous study, it was reported that a high BMI was associated with a longer delay in the time of diagnosis compared to patients with low BMI [70].

This may, in part, be explained by the fact that overweight women had less sexual dysfunction than normal-weight women. In a study of 187 women with chronic pelvic pain, low educational level and a low socioeconomic status were found to be risk factors for endometriosis symptoms. It was assumed that patients may feel the disease and its debilitating symptoms, through the mechanism defined "pain rationalization" [84].

Another important aspect is represented by infertility together with depression, is highly prevalent in women with endometriosis, and is also associated with the impairment of sexual function [58, 85, 87]. It can cause psychological changes, such as anxiety [59, 79], that might negatively affect physical and emotional health, quality of life, and sexual function. Limited research suggests that the impact, or potential impact, of endometriosis on fertility causes worry, depression, and feelings of inadequacy. Previous studies have demonstrated that infertility is associated with difficulties in interpersonal relations and social interactions, impaired dyadic relations, reduced self-esteem [102], feelings of shame [44], social isolation [8], and compromised mental health [14]. Emotional problems increase with the duration of infertility and with advancing age in women as their childless years increase [86]. It should be noted that reduced sexual desire and sexual intercourse frequency may occur due to the loss of sexual spontaneity [72] that is secondary to certain strategies used to increase the chances of pregnancy [75]. Depressive symptoms are usually associated with changes in sexual function such as reduced sexual satisfaction [71], dysfunction of excitement, and less pleasure during sexual relations [43]. Contrary, a study by [29] reported that infertile females had better partner relationships than fertile females, and [78] demonstrated that several unsuccessful attempts at treatment of infertility were associated with good dyadic consensus and cohesion. It is possible that partners become more supportive by sharing the stressful experiences associated with treatment of infertility, and this may improve the marital relationship and increase dyadic cohesion. Considering that almost half of infertile women have endometriosis, further researches into the social and psychological experience of infertility are necessary among women with endometriosis.

6.5 Medical Management of Endometriosis and Sexuality

Medical treatments are effective in decreasing pain with intercourse [93]. Several pharmacological compounds have been used successfully in women with deep dyspareunia associated with infiltrating endometriosis, including depot intramuscular GnRH agonists, vaginal danazol, oral aromatase inhibitors, progestins directly delivered into the uterine cavity, and oral, intravaginal, transdermal estrogen–progestogen combinations as well as oral progestogens alone [90, 91, 97]. In a randomized controlled trial, a GnRH agonist was more effective than an estrogen–progestin on pain at intercourse [98], and the combination of low-dose norethisterone acetate with an aromatase inhibitor (letrozole) reduced deep dyspareunia to a larger extent than low-dose norethisterone acetate alone in women with rectovaginal endometriosis ([37];24:3033–3041). Moreover, Vercellini et al. compared in a prospective

study [92] the effect of laparoscopic treatment versus low-dose norethisterone acetate for the management of severe deep dyspareunia associated with persistent or recurrent endometriosis after unsuccessful first-line conservative surgery. Authors reported a rapid improvement of sexual functioning and psychological status after surgery but worsened with time, whereas the effect during progestin use increased more gradually and progressively without overall significant differences between the groups at 12-month follow-up. Both surgery and medical treatment with progestins resulted valuable options for improving the impact of dyspareunia on sexual function and quality of life [92]. However, hormonal treatments fail in approximately 1 woman out of 3, are associated with a high recurrence rate after discontinuation [77], cannot be used in women seeking conception as they inhibit ovulation, and may interfere with sexual desire and arousal [77].

6.6 Surgical Management of Endometriosis and Sexuality

Dyspareunia, especially deep dyspareunia, is present in 60–80% of patients who undergo surgery for endometriosis and 50–90% of those receiving conservative treatment for the condition [18]. Several studies have demonstrated that surgical excision of deep endometriotic lesions markedly reduces the severity of dyspareunia and improves the quality of sexual function in these patients [4, 49, 95]. Excision of endometriotic foci has been reported to be effective in reducing pain at intercourse and in ameliorating quality of sex life [4], but surgery for deep lesions requires a high level of technical competence, is associated with potentially severe morbidity, and generally results in only partial or temporary efficacy [90, 91, 97]. It was suggested that women treated for deep infiltrating endometriosis, after surgery, managed to relax and feel more comfortable during intercourse because of the reduction of pain symptoms and the awareness of the removal of endometriotic lesions. Moreover, the complete excision of all endometriotic lesions and the restoration of normal pelvic anatomy might reduce interference with sex by pelvic problems [64]. A positive correlation seems to exist between the extent of endometriosis resection and the degree of postoperative symptom improvement [15]. Garry et al. observed that the excision of endometriotic lesions significantly improved quality of sexual function at 4-month follow-up [47]. Ferrero et al. observed a significant improvement in sexual life at 6-month and 12-month follow-up after surgical excision of DIE. Patients had increased variety in sexual life, frequency of intercourse, more satisfying orgasms with sex, and relaxed easier during sex and were more fulfilled after sex [37].

However, the effect of surgery is generally partial or temporary. Vercellini et al. [92] in a prospective cohort study demonstrated that surgery and low-dose progestin therapy are both effective in improving sexual functioning, psychological well-being, and health-related quality of life in women with endometriosis-associated deep dyspareunia, although with a different chronological pattern. Excision of lesions is followed by a rapid and substantial benefit, with a greater immediate effect with respect to progestin treatment. However, the gradual recurrence of pain at intercourse after 6 months since surgery, combined with progressive amelioration of clinical conditions

in women using low-dose progestin therapy, leads to a final practical equipoise in the considered functional outcomes at 1-year follow-up. Moreover, the mean total Female Sexual Functioning Index score never exceeded the "normality" cutoff limit of 26.55 at any time point [101]. Therefore, the sexual dysfunction observed at baseline was completely corrected neither by surgery nor by medical treatment. In particular, performance in the surgery group improved substantially in the short term and then gradually deteriorated. However, at 12 months of evaluation, desire, arousal, and lubrication scores were still significantly higher than in the low-dose progestin therapy group, resulting in a tendency toward better total FSFI score after surgery. Probably medical therapy induces a decreased desire, arousal, and lubrication, but once intercourse initiates, orgasm capacity and overall satisfaction are not worse than in the surgical group. Conversely, 1 year after surgery, women maintained an unaffected sexual predisposition, but experienced slightly more pain during intercourse. As Ferrero et al. [36] reported, the best results in reduction of pain at intercourse with an improvement in quality of sex life can be obtained with surgery, followed by postoperative medical treatment [36]; thus there is an increasing consensus among experts in endometriosis that hormonal therapies should be systematically proposed to women who do not intend to conceive. Postoperative medical treatment reduces the risk of recurrence inducing the atrophy of the eutopic and ectopic endometrium. Combination of surgery, followed by medical treatment of DIE, was also proven to improve symptoms and quality of life [1, 46, 76].

Women after surgery for bowel endometriosis (with and without bowel resection and reanastomosis) had improved levels of depression, relationship satisfaction, and sexual functioning [89]. Patients with bowel resection had significantly better outcomes in the proportion of women with depressive symptoms as well as lower levels of sexual problems with orgasm and pain during intercourse than patients without. However, it is possible that women with bowel resection experience a greater difference in depression, relationship satisfaction, and sexual functioning after surgery because of the more severe situation before surgery. The extensive surgery of bowel resection gives patients experience of more intensive follow-up due to the nature of their surgery, and this may turn in reduced level of depression [67].

However, laparoscopic management of DIE is a complex surgical procedure, which may be complicated by neurogenic impairment of urinary, intestinal, and sexual function [30, 62, 81], especially in cases where nerve-sparing surgery is not feasible. Complete DIE lesion resection can cause postoperative dysfunction of the genitals, bladder, and bowel, although pain symptoms are greatly alleviated [6, 30, 60]. During dissection of the rectum and uterosacral ligaments, autonomic nerves supplying the rectum and urinary bladder may be damaged leading to urinary, defecatory, and sexual dysfunction. The problem is well recognized, and a nerve-sparing bowel resection technique for endometriosis has been introduced in order to avoid autonomic dysfunctions. Recently, studies have shown that nerve-sparing surgery for DIE can reduce bowel and bladder dysfunction without decreasing surgical efficacy [12, 13, 56, 100]. During this surgical procedure, the branches of the inferior hypogastric nerves that supply the bladder, colon, cervix, and vagina are identified and preserved, whereas DIE lesions are excised [12, 13, 56, 100]. In a recent study,

the total FSFI score and the sub-score for each of the six domains in patients with nerve-sparing surgery or conventional surgery for DIE were below the clinical cut-off scores before surgery [101], and these scores significantly increased after surgery. These findings suggest that sexual dysfunction is reduced by surgery but not by the nerve-sparing technique. The tendency to increased pain symptoms and sexual dysfunction over time after operation may be caused by new DIE lesions or remaining DIE lesions which are caused by incomplete excision during the surgical procedure [92].

Moreover, besides the risk of autonomic nerve injuries, aggressive surgery such as bowel resections and resection of large parts of the uterosacral ligaments with consequent scarring of the pouch of Douglas can adversely contribute to an impaired sex life [80]. Women should therefore always be counseled appropriately prior to surgery so that they are aware of these potential risks. Furthermore, advice should be heeded from the recently published consensus on the current management of endometriosis that those women with a higher stage of disease and/or intractable clinical problems should receive care from a center of expertise with appropriate surgical expertise in the laparoscopic excision of endometriosis [53].

In conclusion, considering the multidimensional impact of endometriosis, the treatment approach should include emotional and social support, stress reduction, psychosexual treatment, and a focus on quality of life issues, sex and relationships, pain management, and career counseling [10, 24]. Psychological and psychosexual counseling should be offered when dealing with endometriosis patients and should be an essential component of endometriosis treatment. It is very important that gynecologists involved in the management of endometriosis offer patients a profound conversation about their sexuality, because these professionals in most cases are the first reference persons for suffering women. Due to the fact that sexuality, especially impaired sexuality, is often a shameful topic, it is the task of the gynecologists to address this delicate issue in a pleasant way. The World Health Organization has included sexual health among human rights [104], and the report of a technical consultation on sexual health held in Genova (2002) [105] states that "sexual rights embrace human rights, including the right of all persons to pursue a satisfying, safe, and pleasurable sexual life." Dyspareunia should not be simplistically considered equivalent to any other organic pain symptom but should instead be considered within the context of global sexual functioning, relational adjustment, psychological well-being, and overall female health.

References

1. Abbott JA, Hawe J, Clayton RD, et al. The effects and effectiveness of laparoscopic excision of endometriosis: a prospective study with 2–5 year follow-up. Hum Reprod. 2003;18:1922–7.
2. Anaf V, Chapron C, El Nakadi I, et al. Pain, mast cells, and nerves in peritoneal, ovarian, and deep infiltrating endometriosis. Fertil Steril. 2006;86:1336–43.
3. Anaf V, Simon P, El Nakadi I. Hyperalgesia, nerve infiltration and nerve growth factor expression in deep adenomyotic nodules, peritoneal and ovarian endometriosis. Hum Reprod. 2002;17:1895–900.

4. Anaf V, Simon P, El Nakadi I, et al. Impact of surgical resection of rectovaginal pouch of douglas endometriotic nodules on pelvic pain and some elements of patients' sex life. J Am Assoc Gynecol Laparosc. 2001;8:55–60.
5. Ballard KD, Seaman HE, De Vries CS, et al. Can symptomatology help in the diagnosis of endometriosis? Findings from a national case–control study-Part 1. BJOG. 2008;115:1382–91.
6. Ballester M, Santulli P, Bazot M, Coutant C, Rouzier R, Daraï E. Preoperative evaluation of posterior deep-infiltrating endometriosis demonstrates a relationship with urinary dysfunction and parametrial involvement. J Minim Invasive Gynecol. 2011;18(1):36–42.
7. Bernuit D, Ebert AD, Halis G, et al. Female perspectives on endometriosis: findings from the uterine bleeding and pain women's research study. J Endometriosis. 2011;2:73–85.
8. Bhatti LI, Fikree FF, Khan A. The quest of infertile women in squatter settlements of Karachi, Pakistan: a qualitative study. Soc Sci Med. 1999;49:637–49.
9. Biberoglu KO, Behrman SJ. Dosage aspects of danazol therapy in endometriosis: short-term and long-term effectiveness. Am J Obstet Gynecol. 1981;15:645–54.
10. Butt F, Chesla C. Relational patterns of couples living with chronic pelvic pain from endometriosis. Qual Health Res. 2007;5:571–85.
11. Carvalho J, Vieira AL, Nobre P. Latent structures of female sexual functioning. Arch Sex Behav. 2012;41:907–17.
12. Ceccaroni M, Clarizia R, Alboni C, et al. Laparoscopic nerve-sparing transperitoneal approach for endometriosis infiltrating the pelvic wall and somatic nerves: anatomical considerations and surgical technique. Surg Radiol Anat. 2010;32:601–4.
13. Ceccaroni M, Pontrelli G, Scioscia M, et al. Nerve-sparing laparoscopic radical excision of deep endometriosis with rectal and parametrial resection. J Minim Invasive Gynecol. 2010;17:14–5.
14. Chachamovich JR, Chachamovich E, Ezer H, Fleck MP, et al. Investigating quality of life and health-related quality of life in infertility: a systematic review. J Psychosom Obstet Gynaecol. 2010;31:101–10.
15. Chapron C, Chopin N, Borghese B. Surgical management of deeply infiltrating endometriosis: an update. Ann N Y Acad Sci. 2004;1034:326–37.
16. Chapron C, Dubuisson JB. Laparoscopic treatment of deep endometriosis located on the uterosacral ligaments. Hum Reprod. 1996;11:868–87.
17. Chene G, Jaffeux P, Lasnier C, et al. Quality of life of women with endometriosis: comparison between epiphenomenon and severe disease. J Endometriosis. 2012;2:77–84.
18. Chopin N, Vieira M, Borghese B, et al. Operative management of deeply infiltrating endometriosis: Results on pelvic pain symptoms according to a surgical classification. J Minim Invasive Gynecol. 2005;12:106–12.
19. Cox H, Henderson L, Wood R, et al. Learning to take charge: women's experiences of living with endometriosis. Complement Ther Nurs Midwifery. 2003;2:62–8.
20. Damewood M, Kresch AJ, Metzger D, et al. Current approaches to endometriosis. Patient Care. 1997;31:34–43.
21. Dancet EAF, Apers S, Kluivers KB, et al. The ENDOCARE questionnaire guides European endometriosis clinics to improve the patient-centeredness of their care. Hum Reprod. 2012;11:3168–78.
22. De Graaff AA, D'Hooghe TM, Dunselman GA, Simoens S, WERF EndoCost Consortium, et al. The significant effect of endometriosis on physical, mental and social wellbeing: results from an international cross-sectional survey. Hum Reprod. 2013;28:2677–85.
23. De Nardi P, Ferrari S. Deep pelvic endometriosis: a multidisciplinary approach. Milan: Springer; 2011.
24. Denny E, Mann CH. Endometriosis-associated dyspareunia: the impact on women's lives. J Fam Plan Reprod H. 2007;3:189–93.
25. Denny E. Women's experience of endometriosis. J Adv Nurs. 2004;6:641–8.
26. Denny E. 'You are one of the unlucky ones': delay in the diagnosis of endometriosis. Divers Health Social Care. 2004;1:39–44.
27. Di Donato N, Montanari G, Benfenati A, et al. Do women with endometriosis have to worry about sex? Eur J Obstet Gynecol Reprod Biol. 2014;179:69–74.

28. Di Donato N, Montanari G, Benfenati A. The impact of socio-economic and anamnestic characteristics on quality of life and sexuality in women with endometriosis. J Endometriosis. 2013; vol 5 issue 4: pp 127–69.
29. Drosdzol A, Skrzypulec V. Evaluation of marital and sexual interactions of Polish infertile couples. J Sex Med. 2009;6:3335–46.
30. Dubernard G, Rouzier R, David-Montefiore E, et al. Urinary complications after surgery for posterior deep infiltrating endometriosis are related to the extent of dissection and to utero-sacral ligaments resection. J Minim Invasive Gynecol. 2008;15:235–40.
31. Dunselman GA, Vermeulen N, Becker C, Calhaz-Jorge C, D'Hooghe T, De Bie B, Heikinheimo O, Horne AW, Kiesel L, Nap A, Prentice A, Saridogan E, Soriano D, Nelen W; European Society of Human Reproduction and Embryology.(ESHRE) guideline: management of women with endometriosis. Hum Reprod. 2014;29(3):400–12.
32. Fagervold B, Jenssen M, Hummelshoj L, et al. Life after a diagnosis with endometriosis: a 15 years follow-up study. Acta Obstet Gyn Scan. 2009;8:914–9.
33. Fauconnier A, Chapron C, Dubuisson JB. Relation between pain symptoms and the anatomic location of deep infiltrating endometriosis. Fertil Steril. 2002;78:719–26.
34. Fauconnier A, Chapron C. Endometriosis and pelvic pain: epidemiological evidence of the relationship and implications. Hum Reprod Update. 2005;11:595–606.
35. Fedor-Freybergh P. The influence of estrogens on the wellbeing and mental performance in climacteric and postmenopausal women. Acta Obstet Gynecol Scand. 1977;64:1–91.
36. Ferrero S, Abbamonte LH, Parisi M, et al. Dyspareunia and quality of sex life after laparoscopic excision of endometriosis and postoperative administration of triptorelin. Fertil Steril. 2007;87:227–9.
37. Ferrero S, Camerni G, Seracchioli R, et al. Letrozole combined with norethisterone acetate compared with norethisterone acetate alone in the treatment of pain symptoms caused by endometriosis. Hum Reprod. 2009;24:3033–41.
38. Ferrero S, Esposito F, Abbamonte LH, et al. Quality of sex life in women with endometriosis and deep dyspareunia. Fertil Steril. 2005;83:573–9.
39. Ferrero S, Ragni N, Remorgida V. Deep dyspareunia: causes, treatments, and results. Curr Opin Obstet Gynecol. 2008;20:394–9.
40. Fourquet J, Báez L, Figueroa M, et al. Quantification of the impact of endometriosis symptoms on health-related quality of life and work productivity. Fertil Steril. 2011;96:107–12.
41. Fourquet J, Gao X, Zavala D, et al. Patients' report on how endometriosis affects health, work, and daily life. Fertil Steril. 2010;93:2424–8.
42. Fritzer N, Haas D, Oppelt P, et al. More than just bad sex: sexual dysfunction and distress in patients with endometriosis. Eur J Obstet Gynecol Reprod Biol. 2013;169:392–6.
43. Frohlich P, Meston C. Sexual functioning and self-reported depressive symptoms among college women. J Sex Res. 2002;39:321–5.
44. Galhardo A, Pinto-Gouveia J, Cunha M, et al. The impact of shame and self-judgment on psychopathology in infertile patients. Hum Reprod. 2011;26:2408–14.
45. Gao X, Yeh YC, Outley J, et al. Health-related quality of life burden of women with endometriosis: a literature review. Curr Med Res Opin. 2006;22:1787–97.
46. Garry R, Clayton R, Hawe J. The effect of endometriosis and its radical laparoscopic excision on quality of life indicators. BJOG. 2000;107:44–54.
47. Garry R. Laparoscopic excision of endometriosis: The treatment of choice? Br J Obstet Gynaecol. 1997;104:513–5.
48. Giamberardino MA, De Laurentis S, Affaitati G, et al. Modulation of pain and hyperalgesia from the urinary tract by algogenic conditions of the reproductive organs in women. Neurosci Lett. 2001;304:61–4.
49. Hidaka T, Nakashima A, Hashimoto Y, et al. Effects of laparoscopic radical surgery for deep endometriosis on endometriosis-related pelvic pain. Minim Invasive Ther Allied Technol. 2012;21:355–61.
50. Howard FM. Endometriosis and mechanisms of pelvic pain. J Minim Invasive Gynecol. 2009;16:540–50.
51. Huntington A, Gilmour J. A life shaped by pain: women and endometriosis. J Clin Nurs. 2005;9:1124–32.

52. Jia SZ, Leng JH, Sun PR. Prevalence and associated factors of female sexual dysfunction in women with endometriosis. Obstet Gynecol. 2013;121:601–6.
53. Johnson NP, Hummelshoj L, for the World Endometriosis Society Montpellier Consortium. Consensus on current management of endometriosis. Hum Reprod. 2013; 28:1552–68.
54. Jones G, Jenkinson C, Kennedy S. The impact of endometriosis upon quality of life: a qualitative analysis. J Psychosom Obstet Gynaecol. 2004;25:123–33.
55. Kamina P. Anatomie ynécologique et obstétricale, vol. 4. Paris: Maloine SA; 1984. p. 298.
56. Kavallaris A, Banz C, Chalvatzas N, et al. Laparoscopic nerve-sparing surgery of deep infiltrating endometriosis: description of the technique and patients' outcome. Arch Gynecol Obstet. 2011;284:131–5.
57. Kennedy S, Bergqvist A, Chapron C, et al. ESHRE guideline for the diagnosis and treatment of endometriosis. Hum Reprod. 2005;20:2698–704.
58. Keskin U, Coksuer H, Gungor S, et al. Differences in prevalence of sexual dysfunction between primary and secondary infertile women. Fertil Steril. 2011;96:1213–7.
59. Klemetti R, Raitanen J, Sihvo S, et al. Infertility, mental disorders and well-being--a nationwide survey. Acta Obstet Gynecol Scand. 2010;89:677–82.
60. Kondo W, Bourdel N, Tamburro S, et al. Complications after surgery for deeply infiltrating pelvic endometriosis. BJOG. 2011;118:292–8.
61. Koninckx PR, Meuleman C, Demeyere S, et al. Suggestive evidence that pelvic endometriosis is a progressive disease, whereas deeply infiltrating endometriosis is associated with pelvic pain. Fertil Steril. 1991;55:759–65.
62. Landi S, Mereu L, Indraccolo U, et al. Laparoscopic excision of endometriosis may require unilateral parametrectomy. JSLS. 2009;13:496–503.
63. Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. JAMA. 1999;281:537–44.
64. Mabrouk M, Montanari G, Di Donato N, et al. What is the impact on sexual function of laparoscopic treatment and subsequent combined oral contraceptive therapy in women with deep infiltrating endometriosis? J Sex Med. 2012;9:770–8.
65. Meana M, Binik I, Khalife S, et al. Affect and marital adjustment in women's rating of dyspareunic pain. Can J Psychiatry. 1998;43:381–5.
66. Meana M, Binik YM, Khalife S, et al. Biopsychosocial profile of women with dyspareunia. Obstet Gynecol. 1997;90:583–9.
67. Meuleman C, Tomassetti C, Wolthuis A, et al. Clinical outcome after radical excision of moderate-severe endometriosis with or without bowel resection and reanastomosis: a prospective cohort study. Ann Surg. 2013;259:522–31.
68. Meuleman C, Vandenabeele B, Fieuws S, et al. High prevalence of endometriosis in infertile women with normal ovulation and normospermic partners. Fertil Steril. 2009;1: 68–74.
69. Montanari G, Di Donato N, Benfenati A, et al. Women with deep infiltrating endometriosis: sexual satisfaction, desire, orgasm, and pelvic problem interference with sex. J Sex Med. 2013;10:1559–66.
70. Nnoaham KE, Hummelshoj L, Webster P, World Endometriosis Research Foundation Global Study of Women's Health consortium, et al. Impact of endometriosis on quality of life and work productivity: a multicenter study across ten countries. Fertil Steril. 2011;96:366–73.
71. Pakpour AH, Yekaninejad MS, Zeidi IM, et al. Prevalence and risk factors of the female sexual dysfunction in a sample of infertile Iranian women. Arch Gynecol Obstet. 2012;286:1589–96.
72. Pinto-Gouveia J, Galhardo A, Cunha M, et al. Protective emotional regulation processes towards adjustment in infertile patients. Hum Fertil (Camb). 2012;15:27–34.
73. Plano Clark VL. The adoption and practice of mixed methods: US trends in federally funded health-related research. Qual Inq. 2010;6:428–40.
74. Porpora MG, Koninckx PR, Piazze J, et al. Correlation between endometriosis and pelvic pain. J Am Assoc Gynecol Laparosc. 1999;6:429–34.

75. Reder F, Fernandez A, Ohl J. Does sexuality still have a place for couples treated with assisted reproductive techniques? J Gynecol Obstet Biol Reprod (Paris). 2009;38:377–88.
76. Redwine DB, Wright JT. Laparoscopic treatment of complete obliteration of the cul-de-sac associated with endometriosis: Long-term follow-up of en bloc resection. Fertil Steril. 2001;76:358–65.
77. Remorgida V, Abbamonte LH, Ragni N, et al. Letrozole and desogestrel-only contraceptive pill for the treatment of stage IV endometriosis. Aust N Z J Obstet Gynaecol. 2007; 47:222–5.
78. Repokari L, Punamaki RL, Unkila-Kallio L, et al. Infertility treatment and marital relationships: a 1-year prospective study among successfully treated ART couples and their controls. Hum Reprod. 2007;22:1481–91.
79. Revonta M, Raitanen J, Sihvo S, et al. Health and life style among infertile men and women. Sex Reprod Healthc. 2010;1:91–8.
80. Roman H, Loisel C, Resch B, et al. Delayed functional outcomes associated with surgical management of deep rectovaginal endometriosis with rectal involvement: giving patients an informed choice. Hum Reprod. 2010;25:890–9.
81. Roman H, Vassilieff M, Gourcerol G, et al. Surgical management of deep infiltrating endometriosis of the rectum: Pleading for a symptom-guided approach. Hum Reprod. 2011;26:274–81.
82. Setälä M, Härkki P, Matomäki J, et al. Sexual functioning, quality of life and pelvic pain 12 months after endometriosis surgery including vaginal resection. Acta Obstet Gynecol Scand. 2012;91:692–8.
83. Tashakkori A, Teddlie C. SAGE handbook of mixed methods in social & behavioral research. 2nd ed. Thousand Oaks: Sage Publications; 2010.
84. Tchernof A, Després JP. Pathophysiology of human visceral obesity: an update. Physiol Rev. 2013;93:359–404.
85. Ter Kuile MM, Brauer M, Laan E. The Female Sexual Function Index (FSFI) and the Female Sexual Distress Scale (FSDS): psychometric properties within a Dutch population. J Sex Marital Ther. 2006;32:289–304.
86. Ter Kuile MM, Van Lankveld JJ, Kalkhoven P, et al. The Golombok Rust Inventory of Sexual Satisfaction (GRISS): psychometric properties within a Dutch population. J Sex Marital Ther. 1999;25:59–71.
87. Ter Kuile MM, Weijenborg PT, Spinhoven P. Sexual functioning in women with chronic pelvic pain: the role of anxiety and depression. J Sex Med. 2007;7:1901–10.
88. Tripoli TM, Stato H, Sartori MG, et al. Evaluation of quality of life and sexual satisfaction in women suffering from chronic pelvic pain with or without endometriosis. J Sex Med. 2011;8:497–503.
89. Van den Broeck U, Meuleman C, Tomassetti C, et al. Effect of laparoscopic surgery for moderate and severe endometriosis on depression, relationship satisfaction and sexual functioning: comparison of patients with and without bowel resection. Hum Reprod. 2013;28:2389–97.
90. Vercellini P, Carmignani L, Rubino T, et al. Surgery for deep endometriosis: a pathogenesis-oriented approach. Gynecol Obstet Invest. 2009;68:88–103.
91. Vercellini P, Crosignani PG, Somigliana E, et al. Medical treatment for rectovaginal endometriosis: what is the evidence? Hum Reprod. 2009;24:2504–14.
92. Vercellini P, Frattaruolo MP, Somigliana E, et al. Surgical versus low-dose progestin treatment for endometriosis-associated severe deep dyspareunia II: effect on sexual functioning, psychological status and health-related quality of life. Hum Reprod. 2013;28:1221–30.
93. Vercellini P, Frontino G, Pietropaolo G, et al. Deep endometriosis: definition, pathogenesis, and clinical management. J Am Assoc Gynecol Laparosc. 2004;11:153–61.
94. Vercellini P, Meana M, Hummelshoj L, et al. Priorities for endometriosis research: a proposed focus on deep dyspareunia. Reprod Sci. 2011;18:114–8.
95. Vercellini P, Pietropaolo G, De Giorgi O, et al. Reproductive performance in infertile women with rectovaginal endometriosis: is surgery worthwhile? Am J Obstet Gynecol. 2006; 195:1303–10.

96. Vercellini P, Somigliana E, Buggio L, et al. “I can’t get no satisfaction”: deep dyspareunia and sexual functioning in women with rectovaginal endometriosis. Fertil Steril. 2012;98:1503–11.
97. Vercellini P, Somigliana E, Viganò P, et al. Endometriosis: current therapies and new pharmacological developments. Drugs. 2009;69:649–75.
98. Vercellini P, Trespidi L, Colombo A, et al. A gonadotropin releasing hormone agonist versus a low-dose oral contraceptives for pelvic pain associated with endometriosis. Fertil Steril. 1993;60:75–9.
99. Vercellini P, Trespidi L, de Giorgi O, et al. Endometriosis and pelvic pain: relation to disease stage and localization. Fertil Steril. 1996;65:299–304.
100. Volpi E, Ferrero A, Sismondi P. Laparoscopic identification of pelvic nerves in patients with deep infiltrating endometriosis. Surg Endosc. 2004;18:1109–12.
101. Wiegel M, Meston C, Rosen R. The female Sexual Function Index (FSFI): cross-validation and development of clinical cut-off scores. J Sex Marital Ther. 2005;31:1–20.
102. Wischmann T, Korge K, Scherg H. A 10-year follow-up study of psychosocial factors affecting couples after infertility treatment. Hum Reprod. 2012;27:3226–32.
103. World Endometriosis Society and the World Endometriosis Research Foundation. Facts about endometriosis. 2012.
104. World Health Organization. Gender and human rights. Definition Sexual health. 2010. Retrieved from http://www.who.int/reproductivehealth/topics/gender_rights/defining_sexual_health/en/index.html. March 8.
105. World Health Organization. Gender and human rights. Sexual health. 2010. Retrieved from http://www.who.int/reproductivehealth/topics/gender_rights/sexual_health/en/index.html. March 8.

POP and Impact of Surgery on Female Sexual Life

7

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Abbreviations

CSP	Colposacropexy
FSFI	Female Sexual Function Index
HSP	Hysterosacropexy
PISQ	Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire
PISQ-12	Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire-short form
PISQ-IR	Pelvic Organ Prolapse/Incontinence Sexual Questionnaire IUGA-Revised
POP	Pelvic organ prolapse
QOL	Quality of life
SSH	Sacrospinous hysteropexy

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_7

7.1 Introduction

Sexual dysfunction is present in almost one third of women with pelvic organ prolapse (POP), as epidemiological studies have shown [1–3]. *POP can cause loss of self-confidence, difficulties in relating to the other sex and even abandonment of sexual intercourse* [4]. Women with POP are less satisfied with their sexual activity compared to women without: they show a lower degree of emotional closeness to their partner and say that they feel desire and pleasure less frequently. Also, they are more likely to feel pain during intercourse [5]. Anatomically, this could be due to mechanical obstruction or laxity of the vaginal wall or both. In addition, when POP and urinary incontinence are associated, sexual activity worsened because of coital incontinence that can be present concomitantly.

It has been shown that women seeking treatment for advanced prolapse have lower quality of life scores [6, 7], and their body image is decreased. *The value of body image in female sexual function* is an aspect that has been investigated only recently. *It often leads to feelings of shame, embarrassment and the impression of being less sexually attractive, all of which reduce quality of life (QoL).* It also has a negative impact on sexual function, particularly on desire and sexual satisfaction [8].

Women not frequently discuss the impact of POP on sexual function with their doctors. Improvement in sexual life is a commonly reported treatment goal for them since they expect that the surgery will allow them to recover the ability to be intimate without feeling embarrassed [9].

Sexual function may improve with anatomical correction of POP. The gains from the correction of POP must be weighed against the effects of surgery in the vaginal area. *Since the vagina is a sexual organ and its adequate functioning is dependent on normal anatomical and neurovascular factors, we can expect that any POP surgery will affect sexual function* [10, 11].

So far, the data on the impact of POP surgery on sexual life are inconclusive. The discrepancy in these results may be due to both cultural and methodological factors. First, expectations from sexual relationships and woman's comfort in expressing her sexual problems vary with their country of origin and cultural background. Secondly, methodological differences make difficult to compare varying outcomes from different studies and to interpret differences in how those outcomes are measured, e.g. how to quantify sexual responses.

The questionnaires are the best instruments to measure sexual function. Among them, the Female Sexual Function Index (FSFI) is a generic questionnaire that has been used in many studies to assess sexual function in women with POP before and after surgery [12]. The Pelvic Organ Prolapse/Urinary Incontinence Sexual Function Questionnaire (PISQ), together with its short form (PISQ-12), is another validated specific sexual function questionnaire that was designed to assess changes in sexual health. It was and still is the first specific questionnaire for the evaluation of sexual function in heterosexual women with pelvic floor disorders who are sexually active [13, 14]. A recently reviewed version of PISQ, the Pelvic Organ Prolapse/Incontinence Sexual Questionnaire IUGA-Revised (PISQ-IR), has been validated. This was designed as a condition-specific measure of sexual function in women

with pelvic floor dysfunction, including urinary and anal incontinence and POP. In addition, the items of the questionnaire were designed to evaluate both sexually active and sexually inactive women and were written to be independent of sexual orientation [15].

Several techniques – including vaginal, open abdominal and laparoscopic approaches – have been proposed to correct POP, with varying success rates. It is also now accepted that different techniques in POP surgery have very different impacts on sexual function. Considering that surgical dissection may result in tissue damage, devascularisation and denervation, which may lead to a drop in vaginal blood flow and more fibrosis, what happens in female sexuality after POP repair with each type of surgery?

7.2 Vaginal POP Surgery Using Native Tissue

For native tissue repair, a recent systematic review has been published [16]. This review only included studies that reported on sexual function of sexually active women with symptomatic prolapse who underwent vaginal surgery with native tissues and in which women acted as their own controls. Papers including patients who had concomitant incontinence surgery or vaginal mesh procedures were excluded from this review. The study concluded that *sexual function is significantly improved after surgery*. Dyspareunia was significantly reduced following native tissue POP surgery, and postoperative chances of improvement or no change in dyspareunia was 4.8 times greater than the risk of deterioration. However, when a colpoperineorrhaphy was performed in combination with an anterior repair, no improvement in sexual function and higher rates of dyspareunia rates were reported [17–19].

The use of levator plication sutures to repair the posterior compartment and enterocele is considered the main reason causing dyspareunia: if performed, levator plication needs careful assessment of vaginal width to ensure that vaginal capacity is not compromised by overtight sutures [20]. Dyspareunia can also be caused by excessive scarring with consequent lack of elasticity of the vagina.

7.3 Vaginal POP Surgery with Mesh

Vaginal surgery with synthetic mesh or graft materials was proposed to reinforce the pubocervical and rectovaginal fascia in order to provide support to the pelvic floor. *Implantation of synthetic mesh* might damage vaginal innervation and vascularisation, which *could cause sexual dysfunction* and in some cases may cause the new onset or worsening of dyspareunia. This may be due to a decrease in stress shielding of the underlying vaginal wall after implantation of mesh, which in turn may result in an atrophic stiff vagina and less lubrication [21]. In addition, considering that the most common complications of POP surgery with mesh are mesh exposure (Fig. 7.1) and shrinkage, we can assume that the first complication could explain, at least

partly, the increased dyspareunia reported by some patients, while the presence of shrinkage may result in a less pliable vagina and consequent pain during intercourse, worsening significantly sexual function [22].

Other aspects of sexual function remain unchanged. No significant difference is reported in degree of sexual activity, sexual desire, sexual arousal, orgasm and satisfaction. There is no correlation between these results and anatomical correction [23, 24] or position of the mesh: there are no beneficial or detrimental effects in sexual function after either anterior only or anterior and posterior mesh positioning.

In the last Cochrane systematic review on the surgical management of POP, Maher et al. analysed sexual function and dyspareunia in women undergoing mesh surgery in the anterior, posterior and apical compartments [25]. After anterior polypropylene mesh repair, no differences in sexual function or de novo dyspareunia were identified when compared with anterior colporrhaphy (Table 7.1).

Finally insufficient informations are available to provide evidence-based recommendations on sexual function after vaginal mesh repair in the posterior compartment. However, after the 2011 FDA advertisement, confirmed in 2014, the use of mesh for posterior compartment is not recommended.

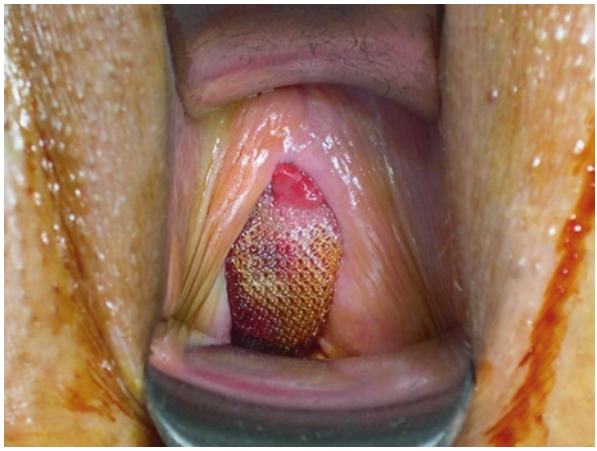

Fig. 7.1 Mesh exposure

Table 7.1 De novo dyspareunia: studies comparing native tissue repair and mesh repair

	Native tissue repair n/N (%)	Mesh repair n/N (%)
Sivaslioglu (2008) [26]	0/42 (0)	2/43 (4.7)
Nguyen (2008) [27]	4/26 (15.4)	2/22 (9.1)
Allahdin (2008) [28]	3/9 (33.3)	3/12 (25 %)
Carey (2009) [29]	13/60 (21.7)	11/62 (17.7)
Iglesia (2010) [30]	3/14 (21.4)	1/11 (9.1)
Altman (2011) [31]	2/101 (1.98)	8/110 (7.3)
Vollebregt (2011) [32]	2/21 (9.5)	3/20 (15)
Withagen (2011) [33]	3/29 (10.3)	3/37 (8.1)

7.4 Abdominal and Laparoscopic Surgery

The most studied procedure for correction of advanced POP is colposacropexy (CSP). Since its introduction by Lane in 1962 [34], CPS has proven to be an effective and durable technique for correcting apical prolapse, giving significant improvements in prolapse symptoms, urinary function and QoL.

Traditionally, CSP has been performed via a laparotomy (abdominal sacral colpopexy); however the use of laparoscopic and robotic approaches is increasing. To date, only few studies have focused on the relationship between CSP and sexuality, but the results so far are positive. Using a validated questionnaire, Costantini et al. showed that patients had significant post-op improvements in their total FSFI scores and in the domains of desire, arousal and orgasm [35]. Thibault et al. demonstrated that this improvement occurs in the first 3 months after surgery [36].

7.5 The Role of Hysterectomy

Over the past few decades, the psychological and emotional value of reproductive organs has changed profoundly. Uterus-sparing surgery is becoming more popular worldwide, and consensus is growing that the uterus can be preserved at the time of pelvic reconstructive surgery in appropriately selected women who desire it.

In fact, anatomical modifications induced by hysterectomy can damage sexuality through lack of uterine contractions and altered perception of orgasm. Moreover hysterectomy can cause vaginal shortening and damage to nerve endings and may also have psychological effects with loss of self-esteem [37]. Damage to the innervation of the uterine cervix and the upper vagina following hysterectomy could affect lubrication and orgasm. The internal orgasm occurs essentially in the cervix and is provoked by stimulation of the fibre endings of the uterovaginal plexus that surround the cervix and the upper vagina. Therefore, the loss of most of the uterovaginal plexus is likely to have adverse effects on sexuality [38]. On the other hand, uterus-sparing surgery raises concerns about the most suitable approach (vaginal, abdominal, laparoscopic), the risks of partial POP resolution, mesh-associated complications, new onset of urinary incontinence, increased recurrence rate with some approaches, failure to improve urinary function, failure to improve bowel and sexual symptoms, longevity of outcome and lifetime risk of cancer [39].

Sacrospinous hysteropexy (SSH) is one of the most studied vaginal techniques for uterus preservation and consists in the unilateral attachment of the posterior uterine cervix or the uterosacral ligaments to the right sacrospinous ligament, about 2 cm medial from the ischial spine, using a combination of permanent and delayed absorbable sutures. Favourable results have been demonstrated on sexuality, although the majority of these studies are flawed by selection and information bias, short follow-up or lack of adequate control groups (Table 7.2).

Abdominal and laparoscopic hysterosacropexy has the main advantage of ensuring a secure proximal and distal vaginal anchorage without tension with a normal vaginal axis and a good vaginal length, which is mandatory for sexual activity.

Table 7.2 Studies comparing vaginal hysterectomy and uterus-sparing sacrospinous hysteropexy

Authors	Uterus preservation (#)	Vaginal hysterectomy (#)	Results
Maher (2001) [40]	34	36	Objective success 74 % vs 72 % Subjective success 78 % vs 86 % Satisfaction 85 % vs 86 % Operating time 59 vs 91 min Mean blood loss 198 vs 402 ml
Hefni and El-Toukly (2003) [41]	61	48	Apical recurrence 6.5 % vs 4.1 % Recurrent cystocele 11.4 % vs 10.4 %
van Brummen (2003) [42]	54	49	Cure rate 88.6 % vs 93 % (ns)
Dietz (2010) [43]	35	31	Apical recurrence 27%vs 3 % Reoperations rate 11 % vs 7 %

Comparing patients who underwent hysterosacropexy (HSP) and hysterectomy with CSP, Costantini et al. demonstrated that there were no differences in outcomes, in terms of subjective, objective and patient's satisfaction [44]. The advantages of HSP include maintaining pelvic anatomy integrity, which is crucial in prolapse resolution, and significantly reducing blood loss, operating times and hospital stays, while the main disadvantage is the continuous surveillance required for cervical and uterine cancer. A further study of the same group demonstrated no substantial differences regarding sexual activity in patients in which the uterus has been spared (HPS group) as opposed to those in whom it has been removed (CSP group): data obtained from FSFI questionnaire demonstrated that there were no significant differences between the two groups in terms of total score and also comparing the examined individual domains – desire, arousal, lubrication, orgasm, satisfaction and pain [45]. The main reason for these results is that, as the uterus preservation is a woman's choice, the two groups represent different kinds of women, i.e. the women who decided to remove the uterus are women in which it doesn't represent as sexual organ. On the contrary the women who prefer uterus-sparing surgery are women in which the uterus plays a role in their femininity and obviously in their sexual life.

Conclusion

Our knowledge on female sexual function is still in its initial stages. In the past, little or no consideration has been given to the problem of sexuality among patients who underwent pelvic surgery for urogenital prolapse, and only in recent years, this aspect has been emphasised.

Much deeper investigations are needed to clarify the effect on sexuality of variables such as educational level, profession and cultural differences, which are not directly related to POP status but could represent major biases. The evaluation of sexual function through specific questionnaires makes easier to identify the sexual dysfunctions associated with the pelvic floor disorders.

Moreover, *data on sexual outcome should be included in preoperative counselling and in outcome evaluation after POP surgery*. The inclusion of a new condition-specific questionnaire as an outcome measure allows an analysis of the impact of POP treatments on women's sexual function. The ability of the PISQ-IR to document condition-specific impact and to measure symptoms suggests this robust tool may be more useful than generic sexual questionnaires for characterising symptoms in women with POP.

References

1. Handa VL, Cundiff G, Chang HH, Helzlsouer KJ. Female sexual dysfunction and pelvic floor disorders. Obstet Gynecol. 2008;111:1045–52.
2. Barber MD, Visco AG, Wyman JF, Fantl JA, Bump RC, Continence Program for Women Research Group. Sexual function in women with urinary incontinence and pelvic organ prolapse. Obstet Gynecol. 2002;99(2):281–9.
3. Ellerkmann RM, Cundiff GW, Melick CF, Nihira MA, Leffler K, Bent AE. Correlation of symptoms with location and severity of pelvic organ prolapse. Am J Obstet Gynecol. 2001;185(6):1332–7; discussion 1337–8.
4. Filocamo MT, Serati M, Frumenzio E, Li Marzi V, Cattoni E, Champagne A, Salvatore S, Nicita G, Costantini E. The impact of mid-urethral slings for the treatment of urodynamic stress incontinence on female sexual function: a multicenter prospective study. J Sex Med. 2011;8(7):2002–8.
5. Athanasiou S, Grigoriadis T, Chalabalaki A, Proptopapas A, Antsaklis A. Pelvic organ prolapse contributes to sexual dysfunction: a cross-sectional study. Acta Obstet Gynecol Scand. 2012;91(6):704–9.
6. Jelovsek JE, Barber MD. Women seeking treatment for advanced pelvic organ prolapse have decreased body image and quality of life. Am J Obstet Gynecol. 2006;194:1455–61.
7. Lowenstein L, Gamble T, Deniseiko Sanses TV, et al. Sexual function is related to body image perception in women with pelvic organ prolapse. J Sex Med. 2009;6:2286–91.
8. Zielinski R, Miller J, Low LK, Sampselle C, DeLancey JO. The relationship between pelvic organ prolapse, genital body image, and sexual health. Neurourol Urodyn. 2012;31:1145–8.
9. Sung VW, Rogers RG, Barber MD, Clark MA. Conceptual framework for patient-important treatment outcomes for pelvic organ prolapse. Neurourol Urodyn. 2014;33(4):414–9.
10. Lakeman MM, Laan E, Roovers JP. The effects of prolapse surgery on vaginal wall sensibility, vaginal vasocongestion, and sexual function: a prospective single centre study. Neurourol Urodyn. 2014;33:1217–24.
11. Weber MA, Lakeman MM, Laan E, Roovers JP. The effects of vaginal prolapse surgery using synthetic mesh on vaginal wall sensibility, vaginal vasocongestion, and sexual function: a prospective single-center study. J Sex Med. 2014;11:1848–55.
12. Rosen R, Brown C, Heiman J, Leiblum S, Meston C, Shabsigh R, Ferguson D, D'Agostino Jr R. The Female Sexual Function Index (FSFI): a multidimensional self-report instrument for the assessment of female sexual function. J Sex Marital Ther. 2000;26:191–208.
13. Rogers RG, Kammerer-Doak D, Villarreal A, Coates K, Qualls C. A new instrument to measure sexual function in women with urinary incontinence or pelvic organ prolapse. Am J Obstet Gynecol. 2001;184:552–8.
14. Rogers RG, Coates KW, Kammerer-Doak D, Khalsa S, Qualls C. A short form of the pelvic organ prolapse/urinary incontinence sexual questionnaire (PISQ-12). Int Urogynecol J Pelvic Floor Dysfunct. 2003;14:164–8.
15. Rogers RG, Rockwood TH, Constantine ML, Thakar R, Kammerer-Doak DN, Pauls RN, Parekh M, Ridgeway B, Jha S, Pitkin J, Reid F, Sutherland SE, Lukacz ES, Domoney C, Sand

P, Davila GW, Espuna Pons ME. A new measure of sexual function in women with Pelvic Floor Disorders (PFD): the Pelvic Organ Prolapse/Incontinence Sexual Questionnaire, IUGA-Revised (PISQ-IR). Int Urogynecol J. 2013;24:1091–103.
16. Jha S, Gray T. A systematic review and meta-analysis of the impact of native tissue repair for pelvic organ prolapse on sexual function. Int Urogynecol J. 2015;26:321–7.
17. Colombo M, Vitobello D, Proietti F, Milani R. Randomised comparison of Burch colposuspension versus anterior colporrhaphy in women with stress urinary incontinence and anterior vaginal wall prolapse. BJOG. 2000;107(4):544–51.
18. Haase P, Skibsted L. Influence of operations for stress incontinence and/or genital descensus on sexual life. Acta Obstet Gynecol Scand. 1988;67(7):659–61.
19. Dua A, Jha S, Farkas A, Radley S. The effect of prolapse repair on sexual function in women. J Sex Med. 2012;9(5):1459–65.
20. Ulrich D, Dwyer P, Rosamilia A, Lim Y, Lee J. The effect of vaginal pelvic organ prolapse surgery on sexual function. Neurourol Urodyn. 2015;34(4):316–21.
21. Jones KA, Feola A, Meyn L, Abramowitch SD, Moalli PA. Tensile properties of commonly used prolapse meshes. Int Urogynecol J Pelvic Floor Dysfunct. 2009;20(7):847–53. doi:10.1007/s00192-008-0781-x. Epub 2009 Mar 28.
22. Vollebregt A, Fischer K, Gietelink D, van der Vaart CH. Effects of vaginal prolapse surgery on sexuality in women and men; results from a RCT on repair with and without mesh. J Sex Med. 2012;9(4):1200–11.
23. Long CY, Juan YS, Wu MP, Liu CM, Chiang PH, Tsai EM. Changes in female sexual function following anterior with and without posterior vaginal mesh surgery for the treatment of pelvic organ prolapse. J Sex Med. 2012;9(8):2167–74.
24. Altman D, Elmér C, Kiilholma P, Kinne I, Tegerstedt G, Falconer C, Nordic Transvaginal Mesh Group. Sexual dysfunction after trocar-guided transvaginal mesh repair of pelvic organ prolapse. Obstet Gynecol. 2009;113(1):127–33.
25. Maher C, Feiner B, Baessler K, Schmid C. Surgical management of pelvic organ prolapse in women. Cochrane Database Syst Rev. 2013;4:CD004014.
26. Sivaslioglu AA, Unlubilgin E, Dolen I. A randomized comparison of polypropylene mesh surgery with site-specific surgery in the treatment of cystocoele. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(4):467–71.
27. Nguyen JN, Burchette RJ. Outcome after anterior vaginal prolapse repair: a randomized controlled trial. Obstet Gynecol. 2008;111(4):891–8.
28. Allahdin S, Glazener C, Bain C. A randomised controlled trial evaluating the use of polyglactin mesh, polydioxanone and polyglactin sutures for pelvic organ prolapse surgery. J Obstet Gynaecol. 2008;28(4):427–31.
29. Carey M, Higgs P, Goh J, Lim J, Leong A, Krause H, Cornish A. Vaginal repair with mesh versus colporrhaphy for prolapse: a randomised controlled trial. BJOG. 2009;116(10):1380–6.
30. Iglesia CB, Sokol AI, Sokol ER, Kudish BI, Gutman RE, Peterson JL, Shott S. Vaginal mesh for prolapse: a randomized controlled trial. Obstet Gynecol. 2010;116(2 Pt 1):293–303.
31. Altman D, Väyrynen T, Engh ME, Axelsen S, Falconer C, Nordic Transvaginal Mesh Group. Anterior colporrhaphy versus transvaginal mesh for pelvic-organ prolapse. N Engl J Med. 2011;364(19):1826–36.
32. Vollebregt A, Fischer K, Gietelink D, van der Vaart CH. Primary surgical repair of anterior vaginal prolapse: a randomised trial comparing anatomical and functional outcome between anterior colporrhaphy and trocar-guided transobturator anterior mesh. BJOG. 2011;118(12):1518–27.
33. Withagen MI, Vierhout ME, Hendriks JC, Kluivers KB, Milani AL. Risk factors for exposure, pain, and dyspareunia after tension-free vaginal mesh procedure. Obstet Gynecol. 2011;118(3):629–36.
34. Lane FE. Repair of posthysterectomy vaginal-vault prolapse. Obstet Gynecol. 1962;20:72–7.
35. Costantini E, Porena M, Lazzeri M, Mearini L, Bini V, Zucchi A. Changes in female sexual function after pelvic organ prolapse repair: role of hysterectomy. Int Urogynecol J. 2013;24(9):1481–7.

36. Thibault F, Costa P, Thanigasalam R, Seni G, Brouzyine M, Cayzergues L, De Tayrac R, Droupy S, Wagner L. Impact of laparoscopic sacrocolpopexy on symptoms, health-related quality of life and sexuality: a medium-term analysis. BJU Int. 2013;112(8):1143–9.
37. Achtari C, Dwyer PL. Sexual function and pelvic floor disorders. Best Pract Res Clin Obstet Gynaecol. 2005;19(6):993–1008.
38. Hasson HM. Cervical removal at hysterectomy for benign disease. Risks and benefits. J Reprod Med. 1993;38(10):781–90.
39. Costantini E, Lazzeri M, Zucchi A, Bini V, Mearini L, Porena M. Five-year outcome of uterus sparing surgery for pelvic organ prolapse repair: a single-center experience. Int Urogynecol J. 2011;22(3):287–92.
40. Maher CF, Cary MP, Slack MC, Murray CJ, Milligan M, Schluter P. Uterine preservation or hysterectomy at sacrospinous colpopexy for uterovaginal prolapse? Int Urogynecol J Pelvic Floor Dysfunct. 2001;12(6):381–4.
41. Hefni M, El-Toukhy T, Bhaumik J, Katsimanis E. Sacrospinous cervicocolpopexy with uterine conservation for uterovaginal prolapse in elderly women: an evolving concept. Am J Obstet Gynecol. 2003;188(3):645–50.
42. van Brummen HJ, van de Pol G, Aalders CI, Heintz AP, van der Vaart CH. Sacrospinous hysteropexy compared to vaginal hysterectomy as primary surgical treatment for a descensus uteri: effects on urinary symptoms. Int Urogynecol J Pelvic Floor Dysfunct. 2003;14(5):350–5; discussion 355.
43. Dietz V, van der Vaart CH, van der Graaf Y, Heintz P, Schraffordt Koops SE. One-year follow-up after sacrospinous hysteropexy and vaginal hysterectomy for uterine descent: a randomized study. Int Urogynecol J. 2010;21(2):209–16.
44. Costantini E, Mearini L, Bini V, Zucchi A, Mearini E, Porena M. Uterus preservation in surgical correction of urogenital prolapse. Eur Urol. 2005;48:642–9.
45. Zucchi A, Costantini E, Mearini L, Fioretti F, Bini V, Porena M. Female sexual dysfunction in urogenital prolapse surgery: colposacropexy vs hysterocolposacropexy. J Sex Med. 2008;5:139–45.

Urinary Incontinence and Mid-urethral Slings: Which Is the Impact on Female Sexual Life?

8

Elisabetta Costantini

List of Abbreviations

FSD	Female sexual dysfunctions
MUS	Mid-urethral sling
SUI	Stress urinary incontinence
TOT	Trans-obturator tape
TVT	Tension-free vaginal tape
UI	Urinary incontinence

Over the last 3 decades, more than 500 studies have been published in the field of urinary incontinence and sexual function. The reason for this increasing interest is that, traditionally, outcome studies in urogynecology focused on anatomic and functional changes after surgery and they neglected quality of life issues. The new condition-specific health-related quality of life instruments have improved our ability to assess the impact of pelvic floor surgery more fully, and the concept of patient-centered care has led us to focus on the patient's perspective or expectations. Furthermore since the World Health Organization declared female sexuality a basic human right [1], sexual well-being is recognized as an important aspect of women's health, and sexual dysfunctions can lessen or worsen quality of life.

Many studies reported that sexual problems are more prevalent in patients with pelvic floor dysfunctions demonstrating that stress urinary incontinence (SUI), overactive bladder, and in general lower urinary tract symptoms impact negatively on all sexual function domains.

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_8

Urinary incontinence (UI) is a common condition affecting 12–46 % of the adult female population [2]. This condition is associated with a significant worsening of the quality of life aspects, influencing social, physical, psychological, occupational, and sexual behaviors [2, 3]. *Sexual dysfunction has been reported in up to 45 % of female incontinent patients* with coital incontinence reported in 11 % of the women attending a urogynecological unit [4]. The reasons for the increased sexual function impairment in women with urinary incontinence may be due to both direct and indirect effects. Stress urinary incontinence (SUI) may affect sexual desire, lubrication, orgasm, and sexual satisfaction; irritation of vulvovaginal region associated with chronic urine leakage usually leads to dyspareunia. Also, patients may fear urine leakage during engaging in any sexual activity which may indirectly contribute to decreased sexual interest and lower sexual self-esteem [5]. So the link between female sexual dysfunctions (FSDs) and urinary incontinence is well demonstrated, and *incontinence impacts upon different sexual domains,* desire, pain, and orgasm, causing *loss of self-confidence, difficulties in relating to the other sex, and even abandonment of sexual intercourse*. In this scenario, assuming that urinary leakage contributes to sexual dysfunction, it is reasonable to presume that an operation designed to recover continence should also improve sexual function.

Today, minimally invasive anti-incontinence procedures (Fig. 8.1) such as a tension-free vaginal tape (TVT), a polypropylene mesh placed at the mid-urethra and anchored through the retropubic space, or a mid-urethral trans-obturator tape (TOT), inserted in a horizontal plane underneath the middle urethra between the two obturator foramen, are currently offered as the best approach to SUI and in the majority of the cases replaced other traditional procedure, such as a Burch colposuspension [6].

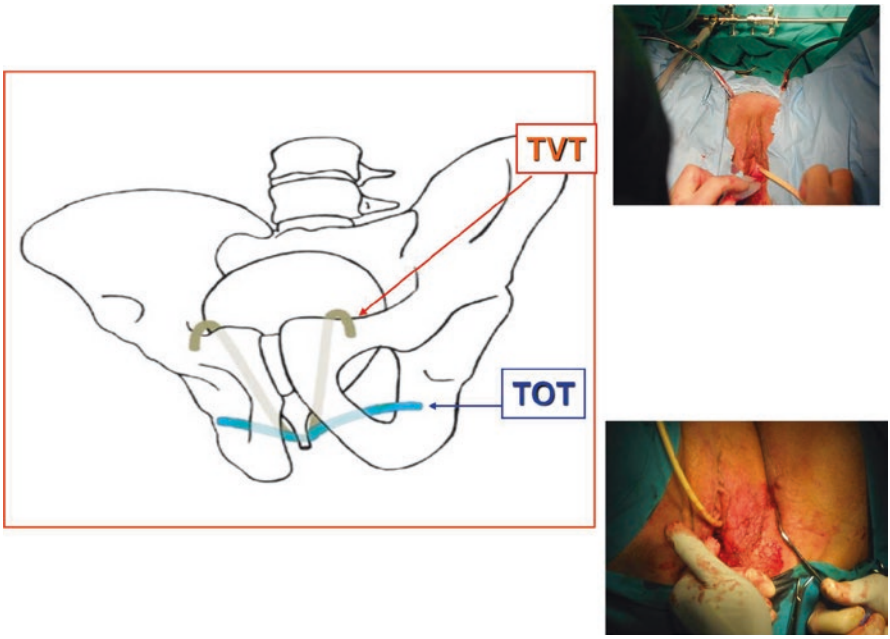

Fig. 8.1 Tension-free vaginal tape (retropubic position of the mesh) and tran-obturator tape (tran-obturator position of the mesh

Only recently the impact of mid-urethral slings (MUSs) on female sexual function has been reported with controversial results [7–11]. The majority of the studies focused on curing the incontinence rather than the impact of these procedures on sexual function so that the effects of MUS procedures on women's sexual lives remain still uncertain. Some studies reported improved function after TVT and TOT procedures [10, 12, 13], but others reported deterioration or no change in symptoms. Study design including varying age, hormonal status, comorbidities, heterogeneity of coexistence of pelvic organ prolapses, difference evaluation criteria on success, and retrospective design of studies have been shown to account for these variable results [14].

The urethrovaginal space plays an important role in female arousal and sensation of orgasm [15], so the insertion of the sling may disrupt the anatomy of this space and modify the innervations of the anterior vaginal wall which leads to dyspareunia and anorgasmia. In addition, it was suggested that TOT and TOT-like procedures may injure the pudendal nerve branches and alter the somatosensitive pathway of the vulva causing sexual dysfunctions de novo. However, such neuroanatomic pathway was not definitely confirmed [16]. On the other hand [17], vaginal surgery for SUI may cause vaginal narrowing, scarring and reduction of the anterior vaginal wall elasticity, reduction of blood supply to the erectile tissue of the clitoris, tape erosion (which cause dyspareunia and partner discomfort), decreased genital sensation and vaginal lubrication, as well as persistent urine leakage during intercourse [16, 18]. All these factors, as confirmed by different authors [11, 19], are however the results of technique failure, and they are not always related to the surgical technique itself [20]. In fact some well-conducted studies demonstrated an increase in desire, arousal, lubrication, orgasm, and satisfaction [11, 21] in female incontinent patients after MUSs as demonstrated in a group of women who filled the Female Sexual Function Questionnaire before and after surgery (Fig. 8.2) [11]. After anti-incontinence procedures the patients improved their quality of sexual life, restored libido, increased sexual self-confidence,

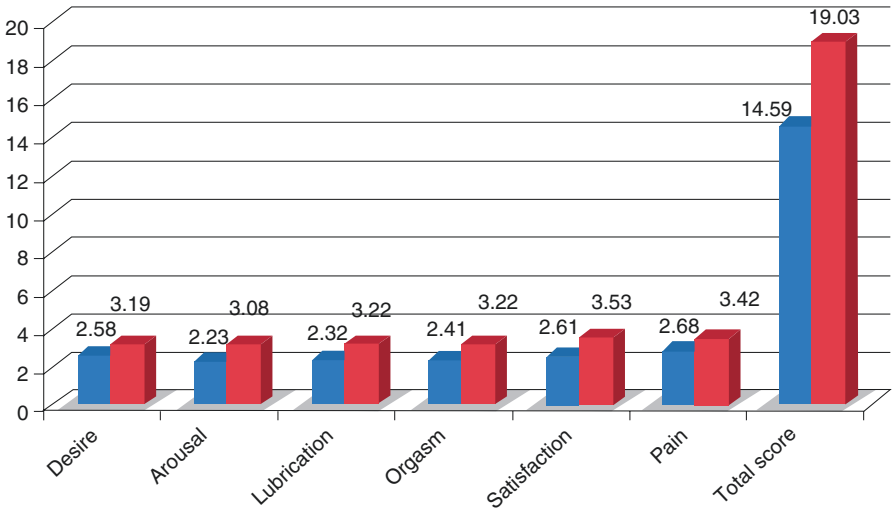

Fig. 8.2 When the total score and all sub-scores in the FSFI questionnaire were compared, a significant improvement in sexual function was found after MUS

and in some cases restored sexual life in those women who avoided and abandoned sexual life for the incontinence problems. It is important in this view to underline that the majority of the studies included only patients who were sexually active and excluded those for whom medical and/or psychological factors have precluded any sexual relationship. Including patients not sexually active, it is possible to recognize a group of women with a restored sexual life with the number of sexually active women increasing from 59 to 71 % after MUS [11]. So *following MUS placement, sexual function improves*, and a relevant percentage of nonsexually active women renews sexual activity because of MUS. *The development of any sexual problem after MUS surgery is probably because of anatomical reasons,* such as an incorrect positioning of the sling, and/or an abnormal scar formation, and/or exposure of the mesh or de novo symptoms such as the de novo storage symptoms.

In conclusion, female sexual dysfunction is a difficult and complex issue, and it is clear that urinary incontinence negatively impacts sexual function. The multidimensional aspects of female sexuality are often difficult to standardize, and we cannot forget the partner's role. After anti-incontinence surgery, the results on sexual function depend on different factors, and generally the studies are not able to give us a clear answer because they did not focus on the different aspect of female sexuality and they did not correlate technique failures and complications with the female sexual dysfunctions. In the attempt to summarize, we *can recognize three different groups of patients* (Fig. 8.3): (1) *women with Hypoactive Sexual Desire Disorders or women with no partner*, who do not wish to have or to restore sexual activity, *and in this case surgery has no impact on sexuality*. (2) *women with incontinence-related female sexual dysfunctions who restored or improved sexual activity after MUS* if incontinence is resolved; and (3) *women with surgery-related persistent or de novo female sexual disorders* after MUS which were often related to a complication such as a mesh exposure or a mesh retraction or to a dissatisfactory outcome. In this group *FSDs persist or compare de novo*.

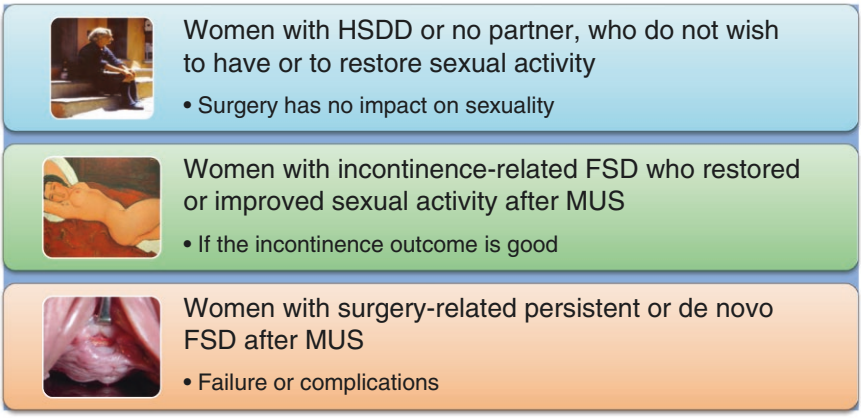

Fig. 8.3 Results on sexuality after MUS depend on 3 different situations due to woman's characteristics

References

1. World Health Organization. Sexual health: working definitions. 2002. Available at: http://www.who.International/reproductive-health/gender/sexual_health.html.
2. Thankar R. Review of current status of female sexual dysfunction evaluation in urogynecology. Int Urogynecol J Pelvic Floor Dysfunct. 2009;20 Suppl 1:S27–31.
3. Pauls RN, Segal JL, Silva WA, Kleeman SD, Karram MM. Sexual function in patients presenting to a urogynecology practice. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17:576–80.
4. Liang CC, Tseng LH, Lo TS, Lin YH, Lin JR, Chang SD. Sexual function following outside-in transobturator midurethral sling procedures: a prospective study. Int Urogynecol J. 2012;23:1693–8.
5. Witek A, Drosdzol-Cop A, Nowosielski K, Solecka A, Mikus K. Long-term impact of surgical repair for stress urinary incontinence on female sexual functions, distress and behaviours. J Clin Nurs. 2012;22:1591–8.
6. Novara G, Artibani W, Barber MD, Chapple CR, Costantini E, Ficarra V, Hilton P, Nilson CG, Waltregny D. Updated systematic review and meta-analysis of the comparative data on colposuspensions, pubovaginal slings, midurethral tapes in the surgical treatment of stress urinary incontinence. Eur Urol. 2010;58:218–38.
7. Glavind K, Tetsche MS. Sexual function in women before and after suburethral sling operation for stress urinary incontinence: a retrospective questionnaire study. Acta Obstet Gynecol Scand. 2004;83:965–8.
8. Helzevier HW, Venema PL, Nijeholt AA. Sexual function after Tension-free Vaginal Tape (TVT) for stress incontinence: Results of mailed questionnaire. Int Urogynecol J Pelvic Floor Dysfunct. 2004;15:313–8.
9. Mazouni C, Karsenty G, Bretelle B, Bladou F, Gamerre M, Serment G. Urinary complications and sexual function after the tension-free vaginal tape procedure. Acta Obstet Gynecol Scand. 2004;83:955–61.
10. Jha S, Radley S, Farkas A, Jones G. The impact of TVT on sexual function. Int Urogynecol J Pelvic Floor Dysfunct. 2009;20:165–9.
11. Filocamo MT, Serati M, Frumenzio E, Li Marzi V, Cattoni E, Champagne A, Salvatore S, Nicita G, Costantini E. The impact of mid-urethral slings for the treatment of urodynamic stress incontinence on female sexual function: a multicenter prospective study. J Sex Med. 2011;8:2002–8.
12. Bekker M, Beck J, Putte H, et al. Sexual function improvement following surgery for stress incontinence: the relevance of coital incontinence. J Sex Med. 2009;6:3208–13.
13. Narin R, Attar R, Narin MA, Koyuncu D, Yencilek E. Impact of transobturator tape procedure on female and their partner sexual function: it improves sexual function of couples. Arch Gynecol Obstet. 2014;290:913–7. doi:10.1007/s00404-014-3259-8.
14. Simsek A, Ozgor F, Yuksel B, et al. Female sexual function after transobturator tape in women with urodynamic stress urinary incontinence. Springerplus. 2014;3:570. Published online 2014 Sep 30. doi: 10.1186/2193-1801-3-570.
15. Battaglia C, Nappi RE, Mancini F, Alvisi S, Del Forno S, Battaglia B, Venturoli S. 3D volumetric and vascular analysis of the urethrovaginal space in young women with or without vaginal orgasm. J Sex Med. 2010;7:1445–53.
16. Elzevier HW, Putter H, Delaere KPJ, et al. Female sexual function after surgery for stress urinary incontinence: transobturator suburethral tape vs. tension-free vaginal tape obturator. J Sex Med. 2008;5:400–6.
17. Caruso S, Rugolo S, Bandiera S, Mirabella D, Cavallaro A, Cianci A. Clitoral blood flow changes after surgery for stress urinary incontinence: pilot study on TVT versus TOT procedures. Urology. 2007;70:554–7.
18. Dragisic KG, Milad MP. Sexual functioning and patient expectations of sexual functioning after hysterectomy. Am J Obstet Gynecol. 2004;190:1416–8. doi:10.1016/j.ajog.2004.01.070.

19. Serati M, Salvatore S, Uccella S, Zanirato M, Cattoni E, Nappi RE, Bolis P. The impact of the mid-urethral slings for the treatment of stress urinary incontinence on female sexuality. J Sex Med. 2009;6:1534–42.
20. Basson R, Wierman ME, van Lankveld J, Brotto L. Summary of the recommendations on sexual dysfunctions in women. J Sex Med. 2010;7:314–26.
21. Pace G, Vicentini C. Female sexual function evaluation of the Tension-free Vaginal Tape (TVT) and Transobturator Suburethral Tape (TOT) incontinence surgery: results of a prospective study. J Sex Med. 2008;5:387–93.

Female Sexual Life During Malignancies

9

Donata Villari

Nevertheless
The bookbag on my back. I'm out the door.
Winter turns to spring
The way it does, and I buy dresses.
A year later, it gets to where
When they say How are you feeling,
With that anxious look on their faces,
And I start to tell them the latest
About my love life or my kids' love lives,
Or my vacation or my writer's block-
It actually takes me a while
To realize what they have in mind-
I'm fine, I say, I'm great, I'm clean.
The bookbag on my back, I have to run.
Alicia Ostriker
from
The Crack in Everything.
University of Pittsburgh Press, 1996

We do not have a body; we are our body. Our body is our self.

Usually, when we are well, we are not aware of our body, and we exist (from the Latin *ex-sistere*) on top of, intent on, and involved in the world. Through the body, in one way or another, we are projected into the world.

In sickness, this project is interrupted, and the body comes into the foreground, announcing its presence with pain and discomfort.

Cancer shuffles the cards. It creates a deep fracture in our being. The body, out of which we look at a horizon, becomes our horizon. It is a horizon marked by medical appointments, surgical interventions, diagnoses, and treatments. In more serious pathologies, especially in the last phases, which lead inevitably to the end of life, our existence is no longer projected into the world; rather it becomes an "insistence" inside our body, to the point where we sink into it.

The Cartesian mind-set has habituated us to a dichotomy between mind and body, psyche and soma. The body is not a living body but a "body thing" that the mind

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_9

observes from outside. In the terms used by the German philosopher Karl Theodor Jaspers, this body thing is the *corp*, while the living body is the *leib*. But the *leib* is nothing but the body that relates us to the world and to others and which identifies our life as an unrepeatable fact. And the psyche is also the body, in the indivisible unity that we call the bodily self. And if the psyche is the set of emotions, thought, and perceptions that signify our relationships with others, the world, and our own subjectivity, it is impossible to understand it as separate from the living body.

Thus, illness is in any case always psychosomatic – *if not in its causes* – at least in its course. So we might say that every person has her/his own illness, whose symptoms are not only signs but also meanings that are part of our biography, expressed in the biography of our body, like our life and our death.

This is why it is important in the treatment of cancer to respect subjectivity, to avoid the homologation and the alienating anonymity all too often proposed and imposed by treatment: not to consider the body as object to be analyzed, classified, subdivided into specializations of organs and diagnosed by infinite deindividualization, but rather as a body "subject," made whole again through the concept of harmony in treatment.

This harmony includes the acceptance of limit, the dialogue with death, and the restructuring of the meanings of one's existence. Thus, confrontation with limit and with pain guides us toward acceptance, but also toward the courage to fight for a return to enjoying the world, to pull ourselves out of disease and adversity as far as possible, governing ourselves with measure and harmony.

It is this context that we feel should frame our discussion of the question of female sexual dysfunctions in the course of oncological diseases.

9.1 The Dimensions of the Problem

Cancer is a worldwide problem, though its global distribution is not uniform [1]. One significant finding that comes to the fore is that women tend to develop neoplasms in a smaller percentage than men. Besides depending on a different lifestyle or hormonal influences, this could be due to the fact that the female immune system is more effective and developed [2], though it is also more susceptible to autoimmune diseases.

It is estimated that in the USA today there are over 13 million persons with a past history of cancer [3]. Fifty-four percent of them are women, and this number is destined to increase. Breast cancer is the prevalent form (41 %), followed by gynecologic cancers (cervix, endometrium, ovary, tube, vulva and vagina) and lastly by cancer of the colon/rectum (9 %) [4].

In 2015 in Italy, not counting skin cancers, almost 366,000 new cases of malign tumors will be diagnosed (approximately 1,000 a day), of which roughly 196,000 (54 %) in men and roughly 169,000 (46 %) in women. Among women, breast cancer is the most frequent, representing 29 % of all tumors, followed by cancers of the colon/rectum (13 %), lung (6 %), thyroid (5 %), and body of the uterus (5 %). Considering the prevalence data as of 2010, it is estimated that roughly 2,250,000

persons (who represent over 4 % of the resident population) living in Italy have previously been diagnosed with a tumor. Of these subjects, almost 1,000,000 are male (44 %) and roughly 1,250,000 female (56 %). The especially high percentage of women with past breast tumors (over 1/5 of all cases prevalent in both sexes) helps to explain the greater presence in the population of women with cancers than of men (66 % of the total prevalent cases) [5].

The incidence of female sexual dysfunctions with oncological causes is certainly greater in countries where the incidence of neoplasia is greater, and it is precisely in these countries that more resources have been invested and more studies carried out concerning this aspect of the quality of life with easier diagnosis.

Women who have had breast cancer make up 23 % of all cancer survivors [6]. Yet there can be no doubt that female sexual dysfunctions represent an underestimated condition in oncology and one that has been less studied than, for example, the aftermath of prostate tumors in men, despite the fact that the average age of onset is far higher in men [6]. There are many reasons for this. All too often the treatment and control of a carcinoma are the only objectives taken into consideration. In a high percentage of cases, it is the physicians themselves, in this case the oncologists, who underestimate the problem and do not give it the time and attention it calls for, also because they are not adequately prepared to suspect, diagnose, classify, and properly treat this condition [7].

This "disattention" on the part of the medical profession is generally attributed to lack of time, so that it is not infrequent for even gynecologic oncologists to fail to perform a careful anamnesis of the sexual life of their patients at the first examination [8]. Similarly, patients undergoing pelvic radiotherapy are not always adequately informed about possible side effects on their sexual life [9]. Women themselves often do not find the courage to ask questions that make their sexual life the focus of the attention of medical staff [10], and this is especially true for older women. We must also bear in mind that fewer than one third of women in the general population who suffer from disturbances linked to some type of sexual dysfunction report this to their doctor [11]. It is therefore opportune for the doctor himself/herself to introduce the subject with naturalness and competence and using clear, explicit language [12].

Nowadays we have finally understood how important it is to focus on this problem, and it has become an essential part of an integrated approach to the treatment of the growing cancer survivor population. To this end, in 2010 a national interdisciplinary network of over 100 doctors, specialists and researchers was instituted in America (National Network), with the precise goal of the prevention, treatment and support of women and girls who suffer from cancer-related sexual dysfunctions [13].

In order to acquire a comprehensive clinical history of their sexual lives and associated problems from our patients before starting any sort of therapeutic treatment, we need not only time but also multidisciplinary competence, empathy and communicative capacity, as well as specifically trained medical personnel. Cancer care providers should also be sure to bring up the "issue" of sexuality with older patients and with those who do not have a fixed partner and to take into consideration the possibility that their patients' sexual lives may involve same-sex partners.

The use of validated questionnaires is crucial for diagnosis and monitoring. Among the many available, one frequently used in clinical studies of cancer populations is the *Sexual Activity Questionnaire (SAQ).* This is a 14-item self-report inventory [14] that takes about 10 minutes to complete. It inquires into the level of existing sexual activity, sorts out the reasons for possible inactivity, and characterizes the sexual behavior of patients using three categories (pleasure, discomfort, and habit). It has also been employed in a study comparing lesbians and heterosexual women with a recent diagnosis of breast cancer [15]. The *Female Sexual Function Index (FSFI)* (a 19-item self-report measure) has been used especially in the study of gynecologic cancers, breast tumors [16] and patients who have undergone radical cystectomy [17].

9.2 Cancer and Sexual Dysfunction

The diagnosis of cancer is the first, critical moment, which causes in hearers feelings of incredulity, fear, and insecurity about their future. Its reverberations also affect the partner, if there is one, who in turn may suffer from preexisting sexual dysfunctions [18, 19].

As is logical, in the past studies on cancer and sexual dysfunction were at first focused on cancers directly or indirectly involving the sexual and reproductive organs. Later research has widened out to include sexual dysfunctions in oncological patients regardless of the seat of the original neoplasia [20].

As can be imagined, gynecologic and breast cancers are those that most frequently have a negative impact on a woman's sexual health. The surgical treatment undergone by these patients creates direct anatomical damage and distorts their body image, causing them to perceive their body as sexually unattractive. This in turn creates changes in the response to the stimuli that influence desire, with inadequate vaginal lubrication and genital swelling that in the end lead to less frequent sex, with the absence of well-being, pleasure and sexual satisfaction, and consequent inability to reach orgasm [21].

A paradigmatic example is represented by premenopausal salpingo-oophorectomy, which leads to the physical and hormonal changes typical of early menopause, seen in the various domains that characterize female sexual dysfunctions as they are currently classified [22]. Besides the aftermaths of surgery, more and more frequently multimodal protocols also consider the consequences of chemo and/or radiotherapy, which can continue to have negative effects after many years. Cancers apparently "distant" from the parts of the body associated with sexuality constitute a separate question. Examples are head and neck tumors, which by causing significant facial alterations (disfigurement), vocal changes (speech), and changes in breathing and salivation, can have a strong impact on self-esteem and therefore on interpersonal and couple relationships [23].

But there is no doubt that there are many other neoplastic sites with a direct negative impact on the sexual lives of men and women, whether as a consequence of primary surgical treatment or of related pharmacologic, radiation, or biological

treatments. Among these are colon/rectal tumors [24], those of the head and neck [25] and of the bladder [26], lymphatic cancers [27], and lung cancers [28].

9.3 Breast Tumors

Breast tumors represent the most frequently diagnosed carcinoma in women in the USA and in industrialized countries in general [28]. Around 12.4% of new cases develop in reproductive age, under 44 years old, and 22.6% in perimenopausal age (between 45 and 54). In the majority of cases, therefore, the diagnosis occurs at an age in which these women are sexually active. As a matter of fact, it is not infrequent that a mammary tumefaction is first discovered by the partner during sexual activity [6].

Treatment consists in mastectomy or conservative surgery followed by radiotherapy. A recent study has shown that women who have had a radical mastectomy followed by reconstruction are less subject to deterioration of their sexual lives than those who have undergone either mastectomy without reconstruction or conservative surgery [29].

Surgical treatment may or may not be followed by adjuvant chemotherapy. A 2005 meta-analysis shows that anthracycline-based protocols are associated with higher toxicity (cardiotoxicity, leukopenia, nausea, vomiting, and alopecia) and also with loss of pubic hair [30]. Cyclophosphamide, methotrexate, and fluorouracil-based regimes cause precocious bleeding and possible amenorrhea within the first year, along with a high risk of infertility. Instead, the use of taxane determines sensory and motor neuropathies at the extremities, which can be correlated to some degree to alterations of clitoral and vulvar sensitivity [31].

When radiotherapy is used, it can have negative effects on the outcomes of reconstructive surgery, which can be esthetically unsatisfactory. Moreover, estrogen modulation and deprivation (through the employment of tamoxifen or LHRH analogues) play an important role in therapies for forms of hormone-receptor-positive breast cancers, seeing as almost one fourth of new diagnoses of breast tumor involve women in pre- or perimenopausal age [32].

As can be easily imagined, therefore, all these therapies and modulations strongly impact the sexual lives of patients, above all through endocrinological manipulation that determines a “menopausal” state as a consequence of estrogen deprivation [33]. The most frequent symptoms are, therefore, vaginal dryness, pain during penetration, fall in or lack of desire, and inability to reach orgasm. Added to these is a state of anxiety about relations with the partner. Some authors report a percentage as high as 76% of sexual dysfunctions discovered through the use of the FSFI questionnaire [34].

9.4 Cancer-Related Pelvic Surgery and Female Sexual Dysfunction

The female pelvic area is often involved in radical surgery in cases of carcinomas that interest the reproductive and genital organs or that develop in the bladder, colon, or anus.

9.4.1 Gynecological Cancer

Roughly 76,500 new cases are diagnosed each year in the USA. The treatment of patients affected with gynecological tumors has important physical, psychological, and social consequences that have a negative impact on their sexual and relational life in 30–100 % of cases [35]. The consequences are the physical ones directly linked to hormonal changes, to the removal of sexual organs (uterus, tubes, ovaries and/or cervix, vulva) [36], and to nerve deterioration, in addition to damage caused by radio- or chemotherapy. There are also psychological repercussions that lead to anxiety, depression, and problems in communicating with the partner [36].

9.4.2 Bladder Cancer

This is the fourth most frequent neoplasia in the USA, with roughly 70,000 new cases diagnosed annually [37].

Radical cystectomy followed by external urinary diversion or by reconstruction of an orthotopic neobladder represents the gold standard for muscle-invasive forms [38]. In cases where it has not been possible to reconstruct a neobladder that allows the patient to urinate "per urethram," the presence of one or two urostomy bags undoubtedly causes a dramatic change in the patient's body image, with consequent strong emotional impact [39].

This carcinoma generally appears at a later age in women and often involves a more negative prognosis, leading to lower cancer-specific survival [40]. Existing literature focuses mainly on male erectile dysfunction, though some partial data concerning women, based on a limited study sample, do exist [18]. Among those who define themselves as sexually active before surgery (87 %), only 37 % declare that they are afterward [17]. The literature on non-muscle-invasive forms, which represent up to 75 % of the incidence of all bladder tumors [41], is even scantier. In 62.5 % of cases, patients report vaginal dryness and in 23.2 % (of both men and women) fear of contaminating the partner either with their disease or because of intracavitary therapies used after endoscopic resection [25].

9.4.3 Colorectal Cancer and Colostomy

The literature chiefly analyzes strictly surgery-linked consequences regarding genitourinary functioning in both men and women [42]. But the crucial influence on the quality of patients' life after surgery is colostomy, which is the most evident and incapacitating aftermath they have to deal with. Patients, in our case women, must be instructed and assisted in managing the doubtlessly undesirable effects of external derivation by using simple strategies to neutralize or minimize smells, gas, and diarrhea – for example, by planning sexual intercourse at a time when the stoma is empty [43, 44].

9.5 Radiotherapy and Female Sexual Dysfunction

Radiotherapy plays an important role in the treatment of rectal, anal, bladder, cervix, and vulvar carcinomas, as well as in breast cancers. It can be administered as primary, neoadjuvant, or adjuvant treatment. Damage occurs to the organ treated, the vessels, and the nerve plexus. What is more, the fibrosis that follows treatment can cause intestinal and ureteral stenosis, pelvic and lower limb lymphedema, endothelial damage, inflammation, ulcers, ischemia, and necrosis with the formation of fistulae that are not always easy to cure [45].

Where there has been massive irradiation of the female pelvis, as, for example, in carcinomas of the cervix, the ovaries undergo a temporary or permanent block of their functional capacity. In premenopausal age this determines the sudden appearance of hot flashes and vaginal dryness. In younger women who have undergone less drastic radiation therapy, as, for example, in the course of treatment for Hodgkin's disease, this condition can be temporary. The woman can become fertile again thanks to the survival of primary follicles that show modest or no proliferation of the granulated cells at the time of radiotherapy.

It is evident, therefore, that below the threshold of 50 years, careful counseling is strongly advised in order to help the patient decide whether or not to interrupt any method of contraception currently being used, knowing that sterility frequently follows this treatment.

Reduced ovarian reserve is also found in survivors of radiation or chemotherapy undergone in childhood. This determines a shortening of the reproductive life span and the onset of early menopause [46].

The damage caused by radiation therapy is progressive and can become symptomatic after a period of latency. The total dosage of radiation administered has a harmful effect on the intestine, the bladder, and the genital organs. In women, bleeding and reduced elasticity of the vaginal walls and the vulva are often reported. These become thin and fragile, and there can be pain and dryness. Due to subsequent scarring, the vagina can become narrower and shorter. This can lead to fear of having sexual relations, with psychological repercussions [47, 48].

The use of vaginal estrogens can help to lessen the effects of radiation therapy by facilitating epithelial regeneration. The regular use of vaginal dilators and fairly frequent sexual relations can also help counter undesirable aftermaths. It should be kept in mind that maintaining adequate vaginal capacity is also important to facilitate oncologic-gynecologic follow-up. However, the use of dilators can be felt as intrusive and can create considerable emotional and relational problems. Medical personnel must be able to deal with these problems positively, giving support to the patient not only in learning how to manage this technique but also in case of possible psychological effects [49].

Up to 28 % of patients report a persistent reduction in lubrication 2 years after treatment [50]. The use of local vaginal estrogens can lessen irritation, facilitating epithelial regeneration.

Bladder functioning can also be affected by radiation therapy, causing frequency, urgency, or vesical instability [51].

Radiation also represents an important component of the therapy used for colon/rectal tumors. A high prevalence of sexual dysfunctions is reported in the literature even when care has been taken to spare nervous structures during surgery [52].

The percentage of sexually active women, over 51 % before surgery, falls to 32.5 % in the 3 months following it and to only 18.4 % after 2 years. In an interesting longitudinal analysis, preoperative radiotherapy represented the only significant risk factor [53].

9.6 Chemotherapy and Female Sexual Dysfunction

As well as the abovementioned symptoms related to the onset of early menopause, numerous antiblastic agents have an irritating effect on the mucous membranes. In particular, the vaginal lining is subject to thinning, dryness, and susceptibility to microtraumas. Weakening of the immune system can determine the risk of infections, including sexually transmitted ones, and a flare-up of genital herpes or genital warts. The general tiredness and nausea that characterize these therapies influence mood and sexual desire. Women are uncomfortable with their body image, feeling themselves "unattractive" to their partner. The perception of their bodies as mutilated contributes to this, as do loss of hair (especially eyebrows and pubic hair), weight loss, or vice versa weight gain due to corticosteroid-based therapy, lessening of the muscle mass, and the presence of infusion catheters or ostomy appliances.

Conclusions

Thanks to the use of multimodal treatments, early diagnosis, and new therapeutic frontiers, patients with a past history of cancer can live long lives "alongside" their disease. Thus, there is a need for constant attention in the treatment of "collateral damage" which, though linked to a positive therapeutic effect, can permanently compromise survivors' life quality. It is no longer enough to deal with the issue of sexual dysfunctions focusing merely on anatomical damage to organs and functions. The mechanisms implicit in sexuality, in health as in sickness, are complicated and often hard to define – they involve psychological, relational, biological, cultural, ethnic, and religious spheres [54]. Thus, in addition to the dramatic bodily changes that are a veritable *representation* of the disease, such as scars and amputations, there are also negative experiences of fear, pain, and fatigue that influence interpersonal relations between couples and with family members and colleagues at work. For this reason, along with strictly medical interventions (gynecological, urological, endocrinological, and rehabilitative), it is necessary to break down the communication barriers that close the cancer patient within her solitude and to speak concretely and reassuringly so as to direct her to parallel therapeutic paths of support and counseling aimed specifically to help her regain a satisfying sexual life.

References

1. World cancer research Fund International. http://wcrf.org/int/cancer-facts-figures/data-specific-cancers/bladder-cancer-statistics. 2012.
2. Purtilo DT, Sullivan JL. Immunological basis for superior survival of females. Am J Dis Child. 1979;133:1251–3.
3. Siegel R, Naishadham D, Jemal A. Cancer statistics, 2013. CA Cancer J Clin. 2013;63:11–30.
4. Howlader N, Noone AM, Krapcho M, Neyman N, Aminou R, Altekruse SF, Kosary CL, Ruhl J, Tatalovich Z, Cho H, Mariotto A, Eisner MP, Lewis DR, Chen HS, Feuer EJ, Cronin KA, editors. SEER cancer statistics review, 1975–2009 (vintage 2009 populations). Bethesda: National Cancer Institute; 2009.
5. AIRTUM Working Group. I Tumori in Italia: rapporto 2010. La prevalenza dei tumori in Italia: persone che convivono con un tumore, lungosopravviventi e guariti. Epidemiol Prev. 2010;34(5–6):Suppl 2.
6. Surveillance, Epidemiology, and End Results (SEER) Program. Limited-use data (1973–2006), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2009, based on the November 2007 submission.
7. Stabile C, Barakat R, Abu-Rustum N, Dickler M, Goldfarb S, Basch E, Carter J. Preliminary data – a survey of female cancer patients' Preference for sexual health intervention. Podium presentation at the International Society for the Study of Women's Sexual Health Meeting; Scottsdale, Feb 2011.
8. Wiggins DL, Wood R, Granai CO, Dizon DS. Sex, intimacy, and the gynecologic oncologists: survey results of the New England Association of Gynecologic Oncologists (NEAGO). J Psychosoc Oncol. 2007;25:61–70.
9. White ID, Allan H, Faithfull S. Assessment of treatment-induced female sexual morbidity in oncology: is this a part of routine medical follow-up after radical pelvic radiotherapy? Br J Cancer. 2011;105:903–10.
10. Ekwall E, Ternestedt BM, Sorbe B. Important aspects of health care for women with gynecologic cancer. Oncol Nurs Forum. 2003;30:313–19.
11. Shifren JL, Monz BU, Russo PA, Segreti A, Johannes CB. Sexual problems and distress in United States women: prevalence and correlates. Obstet Gynecol. 2008;112:970–8.
12. Ende J, Rockwell S, Glasgow M. The sexual history in general medical practice. Arch Intern Med. 1984;144:558–61.
13. Goldfarb SB, Abramsohn E, Andersen BL, Baron SR, Carter J, Dickler M, Florendo J, Freeman L, Githens K, Kushner D, Makelarski JA, Yamada D, Lindau S. A national network to advance the field of cancer and female sexuality. J Sex Med. 2013;10(2):319–25.
14. Thirlaway K, Fallowfield L, Cuzick J. The sexual activity questionnaire: a measure of women's sexual functioning. Qual Life Res. 1997;6(6):606.
15. Fobair P, O'Hanlan K, Koopman C, Classen C, Dimiceli S, Drooker N, Warner D, Davids H, Loulan J, Wallsten D, Goffinet D, Morrow G, Spiegel D. Comparison of lesbian and heterosexual women's response to newly diagnosed breast cancer. Psychooncology. 2001;10(1):40–51.
16. Baser RE, Li Y, Carter J. Psychometric validation of the Female Sexual Function Index (FSFI) in cancer survivors. Cancer. 2012;118(18):4606–18. doi:10.1002/cncr.26739. Epub 2012 Feb 22.
17. Booth BB, Rasmussen A, Jensen JB. Evaluating sexual function in women after radical cystectomy as treatment for bladder cancer. Scand J Urol. 2015;19:1–5.
18. Goldstein I, Fisher WA, Sand M, Rosen RC, Mollen M, Brock G, et al. Womens sexual function improves when partners are administered vardenafil for erectile dysfunction: a prospective, randomized, double-blind, placebo-controlled trial. J Sex Med. 2005;2:819–32.
19. Pitceathly C, Maguire P. The psychological impact of cancer on patients partners and other key relatives: a review. Eur J Cancer. 2003;39:1517–24.

20. Hordern AJ, Street AF. Communicating about patient sexuality and intimacy after cancer: mismatched expectations and unmet needs. Med J Aust. 2007;186(5):224–7. 12. Perz J, Ussher J M, Gilbert E, and the Australian Cancer and Sexuality study team.Feeling well and talking about sex : psycho-social predictors of sexual functioning after cancer. BMC Cancer. 2014;14:228.
21. Bal MD, Yilmaz SD, Beji ES. Sexual health in patients with gynecological cancer: a qualitative study. Sex Disabil. 2013;31(1):83–92.
22. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington, DC: American Psychiatric Association; 2013.
23. Moreno KF, Khabbaz E, Gaitonde K, Meinzen-Derr J, Wilson KM, Patil YJ. Sexuality after treatment of head and neck cancer: findings based on modification of sexual adjustment questionnaire. Laryngoscope. 2012;122(7):1526–31.
24. Panjari M, Bell S, McMurrick PJ, Davis SR. Sexual function, incontinence, and well being in women after rectal cancer. Support Care Cancer. 2013;21(3):2749–58.
25. Kowalkowski MA, Chandrashekar A, Amiel GE, Lerner SP, Wittmann DA, Latini DM, Goltz HH. Examining sexual dysfunction in non –muscle-invasive bladder cancer: results of cross-sectional mixed methods research. Sex Med. 2014;2(3):141–51.
26. Jarden M, Schjodt I, Thygesen KH. The impact of hematopoietic stem cell transplantation on sexuality: a systemic review of the literature. Bone Marrow Transplant. 2012;47:716.
27. Shell JA, Carolan M, Zhan Y, Meneses KD. The longitudinal effects of cancer treatment on sexuality in individuals with lung cancer. Oncol Nurs Forum. 2008;35(1):73–9.
28. Horner MJ, Ries LAG, Krapcho M, Neyman N, Aminou R, Howlader N, et al. SEER cancer statistics review, 1975–2006. Bethesda: National Cancer Institute; 2009.
29. Markopoulos C, Tsaroucha AK, Kouskos E, Mantas D, Antonopoulou Z, Karvelis S. Impact of breast cancer surgery on the self-esteem and sexual life of female patients. J Int Med Res. 2009;37(1):182–8.
30. Trudeau M, Charbonneau F, Gelmon K, et al. Selection of adjuvant chemotherapy for treatment of node-positive breast cancer. Lancet Oncol. 2005;6(11):886–98.
31. Citron ML, Berry DA, Cirrincione C, et al. Randomized trial of dose-dense versus conventionally scheduled and sequential versus concurrent combination chemotherapy as postoperative adjuvant treatment of node-positive primary breast cancer: first report of Intergroup Trial C9741/ Cancer and Leukemia Group B Trial 9741. J Clin Oncol. 2003;21(8):1431–9.
32. Brinton LA, Sherman ME, Carreon JD, Anderson WF. Recent trends in breast cancer among younger women in the United States. J Natl Cancer Inst. 2008;100(22):1643–8.
33. Trinkaus M, Chin S, Wolfman W, Simmons C, Clemons M. Should urogenital atrophy in breast cancer survivors be treated with topical estrogens? Oncologist. 2008;13(3):222–31.
34. Mourits MJ, Bockermann I, de Vries EG, et al. Tamoxifen effects on subjective and psychosexual well-being, in a randomised breast cancer study comparing high-dose and standard-dose chemotherapy. Br J Cancer. 2002;86(10):1546–50.
35. Bodurka D, Sun C. Sexual function after gynecologic cancer. Obstet Gynecol Clin North Am. 2006;33:621–30. Audette C, Waterman J. The sexual health of women after gynecological malignancy.J Midwifery Womens Health. 2010;55:357–62.
36. Abbott-Anderson K, Kwekkebon K. A systematic review of sexual concerns by gynecological cancers survivors. Gynecol Oncol. 2012;124:477–89. doi:10.1016/j.ygyno.2011.11.030. Epub 2011 Nov 28. Review. Erratum in: Gynecol Oncol. 2012 Sep;126(3):501-8.
37. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2015. CA Cancer J Clin. 2015;65(1):5–29.
38. Allareddy V, Kennedy J, West MM, Konety BR. Quality of life in long-term survivors of bladder cancer. Cancer. 2006;106(11):2355–62.
39. Lee RK, Abol-Enein H, Artibani W, Bochner B, Dalbagni G, Daneshmand S, Fradet Y, Hautmann RE, Lee CT, Lerner SP, Pycha A, Sievert KD, Stenzl A, Thalmann G, Shariat SF. Urinary diversion after radical cystectomy for bladder cancer: options, patient selection, and outcomes. BJU Int. 2014;113(1):11–23.
40. Wolff I, Brookman-May S, May M. Sex difference in presentation and outcomes of bladder cancer: biological reality or statistical fluke? Curr Opin Urol. 2015;25(5):418–26.

41. Koya MP, Simon MA, Soloway MS. Complications of intravesical therapy for urothelial cancer of the bladder. J Urol. 2006;175(6):2004–10.
42. Traa MJ, De Vries J, Roukema JA, Den Oudsten BL. Sexual (dys) function and the quality of sexual life in patients with colorectal cancer: a systematic review. Ann Oncol. 2012;23(1): 19–27.
43. Altschuler A, Ramirez M, Grant M, Wendel C, Hornbrook MC, Herrinton L, Krouse RS. The influence of husbands' or male partners' support on women's psychosocial adjustment to having an ostomy resulting from colorectal cancer. J Wound Ostomy Continence Nurs. 2009;36(3): 299–305.
44. Ayaz S, Kubilay G. Effectiveness of the PLISSIT model for solving the sexual problems of patients with stoma. J Clin Nurs. 2009;18(1):89–98.
45. Incrocci L, Jensen PT. Pelvic radiotherapy and sexual function in men and women. J Sex Med. 2013;10 suppl 1:53–64.
46. Larsen EC, Muller J, Schmiegelow K, Rechnitzer C, Andersen AN. Reduced ovarian function in long-term survivors of radiation and chemotherapy- treated childhood cancer. J Clin Endocrinol Metab. 2003;88:5307–14.
47. Frumovitz M, Sun CC, Schover LR, Munsell MF, Jhinggran A, Wharton JT, Eifel P, Bevers TB, Levenback CF, Gershenson DM, Bodurka DC. Quality of life and sexual function cervical cancer survivors. J Clin Oncol. 2005;23:7428–36.
48. Greimel ER, Winter R, Kapp KS, Haas J. Quality of life and sexual functioning after cervical cancer treatment: a long term follow up study. Psychooncology. 2009;18:476–82.
49. Cullen K, Fergus K, Dasgupta T, Fitch M, Doyle C, Adams L. From "sex\toy" to intrusive imposition: a qualitative examination of women's experiences with vaginal dilator use following treatment for gynecological cancer. J Sex Med. 2012;9:1162–73.
50. Jensen P, Groenvold M, Klee M, Thranov I, Petersen M, Machin D. Longitudinal study of sexual function and vaginal changes after radiotherapy for cervical cancer. Int J Radiat Oncol Biol Phys. 2003;56:937–49.
51. Little FA, Howard GS. Sexual function following radical radiotherapy for bladder cancer. Radiother Oncol. 1998;49:157–61.
52. Hendren SK, O'Conner BI, Liu M, Asano T, Cohen Z, Swallow CJ, Macrae HM, Gryfe R, Mc Leod RS. Prevalence of male and female sexual dysfunction is high following surgery for rectal cancer. Ann Surg. 2005;242:212–23.
53. Lange MM, Marijnen CA, Maas CP, Putter H, Rutten HJ, Stiggelbout AM, Meershoek-Klein KE, van de Velde CJ. Risk factors for sexual dysfunction after rectal cancer treatment. Eur J Cancer. 2009;45:1578–88.
54. Bober SL, Varela VS. Sexuality in adult cancer survivors: challenges and intervention. J Clin Oncol. 2012;30(30):3712–9.

Sexuality in Couples with Reproductive Difficulties

10

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Abbreviations

ART	Assisted reproductive techniques
ED	Erectile dysfunction
HSD	Hypoactive sexual desire
ICSI	Intracytoplasmic injection
IUI	Intrauterine insemination
PE	Premature ejaculation
QOL	Quality of life
SD	Sexual dysfunction

10.1 General Aspects of Conjugal Infertility

Infertility is defined as the inability to conceive after 12 months of unprotected regular sexual intercourse [1]. It is a highly prevalent condition worldwide, being present in 3.5–16.7 % of couples in developed countries and 6.9–9.3 % in less-developed countries [2]. The probability of a woman becoming pregnant after 1, 3, 6, and 12 menstrual cycles has been estimated to be 38 %, 68 %, 81 %, and 92 %, respectively [3]. However, a woman's fertility is affected by several environmental, psychological, and biological factors and decreases with advancing age. After age 35 years, a woman's fertility gradually decreases until age 40 years, decreasing markedly thereafter and ending at menopause [4]. This progressive loss of fertility is also

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_10

observed in women who undergo intracytoplasmic injection (ICSI), with delivery rates decreasing significantly with increased age [5].

The limited period of fertility for women stands in contrast to the long fertility period for men. Under normal conditions, male reproductive capacity remains unchanged throughout life [6]. This difference in potential fertility may cause an emotional imbalance, primarily in infertile woman. Indeed, infertile women are more predisposed than infertile men to infertility-associated emotional and mental disorders such as symptoms of depression. Thus, the window of opportunity for women to become pregnant is limited, making it stressful for a couple, especially for the woman, even when fertile. Thus, when couples are diagnosed with infertility, they are exposed to a high risk of emotional and mental injuries that may impact their well-being. Studies have shown that infertile couples often report low self-esteem, impairment of partnership quality, difficulties with sexuality, and loss of spontaneous sexuality during the experience of infertility with negative impacts on their QOL [7]. In addition, infertility may contribute to age-related impairments in sexuality and erectile dysfunction (ED) among men [8].

10.2 Medical Approaches to Infertility

The diagnosis of infertile couples starts with a detailed history of both partners to identify the most likely causes of infertility. In women, tubal obstruction is a cause of infertility [9, 10]. Also infertility may be due to endocrine disorders that can cause menstrual cycle problems, including polycystic ovary syndrome, thyroidopathy, and hyperprolactinemia [11, 12] that are associated with hypoactive sexual desire (HSD) and ED in men, and HSD in women [13, 14]. Sexual dysfunction may lead to reduced sexual frequency, which may, in turn, contribute to subfertility.

Assisted reproductive techniques (ART) are recommended to treat infertile couples. Intrauterine insemination (IUI) involves the introduction of spermatozoa into the uterine cavity; in vitro fertilization (IVF) involves the fertilization of gametes in the laboratory; and ICSI consists on the injection of one spermatozoon into a female gamete. Ovarian hyperstimulation is required prior to IVF and ICSI, with hormones used to promote follicular development, followed by ultrasound-guided transvaginal aspiration of the ovaries under sedation to collect follicle. ART also includes gamete (oocyte or sperm) donation and uterus donation (surrogate motherhood) [15]. Gamete donation is indicated when one or both partners lack gametes or have a genetic condition with a high likelihood of being transmitted to their descendants. These individuals are at high risk of mental, emotional, sexual, and physical comorbidities and low QOL [16].

ART involves a series of complex procedures, which may negatively impact sexual function, mainly among couples that require more complex treatments such as IVF and ICSI [17]. In evaluating couples seeking treatment for infertility, the treatment period they expect will be required for successful pregnancy may be associated with increased expression of negative emotions such as anxiety and

depression [18, 19]. These symptoms may have a negative effect on pregnancy rates, in as much as depressive symptoms have been associated with lower pregnancy rates in women undergoing IVF treatment [20]. Indeed, psychological intervention for infertile couples positively affects their rates of natural pregnancy (i.e., in the absence of ART) [21].

10.3 Impact of Infertility on Female Sexuality

Female sexuality is complex and can be affected by biological, psychological, emotional, and environmental problems commonly associated with the diagnosis of infertility. Table 10.1 shows some of the sexual difficulties experienced by infertile women.

10.3.1 Emotional and Physical Aspects of Infertile Women

The diagnosis and treatment of infertility are negative psycho-emotional experiences for infertile women [22]. Although infertility may destabilize a couple's relationship, women seem to be more affected than men and have a greater tendency to classify the marital relationship as bad when the couple fails to conceive [23]. Most studies have shown that women of infertile couples are more likely to have difficulties with adjustment and lower QOL scores than their male partners [23].

The impact of infertility on a woman's well-being depends on the cause of infertility. Women in couples with an unknown cause of infertility seem to be more

Table 10.1 Highlights of mental and sexual health aspects of infertile women and men

Women	Men
Infertile women have a high risk for depression and anxiety related to: A diagnosis of infertility The duration and cause of infertility Lower self-esteem, fear of impossibility of having a baby Their partner's infertility Social and family pressure for the couple to have a child Failure of ART	Infertile men are at high risk for depression and anxiety related to: Reduced self-esteem Reduced self-confidence Tendency toward social isolation Failure of ART Lower educational level Poor marital relationship Duration of infertility Being the cause of infertility
Infertile women have a high risk for sexual dysfunction such as: Sexual desire/arousal and orgasmic disorder Reduced sexual satisfaction Reduced sexual frequency	Infertile men are at high risk for sexual dysfunctions such as: Hypoactive sexual disorder Erectile dysfunction Reduced sexual frequency Low sexual satisfaction

ART assisted reproductive technique

adaptive, cope better, and have more functional defensive systems than women in couples with a known cause of infertility [24]. Reinforcing this evidence, a study of 564 couples with idiopathic infertility found that the dimensions of life satisfaction and marital relationship, as well as physical and psychological complaints, were similar to those of couples with other medical diagnoses of infertility [25]. The psychological profiles of infertile women clearly differ from those of women who do not have problems conceiving. In comparative studies infertile and fertile women had different personality dynamics. For example, infertile women may experience grief and depression before, during, and after treatment of infertility [26]. They may have a distorted self-image, be more likely to have difficulties with interpersonal relationships due to higher discomfort with closeness, and be more likely to show defense mechanisms such as somatization than fertile women [27]. The repercussions of infertility for women were shown in a qualitative analysis of infertile couples, which identified many problems, including poor body image, impaired sexual relationships, and reduced sexual pleasure during infertility treatment [28]. In addition, case-controlled studies showed that infertile women were less satisfied with their lives as a whole, as they rated life as less interesting, less rewarding, emptier, and more lonely, making them less content than women with children [29].

Psychopathologic factors associated with infertility can exacerbate this condition and cause psychological distress for women. The prevalence of depression is higher in women with a 2- to 3-year history of infertility than in women with a < 1-year or > 6-year duration of infertility [30]. Anxiety, depression, fear of pregnancy, and fear of being infertile have been found to be psycho-emotional causes of infertility [27]. These impairments in mental health contribute to a greater incidence of high-risk behavior in infertile women that are more likely to have a higher probability of certain diseases, including benign uterine tumors, intestinal disease, and infectious diseases [31]. These findings indicate that the psycho-emotional problems of women in infertile couples may interfere with their general health and possibly with their fertility rates as previously demonstrated [32].

An evaluation of women seeking treatment for infertility showed that their increased expression of negative emotions correlated with increased depressive symptomatology [20]. The prevalence of depression is about two-fold higher in infertile than in fertile women, with this prevalence increasing with the duration of infertility [30]. In the early stages of infertility, the main stress is related to a physical inferiority complex, later changing to stress about what others outside the family will say; moreover, infertile women become more depressive the longer treatment persists [33]. An evaluation of overall life satisfaction, as well as marital and sexual adjustment, after the completion of three infertility treatments showed that women who became biological mothers were significantly more satisfied with their lives than women who remained childless after treatment [34]. Moreover, the latter reported that infertility had a significantly greater negative impact on their marriages [34].

10.3.2 Sexual Function of Infertile Women

Women may experience sexual dysfunction (SD) due to a diagnosis of infertility, during its treatment and after the failure of treatment, with SD being prevalent in female partners of infertile couples [35]. The factors predictive of SD in this population included the duration of the marriage, previous infertility treatment, and a long duration (≥3 years) of infertility [36]. This finding was corroborated in a case–control study, which showed that scores on all domains of the Female Sexual Function Index (FSFI), including desire, arousal, orgasm, and pain, were reduced as the duration of infertility became longer [37]. The rates of SD in women with primary and secondary infertility were 64.8 % and 76.5 %, respectively, with women with secondary infertility being at a 9.5-fold higher risk of sexual dysfunction than women with primary infertility [38]. HSD and orgasmic dysfunction were the most prevalent sexual complaints among infertile women [23].

The high rates of mental and sexual disorders recorded among infertile women were associated with psychological problems arising from their diagnosis of infertility. One third of women of infertile couples are affected by anxiety and depression [35]. Psychological problems are common in older infertile women because they are more likely to experience treatment failure, resulting in higher risks of depression and anxiety [39], which may compromise their well-being [22]. Moreover, women with an identified cause of infertility had higher depression scores than women with unexplained or undiagnosed infertility [30]. A controlled multicenter study involving 281 patients awaiting treatment for rheumatoid arthritis showed that the prevalence of negative emotions was associated with the length of time trying to become pregnant, positive or negative changes in the dyadic relationship, sexual problems, and higher rates of anxiety and depressed mood than fertile women [22].

The negative impact of depression on sexual function may be associated with desire/arousal disorders, anorgasmia, and less pleasure during sexual relations [40].

10.4 Impact of Infertility on Male Sexuality

Marital infertility also has a negative impact on male sexuality, with some of these effects listed in Table 10.1.

10.4.1 Emotional and Physical Aspects of Infertile Men

Infertility may represent a source of stress for men, who may be emotionally affected and show reduced self-esteem [41], reduced self-confidence, and low sexual satisfaction. These symptoms may negatively affect their marital relationships. This emotional impact of infertility on men is clearly correlated with their level of education and the duration of infertility [42]. In addition, educational level, the desire to have

children, a poor marital relationship, previous IVF attempts, and duration of infertility were predictors of lower mental health scores in infertile men [43].

Men experience treatment-related distress associated with ICSI, primarily those requiring epididymal or testicular puncture to retrieve spermatozoa. These men feel greater responsibility for the couple's infertility and are concerned about the possibility of treatment failure, particularly about the possible negative impact of childlessness for the couple [44].

Men who discover that they are the cause of infertility may experience low self-esteem [41] and be at high risk for anxiety and depression, with younger infertile men more likely to suffer the negative emotional consequences of being infertile [45]. This suggests that the ability of men to reproduce has a strong impact on their self-concept of value.

10.4.2 Sexual Function of Infertile Men

In men, a diagnosis of infertility is associated with clinically significant sexual problems, personal and social distress, and decreased sexual function [46]. In fact, male factor infertility is independently associated with sexual problems and poorer QOL [47]. Infertile men have more problems with ED, premature ejaculation (PE), and hypoactive sexual desire (HSD), in addition to having less sexual satisfaction than fertile controls [23]. A review of the literature showed that the rates of ED varied from 2 to 30 % and that the rate of HSD was as high as 52 %, in infertile men [48]. Qualitative analysis of a small sample of infertile couples indicated that 63 % of men experienced a period of ED after being diagnosed with azoospermia [49].

Sexual dysfunction in men may represent a factor that discourages their involvement in sexual activity. An analysis of 1,298 infertile men aged 32–39 years, with two years duration of infertility, showed that the average frequency of sexual intercourse was seven times per month, with 24 % of these couples having fewer than four sexual contacts per month. Factors affecting coital frequency included age and the occurrence of ED [50]. A comparative study of 1,468 infertile men showed higher rates of PE and ED than in men with preserved fertility. Mental health was particularly affected in this sample, which demonstrated a higher prevalence of anxiety and depression than fertile men. Depressive and anxiety symptoms are factors that predict sexual dysfunction [51], with ED in particular associated with depressive symptoms [52].

Duration of infertility and educational level may predict sexual function of men in infertile couples. For example, sexual satisfaction scores were significantly higher in those with <3 years than >3 years of infertility and in those with higher than lower levels of education [42]. In contrast, ED was observed in 50.61 % of Chinese infertile men and was more common in infertile men with education levels above high school. These infertile men had a higher prevalence of ED than fertile partners of infertile women [53], indicating that infertility is a predictor of ED. This greater predisposition of infertile men for ED may be due to psychological changes resulting from anxiety about sexual performance and a fear of sexual intercourse due to the need for

an erection. The possibility of losing an erection may provoke in men a fear of intercourse and a predisposition to ED [54]. PE affects about 50% of infertile men, as well as being a source of frustration for both partners [55]. A study of a large Japanese population showed that the rates of PE and ED were significantly higher in infertile than in fertile men, with the risk of PE directly associated with high levels of mental disorders (anxiety and depression) frequently observed in infertile men [51].

10.5 Psycho-emotional Aspects of Infertile Couples

The assisted reproduction process can be long, expensive, and uncertain and may cause deep emotional changes in couples, particularly when the final outcome of the procedure is unfavorable. This may have a negative impact on sexual function and a couple's QOL [56]. Infertility has deep psychological implications for both partners and may interfere with interpersonal relationships [57] and may lead to reduced self-esteem in infertile couples and may reduce a couple's close relationship [58], leading to marital adjustment problems and feelings of anger and a desire for separation, the latter of which is more common among female partners of infertile men [49]. However, there is evidence that a couple's infertility brings them closer together, facilitating the sharing of problems arising from the difficulty of conceiving and strengthening the dyadic relationship [59].

Differences in the impact of infertility on infertile couples may be due to the sociocultural differences of studied populations. For example, in some societies, one partner's emotions are influenced by the other's, with men being more capable of coping with infertility than women as they are more optimistic. Living in a rural area and lower educational level are factors associated with a high risk of psychological problems in infertile couples [60]. Cultural aspects favor an increase in the risk of adjustment problems in infertile couples. The psychosocial consequences for infertile couples are especially severe in poor countries, in which gender is the greatest determinant of types of psychosocial consequences of infertility. Infertile women in these countries are at high risk of domestic violence, union dissolution, and sexual dysfunction [61]. In Ghana, for example, childless couples are socially stigmatized and excluded from leadership roles in their communities. However, the social consequences of infertility are severe; this is particularly the case for women, who are more exposed to violence and are more likely to have high rates of risky sexual behavior by engaging in sex with multiple partners [62], resulting in a high risk of sexual transmitted diseases. In countries where the concepts of masculinity and femininity are centered on reproductive capacity, the psychological consequences of infertility are particularly disastrous [63]. In certain societies the fear of stigma prevents couples from seeking treatment. In Indian couples, for example, the number of years of infertility and sexual dysfunction are never revealed to others. If the husband was sexually dysfunctional, the couple chose to label the situation as "infertility" in order to avoid the stigma [64]. In societies where women have a "mission" to bear children, physical and emotional violence against women are common, especially among couples with low socioeconomic and cultural status [63].

Infertile couples may cope better with their infertility and show better adjustment if they share their experiences of stress during infertility treatment. This may improve their marital relationship and increase dyadic cohesion. When both partners perceive equal levels of social stress associated with infertility and a similar need for parenthood, they report high levels of marital adjustment and marital satisfaction [65].

The stereotypical structure of the family, based on the components father–mother–child, indicates that one of the couple's main purposes is to have a child, thus creating a family. Thus, infertile couples experience constant family [66] and societal [23] pressures to conceive, which may contribute to their social isolation. Infertility can also contribute to a fear of loss of social status and loss of continuity of the family line [66], which may increase the negative impact of infertility on QOL. Also, couples may face questions about which spouse is responsible for infertility, thus contributing to a couple's desire for isolation [67]. This isolation may be provoked by feelings of shame associated with the impossibility of having a child, as well as by anxiety and depression [68].

When male infertility is the cause, sexual dissatisfaction for both partners and relational instability tend to be more pronounced [58]. However regardless of the cause of infertility, women contribute more to marital discord than men, perhaps because women are more susceptible to emotional imbalances following a diagnosis of infertility. Although the diagnosis itself can have profound implications for the sexual function of the couple, the technical procedures involved in ART are quite stressful for the couple. For example, the programming of intercourse may affect its spontaneity and can lead to sexual dysfunction [56]. Also, marital duration, time in treatment, and number of ART procedures were positively related to the stress level of wives, whereas only number of IVF procedures was positively related to the stress level of husbands [69].

The high incidence of anxiety and depression documented in infertile couples [68, 70, 71] may contribute to their sexual difficulties. The impaired emotional states of infertile couples have been associated with difficulties in marital relationships. For example, a study comparing 48 couples who achieved pregnancy with ART and 117 couples who were unsuccessful showed that the level of emotional stress was higher and marital adjustment lower in the latter group [72].

A longitudinal study evaluating the quality of the marital relationship of infertile couples found that complaints of relationship conflicts remained even after the resolution of infertility [73], thus emphasizing the complexity of the psychic and emotional changes associated with infertility. Depression was the most representative emotional change, primarily in women. Depressed women have a poorer QOL than depressed men, and depressive symptoms in women are predictive of poor QOL of their spouses [74]. However, there is evidence that QOL is equally affected in both partners of couples diagnosed with infertility [75].

10.5.1 Sexual Function of Infertile Couples

Several studies have sought to understand the process of sexual dysfunction within the relational dynamics of infertile couples. The main difficulty in assessing the real

impact of infertility on sexual function was that, despite considerable evidence of relational and sexual changes in these couples, no cohort study has assessed the quality of the relationship and sexual life prior to the diagnosis of infertility. A review of the literature showed that infertility and its treatment can lead to changes in sexual self-esteem, sexual relationship, and sexual function [17]. The results of a systematic review showed that infertile couples may have a higher incidence of sexual dysfunction, including reduced sexual frequency and a higher incidence of HSD, which mainly affect men when they are diagnosed as the cause of infertility and women after ART treatment failure [76]. However, the effects of infertility on the sexuality of infertile couples remain unclear.

Quality of sexual life seems to be poorer in infertile than in fertile couples. This may be due to psychosocial aspects of infertility and psychosexual problems that are more common, especially among couples who have not had success with ART [50, 64]. The social stigma attached to infertility may have a negative impact on a couple's self-esteem [77]. During treatment, strategies such as programmed coitus and the requirement for a certain number of coital encounters to reproduce may result in loss of sexual spontaneity [7] and a consequent loss of sexual interest. The incidence of HSD among infertile couples was reported to be 41.5 %, with 52.5 % of these couples reporting reduced sexual satisfaction, especially couples with low educational and marital relationship problems [78]. This sexual dysfunction can result in a reduction in sexual activity over time, without the couple becoming pregnant [64].

There are conflicting data regarding the actual contribution of each partner to impaired sexual function in infertile couples. High rates of ED and lower satisfaction with intercourse in infertile men [23], and reduced marital and sexual satisfaction expressed by women when the male or both partners were infertile [79], can contribute to a more fragile sexual relationship. An evaluation of sexual function in infertile couples showed that female sexual function was positively correlated with male partner sexual function [35]. Wives diagnosed with female infertility experienced poorer self-esteem and less acceptance by in-laws than wives of partners diagnosed with male infertility [79]. It is also important to highlight the various sexual and psychological problems, as well as the dysfunctional attitudes, in infertile couples that can have a negative impact on their mental health. High rates of depression and anxiety have been documented in this population, with depressive symptoms found to predict impaired sexual function and satisfaction [80, 81].

10.6 Strategies to Promote the Psychological and Sexual Health of Infertile Couples

Although the implications of infertility for infertile couples are complex, there is a relative lack of qualitative analysis that could assist in elucidating the complexity of this condition. Infertile couples experience a wide range of emotions, from anxiety, depression, and suicidal feelings to improvements in marital relationships [82]. In addition to revolving problems related to guilt, fear, sadness, expectation of success, and the possibility of treatment failure, there is a need to consider the negative

consequences for the dyadic relationship. Therefore, infertile couples should be treated by a multidisciplinary team of health professionals working in an interdisciplinary manner.

10.6.1 Assessment of Sexual Problems in Infertile Couples

Although most couples consider psychosocial care as important, their demand for psychological support is low and inconsistent with their needs. The few couples who express the desire to seek psychological help are those with high infertility-related stresses in the marital, personal, and social domains [83]. Men and women differ in their attitude toward seeking help. More women than men seek psychological support or consult with a psychologist or sex therapist [47]. Despite the low demand for treatment by a mental health professional, the negative impact of infertility on married life suggests that human reproduction services should provide this type of care to infertile couples. Most women seeking treatment for infertility are dissatisfied with the emotional support they receive during the diagnostic process, with more than half expressing a need for professional psychological counseling [84]. Thus, in conjunction with medical care, infertile couples should have more supportive care psychological assistance [85].

During the evaluation of sexual function in infertile couples, it is important to consider any preexisting sexual changes prior to the diagnosis of infertility. Therefore, it is crucial to discuss each partner's previous subjective judgment of the quality of the partnership and the sexual function of the couple.

An ideal evaluation should include comprehensive assessment of sexual function and mental health. Relationship quality should be evaluated using validated questionnaires, such as the Self-Esteem and Relationship Questionnaire (SEAR), which can determine whether infertility has a negative effect on relationship quality and/or sex life [7]. A semi-structured interview may be used to assess personal distress according to individual necessities as there is no typical psychological profile for infertile couples that prevents their use of standardized psychometric rating methods to assess their sexual complaints [25].

A more objective assessment may be provided by a questionnaire that contains questions addressing the personal, relational, and sexual aspects of an infertile couple [47].

Figure 10.1 shows a possible model to promote holistic care for infertile couples.

10.6.2 Interventions for Sexual Problems in Infertile Couples

Interventions for sexual difficulties associated with a diagnosis of infertility should be individualized, since the damage caused by this condition will depend on the psychological condition of each partner and the dynamics of the marital relationship. However, the methodological heterogeneity of studies on this topic [86] makes it difficult to define behaviors for optimal care of these couples. Therefore,

no protocol can likely meet all the demands of a couple in a human reproduction service. However, common to these couples is the negative psychological impact due to both the diagnosis and the treatment of infertility. Infertile couples will therefore require special attention from mental health professionals to assist in addressing the psychological problems related to infertility. Interventional protocols for the treatment of infertility should include assessment of the psychology of the infertile couple (Table. 10.2). Since depression and lowered self-esteem are common to infertility and sexual dysfunction, actions to improve these conditions can also improve sexual function. The European Society of Human Reproduction and Embryology (ESHRE) developed complete clinical practice guidelines to provide clinical assistance to infertile patients [87]. According to these guidelines, infertile couples should be assisted at different levels, before, during, and after the treatment of infertility.

The inclusion of sex therapy may increase the success of interventions designed to improve the QOL of these couples (Table. 10.1). Sexual therapy combined with psychotherapy can improve acute sexual dysfunctions, including HSD, ED, and dyspareunia through specific techniques [88–90] and can prevent chronic sexual dysfunction and relational difficulties between the partners. Psychological interventions aimed at reducing depressive symptoms should be implemented, especially for women with a definitive diagnosis. A meta-analysis examining whether psychological interventions improved mental health and pregnancy rate among infertile patients found that these interventions did not affect depression, anxiety, and mental distress, but did enhance pregnancy rates in couples not receiving ART [21]. Another study showed that psychosocial interventions were more effective in reducing

Table 10.2 Counseling measures for infertile couples

Invite the patients to talk about the infertility experience and their wish for a child
Encourage patients to talk about their motivations for infertility treatment, which allows the professional to determine their knowledge of treatment, expectations, limitations, and rates of success
Discuss the subjective etiologies of the infertility problem to identify feelings of guilt and anger, and to identify the risk of one partner holding the other responsible for the infertility
Encourage couples to discuss emotional and physical stresses; ambivalence; personal, interpersonal, and social conflicts arising from emotions related to infertility that can impair self-confidence and well-being, as well as partner intimacy
Inform patients in detail on the diagnosis, treatment, and perspectives on the rates of successful and unsuccessful treatment and stimulate patients to actively join in decisions regarding their infertility problems
Encourage patients to discuss the impact of their infertility on their social relationships
Stimulate patients to participate in avoiding social isolation
Refer infertile couples to psychotherapy
Open discussion of religious coping strategies rooted in patients' religious teachings [94]

Modified Van den Broeck et al. model [92]

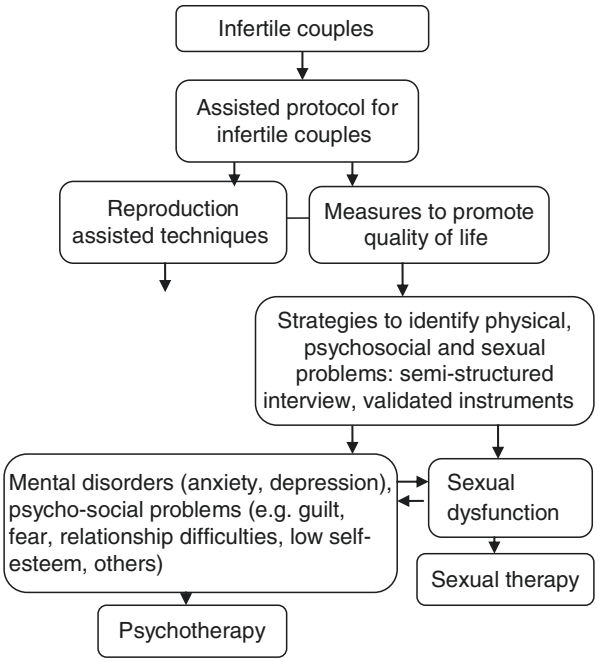

Fig. 10.1 Flowchart of assistance to infertile couples

negative affect than in changing marital and social functioning, but had no effect on pregnancy rates. Moreover, group interventions that emphasized education and relaxation training were significantly more effective in producing positive changes across a range of outcomes than counseling interventions that emphasized emotional expression and support and/or discussion about thoughts and feelings related to infertility [91].

Recognizing that infertility may increase the risk of mood symptoms, physicians should prescribe antidepressants based on the individual needs of each patient. This practice may help couples with symptoms of anxiety and depression. Physicians should also know the characteristics of each drug, including their adverse effects, to make a better choice for individual patients [94].

Conclusion

Assisted reproduction techniques may cause distress, as they involve the use of hormones, intercourse programming, and transvaginal access for ovarian puncture, among others. These techniques may induce tension and anxiety. In addition, social and family attitudes toward infertility can be exhausting for couples who feel pressured to conceive. Moreover, unsuccessful treatment may impair mental health. Additionally, marital relationship problems are common in infertile couples, and many opt for dissolution of marriage. Women tend to perceive their marital relationship as poor, whereas men are more likely to have

sexual dysfunctions such as ED, PE, and less satisfaction with their sexual life. It is necessary to consider the relationship of the couple within a family and social context.

The approach for infertile couples should be interdisciplinary. Better interventions should use assisted reproduction techniques that address the emotional aspects of the couple and the dyadic relationship. Mental health professionals should be provided to treat infertility-related emotional distress, and sex therapy should be offered to provide couples with coping strategies at the start of treatment.

References

1. Zegers-Hochschild F, et al. The International Committee for Monitoring Assisted Reproductive Technology (ICMART) and the World Health Organization (WHO) Revised Glossary on ART Terminology, 2009. Hum Reprod. 2009;24(11):2683–7.
2. Boivin J, et al. International estimates of infertility prevalence and treatment-seeking: potential need and demand for infertility medical care. Hum Reprod. 2007;22(6):1506–12.
3. Gnoth C, et al. Time to pregnancy: results of the German prospective study and impact on the management of infertility. Hum Reprod. 2003;18(9):1959–66.
4. Baird DT, et al. Fertility and ageing. Hum Reprod Update. 2005;11(3):261–76.
5. Osmanagaoglu K, et al. Cumulative delivery rates after intracytoplasmic sperm injection: 5 year follow-up of 498 patients. Hum Reprod. 1999;14(10):2651–5.
6. Nijs M, et al. Correlation between male age, WHO sperm parameters, DNA fragmentation, chromatin packaging and outcome in assisted reproduction technology. Andrologia. 2011;43(3):174–9.
7. Wischmann T, et al. Sexuality, self-esteem and partnership quality in infertile women and men. Geburtshilfe Frauenheilkd. 2014;74(8):759–63.
8. Pal L, Santoro N. Age-related decline in fertility. Endocrinol Metab Clin North Am. 2003;32(3):669–88.
9. Dun EC, Nezhat CH. Tubal factor infertility: diagnosis and management in the era of assisted reproductive technology. Obstet Gynecol Clin North Am. 2012;39(4):551–66.
10. Muzii L, et al. Tubo-peritoneal factor of infertility: diagnosis and treatment. Clin Ter. 2010;161(1):77–85.
11. Sonigo C, Young J, Binart N. [Hyperprolactinemia and infertility: a new physiopathological approach]. Med Sci (Paris). 2013;29(3):242–4.
12. Speer G. The impact of thyroid function in women of reproductive age: infertility, pregnancy and the postpartum period. Orv Hetil. 2013;154(51):2017–23.
13. Atis G, et al. Sexual dysfunction in women with clinical hypothyroidism and subclinical hypothyroidism. J Sex Med. 2010;7(7):2583–90.
14. Kadioglu P, et al. Sexual dysfunction in women with hyperprolactinemia: a pilot study report. J Urol. 2005;174(5):1921–5.
15. Belaisch-Allart J. Assisted reproductive technologies and ethics. Rev Prat. 2014;64(1):106–7.
16. Carter J, et al. A cross-sectional cohort study of infertile women awaiting oocyte donation: the emotional, sexual, and quality-of-life impact. Fertil Steril. 2011;95(2):711–6 e1.
17. Tao P, Coates R, Maycock B. The impact of infertility on sexuality: a literature review. Australas Med J. 2011;4(11):620–7.
18. Klemetti R, et al. Infertility, mental disorders and well-being – a nationwide survey. Acta Obstet Gynecol Scand. 2010;89(5):677–82.
19. Jin X, et al. Survey of the situation of infertile women seeking in vitro fertilization treatment in China. Biomed Res Int. 2013;2013:179098.

20. Demyttenaere K, et al. Coping style and depression level influence outcome in in vitro fertilization. Fertil Steril. 1998;69(6):1026–33.
21. Hammerli K, Znoj H, Barth J. The efficacy of psychological interventions for infertile patients: a meta-analysis examining mental health and pregnancy rate. Hum Reprod Update. 2009;15(3):279–95.
22. Oddens BJ, den Tonkelaar I, Nieuwenhuyse H. Psychosocial experiences in women facing fertility problems – a comparative survey. Hum Reprod. 1999;14(1):255–61.
23. Monga M, et al. Impact of infertility on quality of life, marital adjustment, and sexual function. Urology. 2004;63(1):126–30.
24. Aisenberg Romano G, et al. The psychological profile and affective response of women diagnosed with unexplained infertility undergoing in vitro fertilization. Arch Womens Ment Health. 2012;15(6):403–11.
25. Wischmann T, et al. Psychosocial characteristics of infertile couples: a study by the 'Heidelberg Fertility Consultation Service'. Hum Reprod. 2001;16(8):1753–61.
26. Lukse MP, Vacc NA. Grief, depression, and coping in women undergoing infertility treatment. Obstet Gynecol. 1999;93(2):245–51.
27. Poddar S, Sanyal N, Mukherjee U. Psychological profile of women with infertility: a comparative study. Ind Psychiatry J. 2014;23(2):117–26.
28. Kohan S, Ghasemi Z, Beigi M. Exploring infertile women's experiences about sexual life: a qualitative study. Iran J Nurs Midwifery Res. 2015;20(1):34–9.
29. Callan VJ, Hennessey JF. The psychological adjustment of women experiencing infertility. Br J Med Psychol. 1988;61(Pt 2):137–40.
30. Domar AD, et al. The prevalence and predictability of depression in infertile women. Fertil Steril. 1992;58(6):1158–63.
31. Revonta M, et al. Health and life style among infertile men and women. Sex Reprod Healthc. 2010;1(3):91–8.
32. Vartiainen H, et al. Psychosocial factors, female fertility and pregnancy: a prospective study – Part I: fertility. J Psychosom Obstet Gynaecol. 1994;15(2):67–75.
33. Chiba H, et al. Stress of female infertility: relations to length of treatment. Gynecol Obstet Invest. 1997;43(3):171–7.
34. Leiblum SR, Aviv A, Hamer R. Life after infertility treatment: a long-term investigation of marital and sexual function. Hum Reprod. 1998;13(12):3569–74.
35. Nelson CJ, et al. Prevalence and predictors of sexual problems, relationship stress, and depression in female partners of infertile couples. J Sex Med. 2008;5(8):1907–14.
36. Turan V, et al. Sexual dysfunction in infertile Turkish females: prevalence and risk factors. Eur J Obstet Gynecol Reprod Biol. 2014;182:128–31.
37. Iris A, Aydogan Kirmizi D, Taner CE. Effects of infertility and infertility duration on female sexual functions. Arch Gynecol Obstet. 2013;287(4):809–12.
38. Keskin U, et al. Differences in prevalence of sexual dysfunction between primary and secondary infertile women. Fertil Steril. 2011;96(5):1213–7.
39. Ogawa M, Takamatsu K, Horiguchi F. Evaluation of factors associated with the anxiety and depression of female infertility patients. Biopsychosoc Med. 2011;5(1):15.
40. Frohlich P, Meston C. Sexual functioning and self-reported depressive symptoms among college women. J Sex Res. 2002;39(4):321–5.
41. Lemmens GM, et al. Coping with infertility: a body-mind group intervention programme for infertile couples. Hum Reprod. 2004;19(8):1917–23.
42. Xing X, et al. Impact of male infertility on men's self-esteem and satisfaction with sexual relationship. Zhonghua Nan Ke Xue. 2013;19(3):223–7.
43. Chachamovich JL, et al. Psychological distress as predictor of quality of life in men experiencing infertility: a cross-sectional survey. Reprod Health. 2010;7:3.
44. Beutel M, et al. Treatment-related stresses and depression in couples undergoing assisted reproductive treatment by IVF or ICSI. Andrologia. 1999;31(1):27–35.
45. Fatoye FO, et al. Psychological profile of spouses of women with infertility in Nigeria. Afr J Med Med Sci. 2009;38(1):63–9.

46. Marci R, et al. Procreative sex in infertile couples: the decay of pleasure? Health Qual Life Outcomes. 2012;10:140.
47. Smith JF, et al. Sexual, marital, and social impact of a man's perceived infertility diagnosis. J Sex Med. 2009;6(9):2505–15.
48. Ferraresi SR, et al. Current research on how infertility affects the sexuality of men and women. Recent Pat Endocr Metab Immune Drug Discov. 2013;7(3):198–202.
49. Berger DM. Impotence following the discovery of azoospermia. Fertil Steril. 1980;34(2):154–6.
50. Perlis N, et al. Coital frequency and infertility: which male factors predict less frequent coitus among infertile couples? Fertil Steril. 2013;100(2):511–5.
51. Gao J, et al. Relationship between sexual dysfunction and psychological burden in men with infertility: a large observational study in China. J Sex Med. 2013;10(8):1935–42.
52. Lotti F, et al. Clinical correlates of erectile dysfunction and premature ejaculation in men with couple infertility. J Sex Med. 2012;9(10):2698–707.
53. Pan BC, et al. Impact of perceived male infertility factors on penile erectile function. Zhonghua Nan Ke Xue. 2013;19(12):1087–90.
54. Peterson BD, Newton CR, Feingold T. Anxiety and sexual stress in men and women undergoing infertility treatment. Fertil Steril. 2007;88(4):911–4.
55. Shindel AW, et al. Premature ejaculation in infertile couples: prevalence and correlates. J Sex Med. 2008;5(2):485–91.
56. Ohl J, et al. Impact of infertility and assisted reproductive techniques on sexuality. Gynecol Obstet Fertil. 2009;37(1):25–32.
57. Schmidt L. Infertility and assisted reproduction in Denmark. Epidemiology and psychosocial consequences. Dan Med Bull. 2006;53(4):390–417.
58. Drosdzol A, Skrzypulec V. Evaluation of marital and sexual interactions of Polish infertile couples. J Sex Med. 2009;6(12):3335–46.
59. Repokari L, et al. Infertility treatment and marital relationships: a 1-year prospective study among successfully treated ART couples and their controls. Hum Reprod. 2007;22(5):1481–91.
60. Xu L, Ke HX, He FF. Psychological aspects of infertile couples in China. Zhonghua Fu Chan Ke Za Zhi. 1994;29(4):232–4, 254–5.
61. Akyuz A, et al. Studying the effect of infertility on marital violence in Turkish women. Int J Fertil Steril. 2013;6(4):286–93.
62. Tabong PT, Adongo PB. Infertility and childlessness: a qualitative study of the experiences of infertile couples in Northern Ghana. BMC Pregnancy Childbirth. 2013;13:72.
63. Dhont N, et al. 'Mama and papa nothing': living with infertility among an urban population in Kigali, Rwanda. Hum Reprod. 2011;26(3):623–9.
64. Nene UA, Coyaji K, Apte H. Infertility: a label of choice in the case of sexually dysfunctional couples. Patient Educ Couns. 2005;59(3):234–8.
65. Peterson BD, Newton CR, Rosen KH. Examining congruence between partners' perceived infertility-related stress and its relationship to marital adjustment and depression in infertile couples. Fam Process. 2003;42(1):59–70.
66. Al-Homaidan HT. Depression among women with primary infertility attending an infertility clinic in Riyadh, kingdom of Saudi Arabia: rate, severity, and contributing factors. Int J Health Sci (Qassim). 2011;5(2):108–15.
67. Dyer SJ, et al. 'Men leave me as I cannot have children': women's experiences with involuntary childlessness. Hum Reprod. 2002;17(6):1663–8.
68. Galhardo A, et al. The impact of shame and self-judgment on psychopathology in infertile patients. Hum Reprod. 2011;26(9):2408–14.
69. Lee TY, Sun GH, Chao SC. The effect of an infertility diagnosis on treatment-related stresses. Arch Androl. 2001;46(1):67–71.
70. Ashkani H, Akbari A, Heydari ST. Epidemiology of depression among infertile and fertile couples in Shiraz, southern Iran. Indian J Med Sci. 2006;60(10):399–406.
71. Drosdzol A, Skrzypulec V. Depression and anxiety among Polish infertile couples – an evaluative prevalence study. J Psychosom Obstet Gynaecol. 2009;30(1):11–20.

72. Benazon N, Wright J, Sabourin S. Stress, sexual satisfaction, and marital adjustment in infertile couples. J Sex Marital Ther. 1992;18(4):273–84.
73. Schanz S, et al. Long-term life and partnership satisfaction in infertile patients: a 5-year longitudinal study. Fertil Steril. 2011;96(2):416–21.
74. Cserepes RE, Korosi T, Bugan A. Characteristics of infertility-specific quality of life in Hungarian couples. Orv Hetil. 2014;155(20):783–8.
75. Chachamovich J, et al. Congruence of quality of life among infertile men and women: findings from a couple-based study. Hum Reprod. 2009;24(9):2151–7.
76. Tao P, Coates R, Maycock B. Investigating marital relationship in infertility: a systematic review of quantitative studies. J Reprod Infertil. 2012;13(2):71–80.
77. Wischmann T, et al. A 10-year follow-up study of psychosocial factors affecting couples after infertility treatment. Hum Reprod. 2012;27(11):3226–32.
78. Ramezanzadeh F, et al. Alterations of sexual desire and satisfaction in male partners of infertile couples. Fertil Steril. 2006;85(1):139–43.
79. Lee TY, Sun GH, Chao SC. The effect of an infertility diagnosis on the distress, marital and sexual satisfaction between husbands and wives in Taiwan. Hum Reprod. 2001;16(8):1762–7.
80. Baldwin DS. Depression and sexual dysfunction. Br Med Bull. 2001;57:81–99.
81. Pakpour AH, et al. Prevalence and risk factors of the female sexual dysfunction in a sample of infertile Iranian women. Arch Gynecol Obstet. 2012;286(6):1589–96.
82. Kerr J, Brown C, Balen AH. The experiences of couples who have had infertility treatment in the United Kingdom: results of a survey performed in 1997. Hum Reprod. 1999;14(4):934–8.
83. Schmidt L, et al. Patients' attitudes to medical and psychosocial aspects of care in fertility clinics: findings from the Copenhagen Multi-centre Psychosocial Infertility (COMPI) Research Programme. Hum Reprod. 2003;18(3):628–37.
84. Sundby J, Olsen A, Schei B. Quality of care for infertility patients. An evaluation of a plan for a hospital investigation. Scand J Soc Med. 1994;22(2):139–44.
85. Batool SS, de Visser RO. Experiences of infertility in British and Pakistani women: a cross-cultural qualitative analysis. Health Care Women Int. 2016;37(2):180–96.
86. Matthiesen SM, et al. Stress, distress and outcome of assisted reproductive technology (ART): a meta-analysis. Hum Reprod. 2011;26(10):2763–76.
87. ESHRE. Routine psychosocial care in infertility and medically assisted reproduction – a guide for fertility staff, E.P.a.C.G.D. Group, Editor. 2015. ESHRE: http://www.eshre.eu/~/media/Files/Guidelines/Psychology/Summary%20document.pdf.
88. Lara LA, et al. The assessment and management of female sexual dysfunction. Rev Bras Ginecol Obstet. 2008;30(6):312–21.
89. Farnam F, et al. Compare the effectiveness of PLISSIT and sexual health models on women's sexual problems in Tehran, Iran: a randomized controlled trial. J Sex Med. 2014;11(11):2679–89.
90. Segraves RT. Management of hypoactive sexual desire disorder. Adv Psychosom Med. 2008;29:23–32.
91. Boivin J. A review of psychosocial interventions in infertility. Soc Sci Med. 2003;57(12):2325–41.
92. Van den Broeck U, et al. Counselling in infertility: individual, couple and group interventions. Patient Educ Couns. 2010;81(3):422–8.
93. Latifnejad Roudsari R, Allan HT. Women's experiences and preferences in relation to infertility counselling: a multifaith dialogue. Int J Fertil Steril. 2011;5(3):158–67.
94. Shultz E, Malone Jr DA. A practical approach to prescribing antidepressants. Cleve Clin J Med. 2013;80(10):625–31.

Sexuality After Abuse

11

Valeria Dubini

11.1 Introduction

That violence is a "gender" problem par excellence is widely acknowledged and upheld by authoritative international organizations. Indeed, the term "gender violence" was coined by the World Health Organization (WHO) [1, 2]. With this term, it meant to group a complex panorama of anti-women behaviors that include domestic violence, sexual violence, and psychological violence, as well as mobbing or the use of the female body in an unseemly or disrespectful way. From the very first definition, made in 1993 [1], the WHO stressed the importance of the willfulness of the act regardless of its consequences, as well as the presence of a condition of asymmetry, whether due to physical strength or power, as the main feature.

Like many other worldwide problems, violence is not distributed uniformly between the sexes and/or age groups: it is "fragile" subjects like women, children, and old people, living in a condition of disparity of economic, relational, cultural, or social power, who are the main victims of these acts [3–5].

Even our supposedly civilized Europe is not immune from this problem, seeing that in 2006, the European Parliament felt the need to reaffirm in a plenary session the fact that the violence of men against women represents "a universal problem linked to an unequal distribution of power between genders that still characterizes our society."

In the last 10 years, it has been made increasingly clear that this phenomenon is not only a crime but also a serious problem for society, a violation of human rights, and a cause of "disease." It represents one of the most important global problems of public health, within the broader concept of the recognition of "gender inequality" as an epidemiological reality (along with social, economic, and cultural factors)

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_11

which, through a wide range of structural and institutional conditions (laws, economic, and family systems), influences women's mortality, morbidity, and life quality [1].

There is no doubt that domestic violence negatively influences women's well-being, not only from a psychological and social point of view but also from the physical one: gender-based violence is at the root of many chronic pathologies and psychiatric case histories, and it is certainly present among many of the women who resort to medical services. Out of this awareness comes the WHO's alarmed concern about violence as a risk factor for a serious of important pathologies in the female population and its call for the urgent need to create awareness and competence regarding these issues among health operators [1, 6–8].

The sphere of reproductive health constitutes the most representative parameter of what an asymmetrical relationship can involve: gender violence is at the root of many chronic gynecological pathologies, of the absence of contraceptive protection, and of obstetrical complications, as well as of sexual disorders. Many studies show that it is in this arena that the close tie between domestic violence and sexual violence emerges most clearly. These are associated with and often contemporaneous aspects of violence used to maintain control over reproductive choices and to make sure that the relationship remains unbalanced and asymmetrical. In this context, in 2009 "Lancet" published the results of a multicentric study based on a sample of 24,097 women, which showed a considerable "overlap" between these two phenomena: from 30 to 56 % of the women interviewed reported having suffered both physical and sexual violence, showing that they are both situated in the same well-defined sociocultural sphere of gender relations [9, 10].

11.2 Violence and Abuse: Epidemiology of the Phenomenon

Estimating the incidence of the various forms of abuse is certainly not easy. There exist various studies on the phenomenon, not always comparable because they use different criteria and/or methodologies. What is more, the episodes, especially those that take place inside the home, are often not reported and so remain hidden. Nonetheless, all studies agree on the fact that violence against women is endemic in every country in the world and across all social strata [10]. At the global level, it is calculated that at least one woman out of three has been beaten or sexually abused and that one out of four has been a victim of a form a violence during pregnancy [11–15].

Violence committed within the home on the part of a partner or someone who is known represents the most frequent even. Some studies made in hospital emergency rooms show that about 30 % of traumatic injuries among women are caused by episodes of mistreatment on the part of the partner [16]. In fact, interpersonal violence is the second cause of injuries among women between 15 and 44 years of age, preceded only by road accidents, and is at times so serious as to cause the woman's death [17, 18].

In Italy the main source of information concerning this phenomenon is Istituto di Statistica Italiano (ISTAT). The first inquiry entirely dedicated to the question of

violence and mistreatment against women, inside and outside the family, was conducted in 2006, through telephone interviews of women ages 16 and 70. The findings showed that 31.9 % of the women interviewed had suffered physical or sexual violence at least once in their lifetimes. In the majority of cases, the violence was done by their partner or ex-partner (69.7 %), in 17.4 % by someone they knew, while a mere 6.2 % of the violence was the act of an unknown person [19, 20]. This inquiry was updated between May and December 2014 and presented to the Council of Italian Ministers in June 2015. Though the more recent data are not substantially different from the prior data, some important differences can be seen: while overall there are still 31.5 % of women who have suffered some form of violence, physical and sexual violence have declined from 13.3 to 11.3 %, with a particular reduction of cases of violence against teenagers, which falls from 17.1 to 11.9 %. Psychological violence has also been sharply reduced, from 42.3 % in 2006 to 26.4 % in 2014. Unfortunately, the data relative to violence suffered before the age of 16 shows that it has increased: in 2006, it concerned 6.6 % of the sample interviewed, while in the more recent inquiry, it had risen to 10.6 %.

Fortunately, awareness of the phenomenon seems to have increased: while in 2006 33.9 % of the women interviewed said they had not spoken to anyone of the violence they had suffered, in 2014 the percentage falls to 25.8 %; what is more, the number of women interviewed who hold this violence to be a crime has grown (from 14.3 % in 2006 to 29.6 % in 2014). Reports to police authorities have increased from 6.7 to 11.8 %, as has the percentage of women who seek held in specialized centers (4.9 % against 2.4 % in 2006).

Unfortunately, the percentage of rapes has not fallen (2.1 %), and serious acts of violence with consequent injuries have increased from 26.3 to 40.2 %, along with the percentage of women who were in fear for their lives (from 21.3 to 34.5 %). Finally, for the first time, the 2014 report contains data on "stalking," which involves 16.1 % of the interviewed sample [21].

Confidence in the police has grown (28.5 % declare they are highly satisfied with their performance); instead, the percentage of women who talk with health workers remains very low: only 1.6 %, which reaches 2.5 % if we include emergency room workers [21].

A highly disturbing aspect of the problem, which is also an indicator of the degree of violence against women present in Italy, is revealed by the data on voluntary homicides: the Ricerche Economiche e Sociali Europee (EURES) report, published in 2009 based on data from 2005 to 2006 and updated in 2015 based on data from 2013, confirms first of all that 70.5 % of feminicides are committed in the family, in 68.2 % (data 2013) of cases at the hand of a partner or former partner [22].

Moreover, the report shows that while there has been an overall reduction in cases of homicide, feminicides have remained stable in the last 20 years, with an average of one woman murdered every two days.

Other data concerning Italy refer to interviews conducted by the "Telefono Rosa" ["Pink Telephone"] observatory during the year 2010, involving roughly 2,000 women. Once again, it is confirmed that 4 acts of violence out of 5 occur within the confines of a sentimental relationship, and only 1 out of 100 takes place at the hand

of an unknown person. Violent behaviors are found within domestic walls in 61 % of cases, at least as far as Italian women are concerned. On the contrary, for foreign women in 30 % of cases, violent attitudes are found in public situations. What is more, among Italian women psychological prevails over physical violence (31 vs. 23 %), while among foreign women, the contrary is true, which obliges a far greater percentage to seek hospital treatment (14 vs. 7 %).

In conclusion, in Italy too, violence represents a significant, though often underestimated, reality. Despite noisy press campaigns that appear in the wake of sensational episodes, thereby contributing to create an impression of exceptionality, in everyday affairs the phenomenon of violence against women is virtually ignored, perhaps by reassuring ourselves that it is something rare and distant, something that does not touch us.

11.3 The Repercussions of Violence on Women's Reproductive Health

Many gynecological disorders, even in very young women, can be associated with this phenomenon: lack of family planning, recourse to abortion, sexually transmitted diseases (STD), sexual disorders, and pregnancy disorders are all situations that can mask a situation of violence.

The immediate consequences of sexual violence can naturally be an unwanted pregnancy (roughly 5 % according to the WHO) [8, 23, 24] and infections. As regards the latter, a review from 2000 reported in victims of sexual violence an increased incidence of *N. gonorrhoeae* (2–6.5 %) [24], *Chlamydia trachomatis* (6–17 %), and *Trichomonas* (2.3–14.7 %). Syphilis is another sexually transmitted disease for which screening is usually recommended for victims of sexual violence. However, its prevalence as reported in the majority of studies is extremely low (0–3 %), in countries outside of Italy as well. Certainly, among the risks of infection caused by sexual violence, we should also consider an increase in the transmission of the *Papillomavirus*, with all its consequences. However, since this infective agent has high frequency in the general sexually active population, it is very difficult, if not impossible, to estimate the risk of acquiring the infection following an act of sexual violence.

As regards HIV, we should remember that the average risk of contracting an HIV infection from a single act of unprotected vaginal intercourse with an infected partner is considered to be roughly 1–2 out of 1,000, a risk that increases in cases of unprotected anal penetration (5–30 out of 1,000) and if there are lacerations certainly more frequent in cases of violence. It is clear that this problem is most important in zones where HIV is endemic (e.g.,, in Uganda it is estimated that there is an eightfold increase in the chance of contracting HIV following an unwanted sexual relation). By contrast, in Italy the disadvantages of prophylaxis with antiretroviral drugs certainly outweigh the advantages, except in special cases.

The importance of the problem of STD increases in proportion to how young the victim is, since in the phase of its initial maturation the vaginal mucus is a less

effective barrier against infections. Consequently, young adolescents are more highly exposed to these risks, with possible serious consequences for their future reproductive capacity [25].

Type of violence	Effects on reproductive health
Childhood sexual abuse (once the victims become adults and adolescents)	Gynecological problems, STD, HIV/AIDS, precocious sexual experiences, precocious pregnancies, infertility, unprotected sex, unwanted pregnancies, miscarriages/abortions, re-victimization, risky behaviors, substance abuse, suicide
Rape	Undesired pregnancy, abortions, inflammatory pelvic disease, infertility, STD including HIV infection, suicide
Domestic violence	Scarce nutrition, worsening of chronic diseases, substance abuse, concussion, organ damage, temporary or permanent disability, chronic pain, unprotected sex, inflammatory pelvic disease, gynecological problems, underweight newborns, abortions, negative pregnancy outcomes, maternal death, suicide

From "A Practical Approach to Gender-based Violence: a Programme Guide for Health Care Providers & Managers" United Nations Population Fund, New York, 2001 (cit.)

The problem of STD is also present in situations of chronic violent relations because of the woman's lower capacity to protect herself and the frequent imposition of unprotected intercourse.

Particular attention should be given to the problem of chronic pelvic pain, a recently recognized pathology which involves many specializations and which, according to the American College of Obstetricians and Gynecologists (ACOG), in 40–50 % of cases has its origins in histories of physical and/or psychological abuse. There is no doubt that chronic pelvic pain is a vague, "functional" type of pathology that frequently correlates with a history of mistreatment. To the surprise of many gynecologists, in 1993 Milburn published a work in which chronic pelvic disease was related to a series of possible causes. Though he investigated that pathologies traditionally held to be closely connected to pelvic pain, such as endometriosis and the presence of laparoscopically revealed adhesions, only association with prior sexual violence and a history of depression reached statistical significance ($p < 0.01$).

The mechanisms involved in this type of association are certainly multiple and not easy to identify. It is likely that factors of a psychological and a neurological origin coexist. Some studies suggest that the trauma can provoke biophysical alterations that produce an altered sensitivity to pain. This could occur through actions on the hypothalamus-hypophysis-adrenal axis, with an increase in the production of cortisol and consequent damage to the areas of the hippocampus and circuits sensitive to cortocotropin-releasing hormone (CRH), which could lead to an altered perception of painful stimuli [26], or else through a "deregulation" of the autonomous nervous system with growing hypersensitivity due to "upregulation" of the visceral fibers. It is likely that different mechanisms build up and strengthen each other. Probably depression and sleep disorders, common outcomes of one or more episodes of violence, also constitute factors that make the victims more vulnerable to subsequent painful stimuli [27].

Prestigious scientific societies like the ACOG and the Royal College of Obstetricians and Gynaecologists (RCOG) recommend that medical operators should take into very careful consideration the possibility of a history of violence in the analysis of chronic pelvic pain and should ask explicit questions about this possibility. They identify as crucial in the approach to this pathology the time dedicated to listening and a multidisciplinary approach (evidence level I).

Given the possible risks it can involve, diagnostic laparoscopy, for a long time held to be indispensable in the analysis of chronic pelvic disease, should instead only be taken into consideration only if felt to be necessary after an overall examination (evidence level III) [27, 28].

Strictly reproductive problems include first of all unwanted pregnancy, with consequent recourse to abortion: an article in the British Medical Journal in 2005 showed a significant association between domestic violence and request for abortions, with a trend that grew in proportion to the number of abortions – it was present in 24 % of women at the first abortion, in 30 % at the second, and arriving at almost 40 % in women with histories of more than two abortions [29, 30].

We need to realize that these problems exist even among very young women: a study published by Miller in 2010 on "contraception" reports an incidence of the very young who perceive sex in their relationship as being "forced," amounting to 35% of the young women interviewed, with consequent boycotting of contraceptives (15%) and undesired pregnancies (1:5) [31].

Not even pregnancy represents a period free from the risk of violence and mistreatment: studies show an incidence of abuse in 5 to 20% of pregnant women. Indeed, pregnancy makes a woman more vulnerable, among other things by reducing her emotional and financial independence. The changes related to this period can be experienced by the partner as an opportunity to establish power and control over the woman. It is not by chance that in 30% of cases, abuse starts precisely during pregnancy, especially in the second and third trimester, that 69% of previously abused women continue to suffer abuse during pregnancy, and that in 13% of cases, the episodes become even more frequent and serious. These are rarely isolated episodes: the acts of aggression are repeated at least twice during the course of pregnancy (60%) or even more often (15%).

The women most at risk are those who are already in a violent relationship (OR 67.6), very young women (for the age group between 16 and 19 years, the risk increases by roughly threefold), and women belonging to immigrant ethnic groups. Violence is an important risk factor in pregnancy, with consequences for both the mother and the fetus that can even lead to death: we need only recall that it represents the second cause of maternal death worldwide. Hyperemesis, detachment of the placenta, breaking of the uterus, repeated miscarriages, and blood loss in the first trimester, along with fetal death, premature birth, chorioamnionitis, and low birth weight, are some of the possible complications associated with violence in pregnancy [32–33].

In a study published in 2006 in the *American Journal of Obstetrics and Gynecologists* on a sample of over 118,000 women, Silverman showed that violence in pregnancy constitutes a significant risk factor for hypertension (OR = 1.40), blood loss (OR = 1.66), hyperemesis (OR = 1.63), urinary tract infections (OR = 1.55), premature birth (OR = 1.37), and intrauterine growth restriction (IUGR) (OR = 1.17) [34].

A study carried out by a group in Trieste coordinated by Patrizia Romito and published in 2009 in *Health Care Women* showed that violence on the part of the partner also plays an extremely important role in postpartum depression. Indeed, women who suffer violence are 13 times more likely than others to develop the so-called blue syndrome. Often alcohol, psychotropic medicine, and drug abuse are associated with maltreatment, as a way to control anxiety. It is clear that all this has potentially negative effects on the course of the pregnancy. Another characteristic is an insufficiently self-protective attitude, resulting in delayed recourse to medical assistance and missed appointments, behaviors that contribute to making pregnancy occurring in a violent relationship "at risk" [35].

11.4 Female Sexual Dysfunctions

Female sexual dysfunctions (FSD) are highly prevalent in the general population of women between 18 and 59 years of age. 33.4% of women complain of scarce sexual desire, 24.1% have difficulties in arriving at orgasm, 18.8% have problems with arousal, 14.4% suffer from pain during intercourse, and 21.2% do not find sex pleasurable. These problems increase after menopause, when the most frequent disturbance is the loss of sexual desire. Observational studies show that the decline of desire in women increases with age [36]. However, the distress caused by this disturbance is inversely correlated with age and so is greatest among younger women.

We speak of "hypoactive sexual desire disturbance," which is clinically significant, when the loss of desire is felt as stressful. Vaginal dryness, with or without dispareunia, is the second most frequent disturbance after menopause. Orgasmic disturbance is often present in comorbidity. Given the multisystemic nature of sexual functioning, often found in comorbidity are moodiness (depression is the affective disturbance most frequently associated with decline in desire), urological problems (recurrent and postcoital cystitis, stress, and urge incontinence), gynecological problems (prolapsed uterus, cystocele, rectocele), and proctologic symptoms (obstructive stipsis or, more rarely, the opposite condition of flatulence). The impact of hormone replacement therapy (HRT) on FSD is still being debated.

For FSD, the benchmark nosography today is the interdisciplinary consensus conference, "The Consensus Panel on Definition and Classification of Female Sexual Dysfunction," held in 2003 [37], which updated the definitions made during the first consensus in Boston in October 1998[38].

11.5 Present-Day Classification

11.5.1 Desire Disorders

11.5.1.1 Disorders of Desire and Sexual Interest in Women

The woman has weak or absent feelings of sexual interest or desire; she has no sexual thoughts or fantasies and is also lacking in responsive desire (i.e., response to the partner's advances). She has scarce or no motivation (reasons or incentives)

to attempt arousal. The lack of sexual interest is considered beyond the reduction considered physiological for her phase of the life cycle (at the time of the consultation) and the duration of the relationship.

11.5.1.2 Sexual Aversion Disorders

Extreme anxiety and/or disgust at the mere thought of and/or at the attempt at any sort of sexual activity.

11.5.2 Arousal Disorders

11.5.2.1 Subjective Sexual Arousal Disorder

The mental sensations of sexual arousal (sexual excitement and pleasure) deriving from any type of sexual stimulation are markedly diminished or absent. Nonetheless, vaginal lubrification or other signs of arousal may be present.

11.5.2.2 Genital Sexual Arousal Disorders

Absent or weak genital sexual arousal. The woman may report a minimum response of vulval swelling and/or vaginal lubrication from any type of sexual stimulation and reduced sexual feelings with genital fondling. Subjective mental sexual arousal can, however, be present from non-genital sexual stimuli.

11.5.2.3 Combined Genital and Subjective Arousal Disorder

The absence or marked reduction of feelings of sexual arousal (sexual arousal and pleasure) associated with absent or impaired genital sexual arousal (vulval swelling, vaginal lubrication) in response to any type of sexual stimulation.

11.5.2.4 Persistent Sexual Arousal Disorder

Spontaneous, intrusive, and unwanted genital arousal (congestion, pulsating, lubrication) in the absence of desire or sexual interest. Awareness of sexual arousal is typically – but not invariably – unpleasant. The arousal is not relieved by one or more orgasms. The sensation of physical arousal can last for hours or days or longer.

11.5.3 Orgasmic Disorder

Lack of orgasm, markedly diminished intensity of organic sensations or marked delay of orgasm from any type of stimulation, despite the self-report of a high level of sexual arousal/excitement.

11.5.4 Sexual Disorders Characterized by Pain

11.5.4.1 Dyspareunia

Persistent or recurrent genital pain during attempts at penetration or during complete vaginal penetration in intercourse.

11.5.4.2 Vaginismus

Persistent or recurring difficulties of the woman to allow vaginal entry of a penis, a finger or any object, despite her expressed wish to do so. There is often a phobic avoidance and anticipation of pain. Anatomic or other physical abnormalities must be ruled out or treated.

11.6 Abuse and Sexuality

Considering the close connection between sexuality and the psyche, it is predictable that histories of abuse involve the self-image and the sphere of desire and in the end lead to various types of sexual disorders. However, there are some differences to be taken into consideration between a history of chronic abuse, maybe in childhood, and "acute" sexual violence, like a single incident outside of the context of a violent relationship.

Disorders of the sexual sphere are found more frequently in the context of a violent relation, as the direct consequence of an unbalanced relationship, and also through the mechanism of activation of the ANS and consequent alternations of the hypothalamus-hypothesis-suprarenal axis described above. In a review of 2007, Cocker evidences the strong association between disorders of the sexual sphere and domestic violence, present in 17 of the 18 studies considered. These were mainly vulvodynia, dispareunia, and interstitial cystitis. Moreover, being or having been the victim of violence often leads to at-risk sexual behaviors – promiscuous relations and prostitution are not rare. Vice versa, reduced sexual desire and problems in reaching orgasm are also common [39].

Sex represents a form of control and domination within a violent relationship. Not infrequently, unwanted and humiliating sexual practices are imposed. Through them a form of power is achieved that makes the victim feel impotent, insecure, and unable to resist. The devaluation of a woman's body through violent and forced sexual relations makes her feel she is at the mercy of the abusive partner. Frequently she turns to antidepressants, alcohol, or drugs as an attempt to survive in a virtually unbearable situation. What is more, the implementation of a sort of "contraceptive sabotage" leads to a greater incidence of unwanted pregnancies and recourse to repeated abortions, as many studies report [31].

When violence is perpetrated by an unknown person, which as we know is actually less frequent than is commonly believed (6.2% in Italy, according to ISTAT data), the most important consequences for the woman are fear, insecurity, and lack of trust in the opposite sex. Following episodes of this type, we often see post-trauma stress syndrome, which can be accompanied by attacks of panic, flashback, or the activation of repression mechanisms. At times the pathology shows up immediately after the violent episode, but it is not infrequent to find sequela afterward, with lasting difficulties in sexual relations [40].

Many studies show that young age and penetration in the course of sexual violence are more important risk factors for future sexual problems than the use of physical violence. Immediate reactions such as anger or a sense of guilt or shame can correlate with future sexual problems, while living in a stable, loving relationship can be an important and essential element in helping to overcome the trauma [41, 42].

Certainly, one of the most difficult conditions to deal with is that of childhood abuse. These are cases in which the child places her/his trust in an adult, often a figure that ought to be protective (the father, the mother's partner, an uncle, or family friend). She trusts him also because she thinks that the adult can only want what is good for her and so the things he asks for cannot be "bad": thus the child finds himself/herself in a dramatic conflict in which he/she no longer knows what is right and what is wrong and does not even completely understand what his/her perceptions really are. Pleasure turns into pain and shame, and trust is slowly transformed into fear and suspicion.

Childhood abuse does not always come to light, because there exists a sort of blackmail or pact of mutual complicity that often keeps these episodes hidden until adulthood. Mothers who do not wish to see what is happening, perhaps because even they cannot believe it to be possible, become parts of a mechanism whose specific function is to keep these "inconfessable" episodes hidden. Seemingly small clues like lower achievement at school, behavioral changes, less willingness to play with friends, or moments of depression should arouse suspicion in these cases.

Numerous studies show that childhood abuse constitutes an extremely serious trauma that can trigger a series of physical changes that lead to alterations in behavior and sensitivity. These alterations remain over time and can show up as chronic pathologies like chronic pelvic pain, fibromyalgia, and various other disorders. In many studies the altered sensitivity to painful stimulus represents a sort of "marker" that should signal a history of abuse and cause us to ask pertinent questions (evidence level III).

Thus, though the mechanisms involved in repercussions on sexuality can be many and various, according to the type of abuse, there exist some recognizable shared features, of which the most frequent are:

Avoidance or fear of sexuality
Considering sex as an obligation
Negative feelings about contact, such as anger, disgust, or guilt
Difficulties in enjoying preliminaries or pleasurable sensations
Emotional distance during sex
Disturbing thoughts or images about sex
Unsuitable or compulsive sexual behaviors (such as sexual consumption or selling one's body)
Problems in reaching orgasm
Pelvic pain, vaginismus, and pain at penetration
Problems in holding a stable relationship

It is clear that the conditions frequently associated with histories of abuse can emerge as diverse forms of sexual dysfunction. The most common of these are those that aim at holding back intrusion by refusing sexuality as pleasure (vaginismus, disturbances of desire, anorgasmia) and those that become pain (dispareunia, chronic pelvic pain).

Conclusions

Episodes of violence and abuse have important effects on women's health (WHO). Among these we must include repercussions on sexuality, which is an essential part of the sense of well-being.

At times the history of abuse or violence can emerge by chance in the course of a gynecological, urological, or sexological examination. At other times it is the woman herself who looks for help and wants to tell her story. It is important for all medical operators to know the incidence and importance of these phenomena, as the first approach taken is crucial for working through after a trauma and also because their awareness can help uncover hidden wounds and lead the victim toward a self-awareness that is in itself therapeutic.

References

1. 85th plenary meeting ONU. 48/104. Declaration on the elimination of violence against women, 20 December 1993.
2. Krug EG, Dahlberg LL, Mercy JA, Zwi AB, Lozano R. Worl report on violence and health. Geneva: World Health Organization; 2002.
3. Heise L. Aide-Mémoire, n. 241, n.200, OMS.
4. Population Report. Ending violence against women, Series L, N°11, December 1999.
5. Sadik, N. The state of world population 2000: Lives together, worlds apart: Men and women in a time of change. New York: United Nations Population Fund (UNFPA). 2000.
6. Koss MP. The impact of crime victimization on women's medical use. J Womens Health. 1993;2(1):67–72.
7. Golding JM, Stein JA, Siegel JM, Burnam MA, Sorenson SB. Sexual assault history and use of health and mental health service. Am J Community Psychol. 1988;16(5):625–44.
8. Janssen PA, Holt VL, Sugg NK, Emanuel I, Critchlow CM, Henderson AD. Intimate partner violence and adverse pregnancy outcomes: a population-based study. Am J Obstet Gynecol. 2003;188(5):1341–7.
9. Elisberg M, Ellsberg M, Jansen HFA, Heise L, Watts CH, García-Moreno C. Intimate partner violence and woman's physical and mental health in the WHO multi-country study on women's health and domestic violence: an observational study. Lancet. 2008;371(9619):1165–72.
10. Claudia García-Moreno Henrica AFM, Jansen ME, Lori H, Charlotte W. WHO multi-country study on women's health and domestic violence against women: summary report of initial results on prevalence, health outcomes and women's responses. Geneva: World Health Organization. 2005.
11. Khurram N, Heyder AA. Violence against pregnant women in developing countries. Eur J Public Health. 2003;13(2):105–7.
12. Valladares E, Ellsberg M, Peña R, Högberg U, Persson LA. Physical partner abuse during pregnancy: a risk factor for low birth weight in Nicaragua. Obstet Gynecol. 2002;100:700–5.
13. Beck L, Morrow B, Lipscomb LE, Johnson CH, Gaffield ME, Rogers M, Gilbert BC. Prevalence of selected maternal Behaviors and experiences, Pregnancy Risk Assessment Monitoring System (PRAMS), 1999. Surveill Summ. 2002;51:1–34.
14. Connolly AM, Katz VL, Bash KL, McMahon MJ, Hansen WF. Trauma and pregnancy. Am J Perinatol. 1997;14(6):331–6.
15. Berenson AB, Wiemann CH, Wilkinson GS, Jones WA, Anderson GD. Perinatal morbidity associated with violence experienced by pregnant women. Am J Obstet Gynecol. 1994;170(6):1760–9.
16. McGrath ME, Hogan JW, Peipert JP. A prevalent survey of abuse and screening for abuse in urgent care patients. Obstet Gynecol. 1998;91(4):511–4.
17. Why mothers die1997–1999: the fifth report of the Confidential Enquires into Maternal Deaths in UK 1997–99. London: RCOG Press; 2001.
18. Saving mothers lives 2006–2008 The eighth report of the confidential enquires into maternal deaths in UK 2006–2008. London: RCOG Press; 2011.
19. ISTAT. Indagine sulla sicurezza dei cittadini ′97–′98.
20. ISTAT. Statistiche giudiziarie penali, anno 1997, Annuari. 1999.

21. ISTAT. La violenza contro le donne dentro e fuori la famiglia, Presidenza del consiglio dei Ministri, June 2015. 2014.
22. EURES Secondo Rapporto sul femminicidio in Italia. Caratteristiche e tendenze del 2013, Roma, Nov 2014.
23. Sharps P, Koziol-McLain J, Campbell J, McFarlane J, Sachs C, Xu X. Health care providers missed opportunity for preventing femicide. Prev Med. 2001;33(5):373–80.
24. Saunders E. Screening for domestic violence during pregnancy. Int J Trauma Nurs. 2000;6:44–7.
25. Stevens L. A practical approach to gender-based violence: a programme guide for health care providers & managers united nations population fund. Int J Gynaecol Obstet. 2002;78(1):111–7.
26. Sapolski et al. Hormone secretagogue release. Relationship to corticosteroid receptor occupancy in various limbic sites. Neuroendocrinology. 1990;51(3):328–36.
27. Barclay L. ACOG Issues new guidelines for chronic pelvic pain. Obstet Gynecol. 2004;103: 589–605.
28. Chronic pelvic pain, initial management, green top guideline N° 41. London: RCOG; 2012.
29. Woo J, Fine P, Goetzl L. Abortion disclosure and the association with domestic violence. Obstet Gynecol. 2005;105(6):1329–34.
30. Spurgeon D. Women requesting a second or subsequent abortion should be screened for abuse. BMJ. 2005;330:560.
31. Miller E, Decker MR, McCauley HL, Tancredi DJ, Levenson RR, Waldman J, Schoenwald P, Silverman JG. Pregnancy coercion, intimate partner violence and unintended pregnancy. Contraception. 2010;81(4):316–22.
32. Johonson JK, Haider F, Ellis K. The prevalence of domestic violence in pregnant women. BJOG. 2003;110:272–5.
33. Campbell J. Health consequences of intimate partner violence. Lancet. 2002;359(9314): 1331–6.
34. Silverman J, Decker MR, Reed E, Raj A. Intimate partner violence victimization prior to and during pregnancy among women residing in 26 U.S. states: associations with maternal and neonatal health. Am J Obstet Gynecol. 2006;195(1):140–8.
35. Romito P, Pomicino L, Lucchetta C, Scrimin F, Turan JM. The relationships between physical violence, verbal abuse and women's psychological distress during the postpartum period. J Psychosomatic Obstet Gynecol. 2009;30(2):115–21.
36. Dennerstein L, Lehert P, Burger H, Dudley E. Factors affecting sexual functioning of women in the midlife years. Climateric. 1999;2(4):54–62.
37. Basson R, Berman J, Burnett A, Derogatis L, Ferguson D, Fourcroy J, Goldstein I, Graziottin A, Heiman J, Laan E, Leiblum S, Padma-Nathan H, Rosen R, Segraves K, Segraves RT, Shabsigh R, Sipski M, Wagner G, Whipple B. Report of the International consensus development conference on female sexual dysfunction: definition and classification. J Urol. 2000;163(3):888–93.
38. Basson R, Leiblum S, Brotto L, Derogatis L, Fourcroy J, Fugl-Meyer K, Graziottin A, Heiman JR, Laan E, Meston C, Schover L, van Lankveld J, Schultz WW. Definitions of women's sexual dysfunction considered: expansion and revision. J Psychosomatic Obstet Gynecol. 2003;24(4):221–9.
39. Cocker AL. Does physical intimate partner violence affect sexual health? A systematic review. Criminol Penol. 2007;5:55.
40. Maltz W. The sexual healing journey: a guide for survivors of sexual abuse. New York: Quill; 2001.
41. Van Berlo W, Ensinkb B. Problems with sexuality after sexual assault. Annu Rev Sex Res. 2000;11(1):235–57.
42. Dubini V. Violenza contro le donne: compiti e obblighi dei ginecologi. EDITEAM; 2007

Female Sexual Dysfunction in Alcohol and Drug Abuse

12

Marina Ziche

12.1 Introduction

Sexual dysfunction is a very common problem that affects many men and women in their lifetime, but, unfortunately, this condition remains a largely unexplored field in medicine and pharmacology. The incidence of this condition is more prevalent in women (40–45 %) than in men (20–30 %) [28]. Research suggests that the majority of women experience sexual dysfunction at some point in their lives, and for many it is an ongoing or recurring issue.

Female sexual dysfunction has many causes, related to physiological, psychological, and pharmacological events. At present, in women sexual dysfunction consists of four recognized disorders with the following symptoms [5]:

(a) Hypoactive sexual desire disorder (HSDD, decreased arousal and sexual aversion)
(b) Female sexual arousal disorder (FSAD, decreased arousal and/or arousal problems)
(c) Orgasmic disorder (difficulty or inability to achieve orgasm)
(d) Sexual pain disorder

A woman can have more than one of these problems and can actually move between the four recognized disorders, making diagnosis and treatment a challenge. The two subtypes of female sexual dysfunctions that have been the focus of pharmacological research are FSAD and HSDD. However, the incomplete knowledge and understanding of physiology and pathophysiology of human sexual function reduced the development of new drugs.

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_12

At physiological level, reduction of sexual desire has been related to dopamine imbalance, decreased sexual arousal to deficiencies in nitric oxide and acetylcholine pathways, and orgasmic disorders to changes in serotonin and norepinephrine levels. Hormonal changes occurring during menarche, menstrual cycling, pregnancy, lactation, the post-partum period, and the menopause also have a negative impact on sexual function. It is not therefore surprising that a decline in sexual arousal and desire are linked to the menopause; in fact physiological changes during menopause decrease tactile stimulation, decrease mucous secretions, and induce atrophy of reproductive organs. For these reasons, age has been proven to be a substantial risk factor for increased rates of female sexual dysfunction. However, very few studies outline the prevalence of sexual dysfunction with age. For example, the relationship between sexual pain prevalence and age appeared controversial, with some studies showing a direct relationship between age and pain prevalence and others suggesting that the relationship is inverse. Certainly, the prevalence of low "interest" and low desire in women appears to be age dependent. Lewis and coworkers outlined that low desire had a prevalence of 10 % in women up to 49 years of age and then increases to 22 % among the 50- to 65-year-old group [27].

At the pharmacological level, medical conditions, particularly those that affect the neurological, vascular, and endocrine systems, can result in female sexual dysfunction. Prescription drugs (e.g., antidepressants, as selective serotonin reuptake inhibitors), alcohol, tobacco, and illicit drugs (i.e., cocaine and heroin) contribute to sexual dysfunction. At present, the level of illicit drug use is about twice the rate for men compared to women; however, women tend to abuse drugs at an earlier age than males. In addition, the drug abuse in women is increasing, and the younger generation of women is more likely to try drugs than in the past years [15, 46]. Further, drug abuse and alcohol lead women to having unprotected sex and to being caught in a cycle of violence that are responsible of many sexual dysfunctions.

In conclusion, the incidence of sexual disorders in women is closely related to physiological and psychological/emotional characteristics, but also to socioeconomic status and drug and alcohol abuse.

12.2 Sex Differences in Drug Pharmacokinetics

Recently, a large number of evidences outline the differences between man and woman as regards to drug pharmacokinetics and pharmacodynamics [17, 29, 35]. Firstly, there are many physiological parameters that characterize men and women and that influence the drug fate in the body. As reported in Table 12.1, the body mass and the body surface area are higher in men than women, and other differences are clearly visible also in tissue mass (i.e., liver) and blood flow rate [29].

Second, there are substantial differences between men and women in all the pharmacokinetic steps driving drug activity (absorption, distribution, metabolism, and elimination) [17]. Changes in drug bioavailability will depend on the route of administration and absorption. As reported, gastrointestinal motility is affected by sexual hormones and the gastrointestinal emptying time is higher in women than in men [30, 40]. Similarly, the gastrointestinal proteins/enzymes responsible for drug

transport and metabolism are different between men and women. Also drug distribution is influenced by sex, since the differences in body mass index, plasma volume, and plasma-binding protein influence drug bioavailability (Table 12.2) [45].

In addition, a great deal of new publications outlined gender differences in adverse drug reaction (ADR), since women have been reported to have a 1.5–1.7-fold greater risk than men in experiencing an ADR (Table 12.3) [31]. These events are linked to multiple factors including differences in several drug metabolizing enzymes as CYP3A [35], CYP1A2 [14], and CYP2D6 [47]. In fact, it is known that women have higher rates of CYP3A substrate metabolism compared with men [24, 43]. However, the fundamental biological differences in drug metabolism between sexes are poorly understood in molecular and cellular terms.

At last, there are sex differences in drug elimination with respect to renal drug clearance as a result of differences in blood flow [17].

Table 12.1 Physiological parameters that characterize men and women

	Male	Female
Mass (kg)	73	60
Height (cm)	176	163
Body surface area (m^2)	1.90	1.66
Fat (kg)	14.6	18.0
Tissue mass (g, varies with age)		
Liver	1.800	1.400
Lung	500	420
Kidneys	310	275
Fat (storage fat)	14.600	18.000
Blood flow rate (% cardiac output)		
Liver	25.5	27
Kidneys	19	17
Fat	5.0	8.5
Skeletal muscle	17	12

Modified from Mattison [29]

Table 12.2 Sex differences in drug pharmacokinetics

Parameter	Physiologic difference	Pharmacokinetic impact
Gastric pH	Acidity M>F	Absorption
Gastric emptying	M>F	Absorption
Intestinal motility	M>F	Absorption
Body surface area	M>F	Absorption
Plasma volume	M>F	Distribution
Body mass index	M>F	Distribution
Total body water	M>F	Distribution
Body fat	F>M	Distribution
Plasma protein	M>F	Distribution

Modified from Mattison [29]

Table 12.3 Why women experience more ADR than men?

Factors	Explanation
Physiological and lifestyle factors	Women take more medications than men Women report ADR more often than men (reporting bias) Women are exposed to drugs in different ways to men (i.e., occupation or diet)
Pharmacokinetic differences	Dose recommendations are not sex specific Drug bioavailability is sex dependent Different tissue distribution between men and women Differences in metabolism (i.e., different CYP expression) Elimination rate might be different
Differences in pharmacodynamics	Differences in drug target Membrane phenomena (differences in membrane transport) Receptor pehomena (differences in receptor number and binding) Different interaction with macromolecules
Differences in hormones	Influence of sex hormones, especially estrogen effects

Modified from Mennecozzi et al. [31]

12.3 Antidepressants and Sexual Dysfunction

Antidepressants are prescription drugs that have been used effectively and appropriately to treat both medical and psychiatric illness in the vast majority of patients, but in the past years, rates of prescription abuse have escalated and have reached epidemic proportions. The use/abuse of antidepressant is very high, and, for example, in the USA, the National Center for Health Statistics (NCHS) reported that the rate of antidepressant use in this country among teens and adults (people ages 12 and older) increased by almost 400 % between 1988–1994 and 2005–2008 [38]. Concerning female use/abuse of antidepressant, data indicate that 23 % of women in their 40s and 50s take antidepressants, a higher percentage than any other group (by age or sex), and that women are 2½ times more likely to be taking an antidepressant than men. The different use of antidepressant depends also on women's age (Fig. 12.1).

The reported incidence of sexual dysfunction associated with all the different classes of antidepressants varies between studies, but is very high (from 25 to 90 % depending on the studied compound). As shown in Table 12.4 perhaps all the antidepressants can induce sexual dysfunction.

All the drugs inhibit the breakdown of norepinephrine causing an increased neurotransmitter availability at the synapse. In addition, most antidepressants modulate the serotonin concentration, a well-known modulator of sexual function, since elevated serotonin levels diminish sexual functions. In the brain, serotoninergic nerve terminals interfere with dopamine and norepinephrine pathways blocking their activity. Similarly in the periphery, serotonin directly reduces sensation in the anatomical structure of the reproductive tract by diminishing vaginal lubrication and orgasm [22].

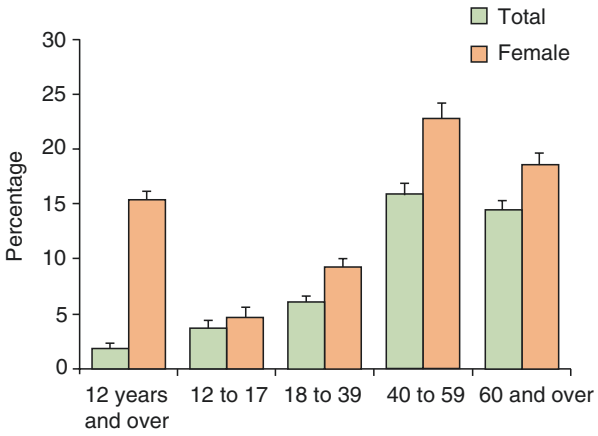

Fig. 12.1 Distribution of antidepressant drug users in the USA by age and sex [38]

Table 12.4 Antidepressants and female sexual dysfunction

Drug class	Medication	Mechanism of action	Prevalence and symptoms of female sexual dysfunction
TCAs	Amitriptyline, desipramine, doxepin, imipramine	Inhibition of serotonin and norepinephrine reuptake	Prevalence: 30 % Breast enlargement and decreased orgasm
SSRIs	Citalopram, escitalopram, fluoxetine, sertraline	Inhibition of serotonin reuptake	Prevalence: from 25 to 75 % Decreased libido and delayed or inability to reach orgasm
SNRIs	Duloxetine, venlafaxine	Inhibition of serotonin and norepinephrine reuptake	Prevalence: 58–70 % Delayed or absent orgasm
MAOIs	Phenelzine	Monoamine oxidase inhibitors	Prevalence 40 % Decreased libido, delayed orgasm
	Bupropion	Inhibition of serotonin, dopamine, and norepinephrine reuptake	Prevalence 10–25 %

Modified from [22]

12.4 Alcohol and Sexual Dysfunction

An association between chronic and high amount of alcohol consumption and sexual dysfunction has been widely reported [37]. Several clinical and experimental studies concluded that in male alcoholics, the greater quantity, frequency, and duration of drinking are associated with inhibited libido and retarded ejaculation. In alcohol-dependent women, the incidence of sexual dysfunction is very high when

compared with nonalcoholic women [37], and the most common forms of sexual dysfunctions observed include dyspareunia, high rates of genitourinary problems, and low vaginal lubrication, revealing problems with sexual arousal [12, 41]. At present epidemiological data correlate gender prevalence of sexual dysfunction to alcohol abuse but, in general, women tend to be more vulnerable to alcohol-induced physical illness [8, 23] and display more severe cognitive and motor impairment with lower alcohol assumption when compared with men [33].

Barquin and colleagues in a recent paper showed the different effects of acute alcohol consumption in men and women. Fifteen minutes after drinking 95 ml of wine, almost half of the female participants had breath alcohol levels above the legal limit for driving in Europe. None of the males had levels above that limit. Over the time the women's alcohol levels continued to remain higher than in men [4]. This is a simple example that clearly demonstrates the differences after alcohol assumption in women and men. Indeed, several studies have demonstrated gender differences in alcohol pharmacokinetics both in animal and human models [10, 42]. Women have higher and persistent blood alcohol levels when compared with men for a given dose indicating gender differences in drug bioavailability, linked to speed of gastric emptying, gastric enzyme activity, and first-pass metabolism [19]. In fact, the activity of gastric alcohol dehydrogenase (ADH) is lower in women than in men [18]. Moreover, also the alcohol distribution is different, being the alcohol volume of distribution lower in women than in men [3].

12.5 Tobacco and Sexual Dysfunction

Cigarette smoking is the most preventable cause of morbidity and premature mortality worldwide. Impairment in sexual functioning has also been reported as a consequence of long-term tobacco intake both in men and in women. Several reports indicate that tobacco is an independent risk factor for promoting erectile dysfunction in men, but limited research has investigated the link between sexual dysfunction and tobacco in women [7, 9, 36]. The pathological processes by which tobacco may affect physiological mechanisms underlying sexual function are not understood. Sexual arousal in men and women is a complex neurovascular event in which smooth muscle relaxation promotes arterial inflow to the genitals thereby facilitating vasocongestion [39]. Nitric oxide (NO), produced within endothelial cells, has been identified as the principal mediator of vascular events in men and the primary regulator of vaginal hemodynamics in women [6, 25], and several studies have clearly shown that smoking is associated with decreased NO in arteries and veins. For these reasons, investigators have proposed that free radicals and other compounds found within cigarettes may decrease the synthesis of NO directly, or indirectly, resulting in decreased genital vasoengorgement in men [13]. Harte and Meston, in two different works, studied the acute effects of isolated nicotine in nonsmoking men and women [20, 21]. They observed that nicotine significantly reduced physiological sexual arousal responses [20, 21]. In addition Diehl and colleagues, in a

study on female sexual dysfunction associated with an history of drug abuse and smoking, showed that the sexual symptoms are primarily associated with high levels of nicotine dependence in respect to other drugs (i.e., cocaine, crack) [11]. Considering that in women and men the hemodynamic processes of sexual arousal are similar and considering that NO isoforms have been shown to be present in genital epithelia of both men and women, smoking may affect physiological mechanisms underlying sexual arousal similarly in men and women. This may be the result of nicotine and/or particular tobacco compounds exerting their effects in the different ways: (a) centrally, by eliciting dose-dependent neurotransmitter and neuroendocrine effects; (b) peripherally, by acting as a sympathetic nervous system agonist; or (c) by disrupting NO synthesis directly or indirectly by targeting biochemical precursors.

12.6 Illicit Drug Abuse/Dependence and Sexual Dysfunction

Illicit drugs as cocaine, heroin, and cannabis have long been associated with sexual dysfunction although with not unequivocal data since cocaine, for example, facilitates sexual behavior directly through its acute pharmacological activity [2, 26, 32]. In fact, cocaine has a reputation as an aphrodisiac, and ends up having the reverse effect. It increases sexual desire while impairing or delaying orgasm. However, a symptom of heavy cocaine abuse is a massive decline in sex drive and activity. Chronic cocaine use can impair sexual function in men and women.

As for alcohol dependence, men are more likely to be cocaine dependent than women, but women have a more rapid progression of the illness and higher incidence of comorbid psychiatric disorders [34]. At this time very few studies have investigated sex differences in cocaine pharmacokinetics. Evans and Foltin, in a recent review, concluded that there are minimal sex differences in the pharmacokinetics of both smoked and intranasal cocaine administration, while these differences appear to be related to progesterone levels [16]. Chronic cocaine abuse is also associated with hyperprolactinemia and sexual dysfunction symptoms, such as diminished libido and difficulty reaching orgasm.

Among chronic heroin and morphine users, a review study noted decreases in sexual intercourse frequency, in the quality and frequency of orgasm and masturbation [41]. These effects are linked to opioid-induced inhibition of the hypothalamic-pituitary-gonadal axis and increase of prolactin levels, which affect both the male and female sexual response [2, 26].

Experimental studies, examining the effects of cannabis abuse on sexual function, have identified potential links between chronic cannabis smoking and inhibited orgasms [1, 44].

Conclusions

Sexuality is a complex phenomenon that is being influenced by psychological as well as physiological factors. Sexual dysfunctions, including desire, arousal, orgasmic, and sex pain disorders, are coordinated by neurologic, vascular, and

endocrine systems. The incidence of sexual disorders in women is higher than in men and previous studies indicated that about 76 % of women experience sexual disorders. In particular it is clear that drug abuse, including alcohol consumption and smoking, affects women sexuality; however, the knowledge about association between sexual dysfunction and substance abuse remains largely unexplored.

Physiological/anatomical and genetic differences between men and women may partially account for the higher incidence of sexual dysfunction in women abusing of illicit drugs or alcohol or smoking; however, psychological/emotional and sociocultural differences among women make it difficult to clearly correlate substance abuse with sexual dysfunction.

Although the focus on gender is relatively new in medicine and pharmacology, it is now well known that gender's impact is complex and far to be reached. A broad and detailed picture of gender-related sexual dysfunctions, linked to drug abuse/misuse, tobacco, or alcohol, can lead to improvements in prevention efforts that bring us closer to the goal of gender-specific interventions.

References

1. Aversa A, Rossi F, Francomano D, et al. Early endothelial dysfunction as a marker of vasculogenic erectile dysfunction in young habitual cannabis users. Int J Impot Res. 2008;20(6):566–73.
2. Bang-Ping J. Sexual dysfunction in men who abuse illicit drugs: a preliminary report. J Sex Med. 2009;6(4):1072–80.
3. Baraona E, Abittan CS, Dohmen K, et al. Gender differences in pharmacokinetics of alcohol. Alcohol Clin Exp Res. 2001;25(4):502–7.
4. Barquín J, Luna Jde D, Hernández AF. A controlled study of the time-course of breath alcohol concentration after moderate ingestion of ethanol following a social drinking session. Forensic Sci Int. 2008;177(2-3):140–5.
5. Basson R, Berman J, Burnett A, et al. Report of the international consensus development conference on female sexual dysfunction: definitions and classifications. J Urol. 2000;163(3):888–93.
6. Burnett AL, Lowenstein CJ, Bredt DS, et al. Nitric oxide: a physiologic mediator of penile erection. Science. 1992;257(5068):401–3.
7. Cayan S, Akbay E, Bozlu M, et al. The prevalence of female sexual dysfunction and potential risk factors that may impair sexual function in Turkish women. Urol Int. 2004;72(1):52–7.
8. Ceylan-Isik AF, McBride SM, Ren J. Sex difference in alcoholism: who is at a greater risk for development of alcoholic complication? Life Sci. 2010;87(5-6):133–8.
9. Christensen BS, Grønbaek M, Pedersen BV, et al. Associations of unhealthy lifestyle factors with sexual inactivity and sexual dysfunctions in Denmark. J Sex Med. 2011;8(7):1903–16.
10. Crippens D, White ML, George MA, et al. Gender differences in blood levels, but not brain levels, of ethanol in rats. Alcohol Clin Exp Res. 1999;23(3):414–20.
11. Diehl A, Lopes da Silva R, Laranjeira R. Female sexual dysfunction in patient with substance-related disorders. Clinics (San Paulo). 2013;68(2):205–11.
12. Dişsiz M, Oskay UY, Beji NK. Use of alcoholic beverages and other psychoactive substances among women in Turkey: medical, biological, and social consequences. A pilot study. Subst Use Misuse. 2010;45(7-8):1060–76.

13. Elhanbly S, Abdel-Gaber S, Fathy H, et al. Erectile dysfunction in smokers: a penile dynamic and vascular study. J Androl. 2004;25(6):991–5.
14. Ereshefsky L, Saklad SR, Watanabe MD, et al. Thiothixene pharmacokinetic interactions: a study of hepatic enzyme inducers, clearance inhibitors, and demographic variables. J Clin Psychopharmacol. 1991;11(5):296–301.
15. European monitoring center for drug and drug addiction, annual report 2006.
16. Evans SM, Foltin RW. Does the response to cocaine differ as a function of sex or hormonal status in human and non-human primates? Horm Behav. 2010;58(1):13–21.
17. Franconi F, Brunelleschi S, Steardo L, et al. Gender differences in drug responses. Pharmacol Res. 2007;55(2):81–95.
18. Frezza M, di Padova C, Pozzato G, et al. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. N Engl J Med. 1990;322(2):95–9. Erratum in: N Engl J Med 1990 323(8):553. N Engl J Med 1990 322(21):1540.
19. Graziani M, Nencini P, Nisticò R. Genders and the concurrent use of cocaine and alcohol: pharmacological aspects. Pharmacol Res. 2014;87:60–70.
20. Harte CB, Meston CM. Acute effects of nicotine on physiological and subjective sexual arousal in nonsmoking men: a randomized, double-blind, placebo-controlled trial. J Sex Med. 2008;5(1):110–21. Erratum in: J Sex Med. 2010 7(11):3803.
21. Harte CB, Meston CM. The inhibitory effects of nicotine on physiological sexual arousal in nonsmoking women: results from a randomized, double-blind, placebo-controlled, cross-over trial. J Sex Med. 2008;5:1184–97. Erratum in: J Sex Med. 2010 7(11):3803.
22. Higgins A, Nash M, Lynch A. Antidepressant-associated sexual dysfunction: impact, effects, and treatment. Drug Healthc Patient Saf. 2010;2:141–50.
23. Hommer D, Momenan R, Kaiser E, et al. Evidence for a gender-related effect of alcoholism on brain volumes. Am J Psychiatry. 2001;158(2):198–204.
24. Hunt CM, Westerkam WR, Stave GM. Effect of age and gender on the activity of human hepatic CYP3A. Biochem Pharmacol. 1992;44(2):275–83.
25. Kim SW, Jeong SJ, Munarriz R, et al. Role of the nitric oxide-cyclic GMP pathway in regulation of vaginal blood flow. Int J Impot Res. 2003;5(5):355–61.
26. Kopetz CE, Reynolds EK, Hart CL, et al. Social context and perceived effects of drugs on sexual behavior among individuals who use both heroin and cocaine. Exp Clin Psychopharmacol. 2010;18(3):214–20.
27. Lewis RW, Fugl-Meyer KS, Bosch R, et al. Epidemiology/risk factors of sexual dysfunction. J Sex Med. 2004;1(1):35–9.
28. Lewis RW, Fugl-Meyer KS, Corona G, et al. Definitions/epidemiology/risk factors for sexual dysfunction. J Sex Med. 2010;7(4 Pt 2):1598–607.
29. Mattison DR. Pharmacokinetics in real life: sex and gender differences. J Popul Ther Clin Pharmacol. 2013;20(3):e340–9.
30. Mearadji B, Penning C, Vu MK, et al. Influence of gender on proximal gastric motor and sensory function. Am J Gastroenterol. 2001;96(7):2066–73.
31. Mennecozzi M, Landesmann B, Palosaari T, et al. Sex differences in liver toxicity-do female and male human primary hepatocytes react differently to toxicants in vitro? PLoS One. 2015;10(4):e0122786.
32. Mialon A, Berchtold A, Michaud PA, et al. Sexual dysfunctions among young men: prevalence and associated factors. J Adolesc Health. 2012;51(1):25–31.
33. Miller MA, Weafer J, Fillmore MT. Gender differences in alcohol impairment of simulated driving performance and driving-related skills. Alcohol Alcohol. 2009;44(6):586–93.
34. Najavits LM, Lester KM. Gender differences in cocaine dependence. Drug Alcohol Depend. 2008;97(1-2):190–4.
35. Nicolson TJ, Mellor HR, Roberts RR. Gender differences in drug toxicity. Trends Pharmacol Sci. 2010;31(3):108–14.
36. Oksuz E, Malhan S. Prevalence and risk factors for female sexual dysfunction in Turkish women. J Urol. 2006;75(2):654–8.

37. Peugh J, Belenko S. Alcohol, drugs and sexual function: a review. J Psychoactive Drugs. 2001;33(3):223–32.
38. Pratt LA, Brody DJ, Qiuping GU. Antidepressant use in person aged 12 and over: United states, 2005-2008. NCHS data brief 76. Hyattsville: U.S. Dept. of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; 2011.
39. Rosen RC, McKenna KE. PDE-5 inhibition and sexual response: pharmacological mechanisms and clinical outcomes. Annu Rev Sex Res. 2002;13:36–88.
40. Sadik R, Abrahamsson H, Stotzer PO. Gender differences in gut transit shown with a newly developed radiological procedure. Scand J Gastroenterol. 2003;38(1):36–42.
41. Saso L. Effects of drug abuse on sexual response. Ann Ist Super Sanita. 2002;38(3):289–96.
42. Sato N, Lindros KO, Baraona E, et al. Sex difference in alcohol-related organ injury. Alcohol Clin Exp Res. 2001;25(5 Suppl ISBRA):40S–5.
43. Schmidt R, Baumann F, Hanschmann H, et al. Gender difference in ifosfamide metabolism by human liver microsomes. Eur J Drug Metab Pharmacokinet. 2001;26(3):193–200.
44. Shamloul R, Bella AJ. Impact of cannabis use on male sexual health. J Sex Med. 2011;8(4):971–5.
45. Soldin OP, Mattison DR. Sex differences in pharmacokinetics and pharmacodynamics. Clin Pharmacokinet. 2009;48(3):143–57.
46. Stengel C, Fleetwood J. GDPO, global drug policy observatory. 2004.
47. Walle T, Walle UK, Cowart TD, et al. Pathway-selective sex differences in the metabolic clearance of propranolol in human subjects. Clin Pharmacol Ther. 1989;46(3):257–63.

Female Sexual Function During Male Sexual Dysfunction and Vice Versa

13

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Abbreviations

BDI	Beck Depression Inventory
ED	Erectile dysfunction
FSD	Female sexual dysfunction
FSFI	Female Sexual Function Index
IELT	Intravaginal ejaculation latency time
ISSM	International Society of Sexual Medicine
PC	Prostate cancer
PE	Premature ejaculation
PEP	Premature ejaculatory profile
PGE	Prostaglandin E
RRP	Retropubic radical prostatectomy

Epidemiologic data indicate that female sexual dysfunctions concern from 20 to 60 % of the population [1–9].

The most frequent disorders of the sexual sphere are desire disorder (64 %: range 16–75 %), followed by orgasm difficulty (31 %: range 12–64 %), arousal difficulty (31 %: range 12–64 %), and sexual pain (26 %: range 7–58 %) [10].

Many factors may influence female sexual function: age, race, education, psychophysical health, menopause [1, 8, 9], and male partner's sexual dysfunctions, as well. In fact, female partners of men suffering from ED present a higher rate of sexual dysfunctions [11].

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_13

Sexology and clinical practice have always highlighted how sexuality influences a relationship stability. To this end, it is interesting to mention Herbenick's study [12], which highlights how female sexuality is a complex field deserving an in-depth analysis.

Sexologists have always known that men and women live their sexuality very differently: generally, women seem to have a weaker sexual drive than men and to be more influenced by cultural variables [13–20].

In general, female sexual drive is focused on a "primary partner" and strictly connected to sexual satisfaction [13, 15].

Herbenick underlines how sexuality is a subtle interaction between a couple's members and how this may vary, when changing even a single parameter. For example, a decline in desire in one or both of the partners is one of the main problems experienced by couples engaged in a long-term relationship [14, 18], and it seems to concern from 10 to 40 % of women. Sex drive is the fruit of complex social interactions between a woman and her partner [21–24].

It is difficult to understand and study these issues, because couples do not often perceive these as a problem that needs physician's help, but just as a normal evolution of their relationship.

However, the management of sexual problems calls for coordination of the evaluation and treatment of male and female sexual problems as one unit within the context of the couple [1, 26].

In this chapter, we want to explain in detail the effect that male dysfunctions have on female sexuality and vice versa.

13.1 Premature Ejaculation (PE)

The definition of premature ejaculation has been modified over the time, until 2007, when the International Society of Sexual Medicine (ISSM) gathered a group of experts to find a univocal definition.

The panel defined PE as having three components:

1. Ejaculation that always or nearly always occurs before or within about 1 min of vaginal penetration
2. The inability to delay ejaculation all or nearly all vaginal penetration
3. Negative personal consequences such as distress, bother, frustration, and/or the avoidance of sexual intimacy [27]

PE has generated great interest in the last few years, both because new drugs have been discovered lately to cure this pathology and because of the frequency of this disorder, which concerns from 10 to 30 % of male population [1, 28].

More than in other pathologies, in PE, the female partner's perception of the problem is judged to be fundamental [5, 7, 29]. In a study conducted in 2014 [30],

Burri underlines that 77.9 % of male partners suffering from PE report at least one problem belonging to the sexual sphere:

- 49.8 % reduced libido
- 41.3 % sexual dissatisfaction

The correspondence between the data inherent to a subjective perception of dissatisfaction and the data emerged from Female Sexual Function Index (FSFI) score were statistically significant for all the fields, except for sexual desire.

It is interesting to notice that in 45.9 % of cases, interviews indicate duration of intercourse as fundamental to classify a sexual performance as good, in 60.9 % of cases because the duration of the intercourse allows them to reach orgasm and in 60.4 % of cases because the duration creates intimacy.

Patients suffering from PE have an average intravaginal ejaculation latency time (IELT) of about a minute, in contrast both with the female and male ideal of duration of sexual intercourse, which varies from about 14.34 min to about 23.2 min (from penetration to ejaculation) for her and 18.45 min for him [31].

24.9 % of women, who think that their partner's PE is supported by their "selfish" attitude, present a higher incidence of sexual problems [30].

With regard to orgasm, 3.9 % of women report they never experience it, 7.3 % never experience it during the intercourse, and 4.4 % never experience it with the current partner suffering from PE (younger women tend to have more troubles reaching an orgasm).

A number of studies highlight that these patients' female partners are less sexually satisfied and have more problems concerning sex drive, arousal, and orgasm than those with "healthy" partners [10].

There is a correlation between the "ideal duration of intercourse" and the ability to reach an orgasm and between the difficulty to reach a vaginal orgasm and the fact that the partner prefers sexual "fantasy" than duration of intercourse.

Moreover, there is a positive correlation between the global female sexual function and the ability to keep control of ejaculation and distress.

22.8 % of women report that the ejaculatory disorder underlies the coupe's breakup. This is most frequent when women have higher expectations on the ideal duration of intercourse [30].

Women suffering from sexual dysfunctions face breakup more often, because the female partners of men suffering from PE have a lower perception of the quality of sexual life and sexual relationship than the control groups. These results may be secondary to the sexual dysfunction or may depend directly on the real dissatisfaction with the couple's sexual relationship [10].

26.9 % of women report that the lack of control of ejaculation undermines the relationship.

It looks like female partners are much more interested in the perceived IELT than in the real one. A low IELT causes a woman's distress more than the lack of control [30].

Two thirds of women who report problems regarding the sexual sphere started having problems once they met the partner with PE; nevertheless, some authors suppose FSD might depend not only from the partner's PE but also from other factors such as, for example, the duration and the satisfaction with the relationship, or the fact that PE is lifelong or acquired.

There is a small vacuum to fill in literature: we should try to understand if there is a precise correlation between the appearance of the female sexual disorder and the partner's PE.

It is interesting to notice that we cannot affirm that a male sexual disorder creates a sexual disorder in the female partner, but we can state that different male disorders generate different female disorders. In fact, while female partners of PE patients have mainly orgasm and arousal problems, female partners of men suffering from ED have orgasm and sex drive problems.

In some cases, reaching and maintaining arousal is particularly difficult both because completing the act is often perceived like a "race" and because PE generates anxiety [10].

While conducting a premature ejaculatory profile (PEP) analysis on the female partners of men with PE, a reduced value has been observed than in the partners of men not suffering from PE. This indicates that partners have a correct perception of the problem. 45.7 % of female partners report that their partners' ejaculation control is "poor" or "very poor" (against 3.4 % of controls); likewise sexual satisfaction is "poor" or "very poor" in 25.6 % of cases, against 1.1 % of control groups. There is a similar trend in personal distress (52.8 % against 5.9 %) and interpersonal issues (32.8 % against 1.9 %) [32].

Dapoxetine administration for PE treatment in the male subject leads to a "dose-dependent" sexual satisfaction improvement for the partner. This goes from 53 to 72 % with Dapoxetine 30 mg and from 58 to 78 % with Dapoxetine 60 mg [33].

13.2 Erectile Dysfunction (ED)

ED is defined as the incapability to maintain or reach erection.

In the most thoroughly studied sexual dysfunction in the contest of epidemiologic research, ED is estimated to carry an overall adult male (older than 20 years of age) prevalence rate of 10–20 % worldwide, with the majority of studies reporting a rate closer to 20 %.

Current data have also confirmed that the prevalence of ED increases with increasing age and the presence of comorbid medical conditions, which include type 2 diabetes mellitus, obesity, cardiovascular disease, hypertension, dyslipidemia, depression, and prostate disease/benign prostatic hypertrophy (BPH) [27]. Many female partners of men suffering from ED report interpersonal issues and dissatisfaction with their relationship [7, 29].

Optimal management of ED requires the partner's collaboration. Partners' resentment of "artificial" means of erection may cause discontinuation of effective treatments for ED [34].

Erectile dysfunction (ED) reduces the quality of life of both the male and his female partner. Effective treatments for ED are available, but reestablishing erectile function does not necessarily reestablish a satisfying sexual relationship of the couple due to an existing female sexual dysfunction (FSD) [25].

The high proportion of female partners with FSD, in Greenstein's study the 55 % in total, in other studies even the 35 % presenting more than a sexual dysfunction [35], demonstrated the importance of taking a sexual dysfunction history from both partners. FSD, which appeared to have a strong negative effect on the success of the male partner's treatment, is a factor that needs to be recognized by urologists who treat male sexual dysfunction [36].

Relational factors, in addition to organic, psychological, could coexist in men with ED, and FSD may play a role on interrelationship of the couple.

Therefore, for optimal outcome of ED treatment, evaluation and treatment of male and female sexual dysfunction should be addressed as one unit within the context of the couple and be incorporated into one clinic of sexual medicine.

As mentioned above, speaking about EP, female sexual dysfunctions have different aspects, in particular:

- Reduced sexual interest
- Reduced pleasure during intercourse
- Difficult lubrication
- Failure to reach orgasm
- Physical pain during intercourse

In 2014, Zhang et al. published an interesting study based on the interviews of 1518 women taken from the "healthy" population, which seem to scale ED down on FSD [37].

Among all the couples interviewed, in 27 % of cases, the female partner suffered from disorders belonging to the sexual sphere, and in 5 % of cases, the male partner suffered from ED.

Twenty-seven percent of women who suffered from FSD were divided as follows:

- 10.9 % reduced sexual interest
- 10.3 % failure to feel pleasure during intercourse
- 9.2 % difficult lubrication
- 8.5 % failure to reach orgasm
- 9.1 % delayed orgasm
- 9.5 % physical pain during intercourse

FSD presence had increased in the female partners of ED suffering couples (38.1 % against 26.4 %), with a higher incidence in all the fields, but the only field where FDS difference increased in a statistically significant way was physical pain during intercourse.

What distinguishes Zhang's study from the others [38], which underlines a greater correlation between FSD and ED, is the fact that it doesn't apply to a specific category of patients, but to population in general.

The minor impact of ED respect of FSD shown in Zhang's study might indicate that the complexity of the subject needs further in-depth analysis.

The scientific evidence of how a treatment for ED (i-PDE5, intracavernous injections or prosthesis) may positively affect female sexuality, even if true, perhaps cannot explain alone the complexity of the phenomenon [39, 40].

Among Zhang's bias, we must take into account that only married couples were enrolled for the study; in this way, excluding part of the population and questionnaires were not validated.

If there is a mismatch between the partner's and the patient's perceptions, there is the same discrepancy between what the patient reports in questionnaires and other forms of communication.

Some authors highlight that the therapy for ED has beneficial effects on the couple.

Vardenafil is an effective ED treatment in men that also significantly improves sexual function/satisfaction in untreated women partners. Women partners' sexual function improvements relate significantly and consistently to treatment-related improvements in men's erectile function. ED management should emphasize both couple's members.

Second-line therapy, like PGE intracavernous injections or the use of vacuum device, may have beneficial effects on female sexuality, as well. Statistical analysis indicated that the women responded equally well to both treatments. They demonstrated significant increases in frequency of intercourse, sexual arousal, coital orgasm, and sexual satisfaction.

The women reported feeling more at ease in their relationships and characterized sex as more leisurely relaxed and assured. Negative responses focused on the lack of spontaneity and hesitation about initiating sex. Self-injection and vacuum pump therapy restore potency in men and secondarily facilitate improved sexual function in women [41].

In the last few years, a number of in-depth analyses were conducted on the couple's/woman's response to radical prostatectomy complications.

Two of the main radical prostatectomy complications are impotence and incontinence, independently from the used technique.

Partial potency recovery is reached using the nerve-sparing technique, whether using i-PDE5 or not, in:

95 % of 40-year-old patients
85 % of 50-year-old patients
75 % of 60-year-old patients
50 % of 70-year-old patients [27]

After the RRP, the couple needs to cope not only with the DE but with the acceptance of the neoplastic illness, as well [42]. In fact, patients face prostatic tumor both as individuals and as couples [43].

The issues related to neoplastic presence may cause changes in the functional abilities to such an extent that it may affect the roles and the interpersonal

relationships in a family [44]. This has a great impact on the psychological asset of the partners of radical prostatectomy patients [45, 46]; in fact, they experience a greater emotional distress than their male partners [46].

Most studies are about the short-term effects of RRP (2–8), but Wittmann's has the merit of being one of the first studies to investigate the long-term ones [42].

The sexual function recovery in couples shows a better trend when the couples are guided by means of therapeutic and behavioral suggestions, instead of being left to themselves [47–55].

Certainly, the couple support during the recovery of sexuality cannot be based only on the acceptance of the tumor, as demonstrated by Manne's study [56].

The biopsychosocial impact of post-RRP ED is known [57–59]; notwithstanding, the urologist, thanks to his/her clinical skills, tends to focus on curing the disease and the ED by means of a pharmacological therapy, rather than on giving the right psycho-sexological tools to the couple.

Wittmann's purpose is to build a model that may intervene on the bio (ED and female sexual function), on the psycho (sexual confidence), and on the social (sexual interactions).

Sometimes, the recovery of the erection is not enough for couples to consider themselves completely satisfied.

After the surgery, couples initially experience the loss of the bio, the psycho, and the social.

Couples with the best recovery abilities usually work on dialogue and acceptance of loss [60], on top of the erectile deficit in itself.

The intentional commitment in improving sexuality would take to an arousal and to a female sex drive improvement [61].

The incapability of a couple to recover their sexuality arises from their inability to accept changes such as urinal incontinence or interpersonal issues, from their refuse to accept medical aid to improve erection.

Interesting data emerge from Wittmann's study: independently from the partner's postmenopausal sexuality, the recovery of sexuality seems to be directly proportional to the couple's interest in recovering, contrary to what reported by Shindel's study conducted in 2005 [62].

The fact that the partner's postmenopausal state is uninfluential shows that psychosocial/interpersonal factors are as important as the biological ones.

The real recovery of sexuality may require even 2 or 3 years, contrary to the case of ED.

Couples should be exhorted to take a psycho-sexological route and to experience non-penetrative sex.

Kornblith et al. [46] demonstrates that a good therapeutic response to tumor (chemotherapy) may be associated to reduced anxiety and depression in the partner, so we can also hypothesize an improvement of the sexual sphere.

Patients readily show great worries about the decline of their sexual function, while their partners seem to be more focused on pain and physical limitations [63].

While patients try to face their depression when they are part of a consolidated couple, their female partners may feel distressed as an "avoidance of cancer thought"

reaction, hyper-involvement or hyperexcitability, that translates into a decline of the couple's relationship [64].

In the case of a dying patient, his partner's levels of anxiety and depression increase sensibly, most of all during the last 3 months of life [65].

The partner's reaction is clearly correlated to her self-confidence: the more she is confident, the better she will react to the event and the better the quality of her life will be [66]. However, this aspect deserves an in-depth analysis, because the studies conducted up to now have been carried out on precocious neoplasm.

In 1998, Germino studied that white women had a more negative attitude toward illness than black ones, underlying that different races could react differently to the neoplastic disease [67].

According to Gray's study published in 2000, couples face various steps. At first, women show solidarity to their partner; after some months, they try to cope with his depression and irritability, but they lose their reassuring behavior over the time [68].

The distress reported by the patient often is less than the one reported by the partner when interviewed about her husband's distress [69], so it is important to compare both partners' opinions.

One third of women after their husband's prostate cancer (PC) surgery report of being dissatisfied with their own sexual life; one fifth of these decide to undergo a sexual therapy together with their husband, but only one fifth of them will be satisfied with the therapy [53].

Despite being satisfied with their sexuality, many women are afflicted by their husband's sexual dissatisfaction [70].

There are few data in literature about female dysfunction impact on male dysfunction onset.

In our experience, we have often observed how, unfortunately, the lack of couples' therapy specialized hospital structures and sometimes patients' unwillingness to face such issues as a couple drive men to unfaithfulness. However, these data are extremely preliminary.

A review published by Riley [34] highlights that PE incidence in men who are in a relationship with dysfunctional women is higher than in normal population.

The highest PE incidence is found in the partners of women suffering from sex drive disorders (29.9 %), arousal/lubrication disorders (42.7 %), anorgasmy (47.8 %), and women not enjoying sex (51.5 %) [71–76].

A study conducted by Lonnée-Hoffmann in 2014 about female pelvic surgery and male disorders belonging to the sexual sphere highlighted that sexual function of male partners was unchanged or mildly improved after pelvic floor surgery [77].

Numerous studies are necessary to better understand the delicate relationship between female and male sexual dysfunctions; notwithstanding, we believe that it is essential to take the couple into care and not only the single patient, as long as it is possible.

References

1. Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. JAMA. 1999;281:537–44.
2. Spector IP, Carey MP. Incidence and prevalence of sexual dysfunctions: a critical review of the empirical literature. Arch Sex Behav. 1990;19:389–408.

3. Riley A, Riley E. Premature ejaculation: presentation and associations. An audit of patients. Int J Clin Pract. 2005;59:1482–7.
4. Byers ES, Grenier G. Premature or rapid ejaculation: heterosexual couples' perceptions of men's ejaculatory behaviour. Arch Sex Behav. 2003;32:261–70.
5. Giuliano F, Patrick DL, Porst H, La Pera G, Kokoszka A, Merchant S, Rothman M, Gagnon DD, Polverejan E. Premature ejaculation: results from a five-country European observational study. 3004 Study Group. Eur Urol. 2008;53(5):1048–57.
6. McCabe MP. Intimacy and quality of life among sexually dysfunctional men and women. J Sex Marital Ther. 1997;23:276–90.
7. Patrick DL, Althof SE, Pryor JL, Rosen R, Rowland DL, Ho KF, McNulty P, Rothman M, Jamieson C. Premature ejaculation: an observational study of men and their partners. J Sex Med. 2005;2(3):358–67.
8. Hisasue S, Kumamoto Y, Sato Y, Masumori N, Horita H, Kato R, et al. Prevalence of female sexual dysfunction symptoms and its relationship to quality of life: a Japanese female cohort study. Urology. 2005;65:143–8.
9. Abdo CHN, Olivera WM, Moreira ED, Fittipaldi JAS. Prevalence of sexual dysfunctions and correlated conditions in a sample of Brazilian women—results of the Brazilian study on sexual behaviour (BSSB). Int J Impot Res. 2004;16:160–6.
10. Hobbs K, Symonds T, Abraham L, May K, Morris MF. Sexual dysfunction in partners of men with premature ejaculation. Int J Impot Res. 2008;20:512–7; 2008.34.
11. Cayan S, Bozlu M, Canpolat B, Akbay E. The assessment of sexual functions in women with male partners complaining of erectile dysfunction: does treatment of male sexual dysfunction improve female partner's sexual functions? J Sex Marital Ther. 2004;30:333–41.
12. Herbenick D, Mullinax M, Mark KJ. Sexual desire discrepancy as a feature, not a bug, of long-term relationships: women's self-reported strategies for modulating sexual desire. J Sex Med. 2014;11(9):2196–206. Epub 2014 Jul 22.
13. Impett EA, Strachman A, Finkel EJ, Gable SL. Maintaining sexual desire in intimate relationships: the importance of approach goals. J Pers Soc Psychol. 2008;94(5):808–23.
14. Santtila P, Wager I, Witting K, Harlaar N, Jern P, Johansson A, Varjonen M, Sandnabba NK. Discrepancies between sexual desire and sexual activity: gender differences and associations with relationship satisfaction. J Sex Marital Ther. 2008;34(1):31–44.
15. Willoughby BJ, Vitas J. Sexual desire discrepancy: the effect of individual differences in desired and actual sexual frequency on dating couples. Arch Sex Behav. 2012;41(2):477–86. Epub 2011 May 14.
16. Sims KE, Meana M. Why did passion wane? A qualitative study of married women's attributions for declines in sexual desire. J Sex Marital Ther. 2010;36(4):360–80.
17. Davies S, Katz J, Jackson JL. Sexual desire discrepancies: effects on sexual and relationship satisfaction in heterosexual dating couples. Arch Sex Behav. 1999;28(6):553–67.
18. Mark K, Herbenick D, Fortenberry D, Sanders S, Reece M. The object of sexual desire: examining the "what" in "what do you desire?". J Sex Med. 2014;11(11):2709–19.
19. Mark KP, Murray SH. Gender differences in desire discrepancy as a predictor of sexual and relationship satisfaction in a college sample of heterosexual romantic relationships. J Sex Marital Ther. 2012;38(2):198–215.
20. Oliver MB, Hyde JS. Gender differences in sexuality: a meta-analysis. Psychol Bull. 1993; 114(1):29–51.
21. Bancroft J, Loftus J, Long JS. Distress about sex: a national survey of women in heterosexual relationships. Arch Sex Behav. 2003;32(3):193–208.
22. Hartley H. The "pinking" of Viagra culture: drug industry efforts to create and repackage sex drugs for women. Sexualities. 2006;9:363–78.
23. Tiefer L. A new view of women's sexual problems: why new? Why now? J Sex Res. 2001;38:89–96.
24. Bancroft J. The medicalization of female sexual dysfunction: the need for caution. Arch Sex Behav. 2002;31(5):451–5.
25. Chevret M, Jaudinot E, Sullivan K, Marrel A, De Gendre AS. Quality of sexual life and satisfaction in female partners of men with ED: psychometric validation of the Index of Sexual Life (ISL) questionnaire. J Sex Marital Ther. 2004;30:141–55.

26. Basson R, Althof S, Davis S, Fugl-Meyer K, Goldstein I, Leiblum S, et al. Summary of the recommendations on sexual dysfunctions in women. J Sex Med. 2004;1:24–34.
27. Campbell-Walsh urology tenth edition.
28. Althof SE. Prevalence, characteristics and implications of premature ejaculation/rapid ejaculation. J Urol. 2006;175:842–8.
29. Rowland DL, Patrick DL, Rothman M, Gagnon DD. The psychological burden of premature ejaculation. J Urol. 2007;177(3):1065–70.
30. Burri A, Giuliano F, McMahon C, Porst H. Female partner's perception of premature ejaculation and its impact on relationship breakups, relationship quality, and sexual satisfaction. J Sex Med. 2014;11(9):2243–55.
31. Miller SA, Byers ES. Actual and desired duration of foreplay and intercourse: discordance and misperceptions within heterosexual couples. J Sex Res. 2004;41(3):301–9.
32. Jannini EA, Porst H. A practical approach to premature ejaculation. Introduction: the asynchronous couple. J Sex Med. 2011;8 Suppl 4:301–3.
33. Buvat J, et al. Improvement in sexual functioning of female partners of men with premature ejaculation (PE) treated with dapoxetine (DPX). Eur Urol Suppl. 2008;7(3):186, ABS 464.
34. Riley A. The role of the partner in erectile dysfunction and its treatment. Int J Impot Res. 2002;14 Suppl 1:S105–9.
35. Greenstein A, Abramov L, Matzkin H, Chen J. Sexual dysfunction in women partners of men with erectile dysfunction. Int J Impot Res. 2006;18:44–6.
36. Leiblum SR. What every urologist should know about female sexual dysfunction. Int J Impot Res. 1999;11 Suppl 1:S39–40.
37. Zhang H, Fan S, Yip P. The association between female sexual dysfunction and the husband's erectile dysfunction: evidence from married couples in Hong Kong. J Sex Marital Ther. 2014;16:1–9.
38. Jiann BP, Su CC, Tsai JY. Is female sexual function related to the male partners' erectile function? J Sex Med. 2013;10(2):420–9.
39. Goldstein I, Fisher WA, Sand M, Rosen RC, Mollen M, Brock G, Karlin G, Pommerville P, Bangerter K, Bandel TJ, Derogatis LR. Women's sexual function improves when partners are administered vardenafil for erectile dysfunction: a prospective, randomized, double-blind, placebo-controlled trial. Vardenafil Study Group. J Sex Med. 2005;2(6):819–32.
40. Heiman JR, Talley DR, Bailen JL, Oskin TA, Rosenberg SJ, Pace CR, Creanga DL, Bavendam T. Sexual function and satisfaction in heterosexual couples when men are administered sildenafil citrate (Viagra) for erectile dysfunction: a multicentre, randomised, double-blind, placebo-controlled trial. BJOG. 2007;114(4):437–47. Epub 2007 Feb 5.
41. Levine SB, Bodner D, Kursh ED, Resnick MI, Wittmann. Through the eyes of women: the sexual and psychological responses of women. J Urol. 1992;147(4):1024–7.
42. Wittmann D, Carolan M, Given B, Skolarus T, Crossley A, Lawrence A, Clark P, Montie JE. What couples say about their recovery of sexual intimacy after prostatectomy: toward the development of a conceptual model of couples' sexual recovery after surgery for prostate cancer. J Sex Med. 2015;12(2):494–504.
43. Kershaw TS, Mood DW, Newth G, Ronis DL, Sanda MG, Vaishampayan U, Northouse LL. Longitudinal analysis of a model to predict quality of life in prostate cancer patients and their spouses. Ann Behav Med. 2008;36(2):117–28.
44. Harden JK, Northouse LL, Mood DW. Qualitative analysis of couples' experience with prostate cancer by age cohort. Cancer Nurs. 2006;29(5):367–77.
45. Crowe H, Costello AJ. Prostate cancer: perspectives on quality of life and impact of treatment on patients and their partners. Urol Nurs. 2003;23(4):279–85.
46. Kornblith AB, Herr HW, Ofman US, Scher HI, Holland JC. Quality of life of patients with prostate cancer and their spouses. The value of a data base in clinical care. Cancer. 1994;73(11):2791–802.
47. Beck AM, Robinson JW, Carlson LE. Sexual intimacy in heterosexual couples after prostate cancer treatment: what we know and what we still need to learn. Urol Oncol. 2009;27(2):137–43.

48. Galbraith ME, Fink R, Wilkins GG. Couples surviving prostate cancer: challenges in their lives and relationships. Semin Oncol Nurs. 2011;27(4):300–8.
49. Bruun P, Pedersen BD, Osther PJ, Wagner L. The lonely female partner: a central aspect of prostate cancer. Urol Nurs. 2011;31(5):294–9.
50. Sanders S, Pedro LW, Bantum EO, Galbraith ME. Couples surviving prostate cancer: long-term intimacy needs and concerns following treatment. Clin J Oncol Nurs. 2006;10(4): 503–8.
51. Wootten AC, Burney S, Foroudi F, Frydenberg M, Coleman G, Ng KT. Psychological adjustment of survivors of localised prostate cancer: investigating the role of dyadic adjustment, cognitive appraisal and coping style. Psychooncology. 2007;16(11):994–1002.
52. Garos S, Kluck A, Aronoff D. Prostate cancer patients and their partners: differences in satisfaction indices and psychological variables. J Sex Med. 2007;4(5):1394–403.
53. Neese LE, Schover LR, Klein EA, Zippe C, Kupelian PA. Finding help for sexual problems after prostate cancer treatment: a phone survey of men's and women's perspectives. Psychooncology. 2003;12(5):463–73.
54. Canada AL, Neese LE, Sui D, Schover LR. Pilot intervention to enhance sexual rehabilitation for couples after treatment for localized prostate carcinoma. Cancer. 2005;104(12): 2689–700.
55. Schover LR, Canada AL, Yuan Y, Sui D, Neese L, Jenkins R, Rhodes MM. A randomized trial of internet-based versus traditional sexual counseling for couples after localized prostate cancer treatment. Cancer. 2012;118(2):500–9.
56. Manne S, Badr H. Intimacy and relationship processes in couples' psychosocial adaptation to cancer. Cancer. 2008;112(11 Suppl):2541–55.
57. WHO SHDS. Defining sexual health: a report of a technical consultation on sexual health. Sexual health document series. Vol. 2002 World Health Organization; Jan 28–31, 2002.
58. Bober SL, Varela VS. Sexuality in adult cancer survivors: challenges and intervention. J Clin Oncol. 2012;30(30):3712–9.
59. Tierney DK. Sexuality: a quality-of-life issue for cancer survivors. Semin Oncol Nurs. 2008;24(2):71–9.
60. Reese JB, Keefe FJ, Somers TJ, Abernethy AP. Coping with sexual concerns after cancer: the use of flexible coping. Supportive Care Cancer Off J Multinatl Assoc Supportive Care Cancer. 2010;18(7):785–800.
61. Basson R. A model of women's sexual arousal. J Sex Marital Ther. 2002;28(1):1–10.
62. Shindel A, Quayle S, Yan Y, Husain A, Naughton C. Sexual dysfunction in female partners of men who have undergone radical prostatectomy correlates with sexual dysfunction of the male partner. J Sex Med. 2005;2(6):833–41. Discussion 841.
63. Cliff AM, MacDonagh RP. Psychosocial morbidity in prostate cancer: II. A comparison of patients and partners. BJU Int. 2000;86(7):834–9.
64. Banthia R, Malcarne VL, Varni JW, Ko CM, Sadler GR, Greenbergs HL. The effects of dyadic strength and coping styles on psychological distress in couples faced with prostate cancer. J Behav Med. 2003;26(1):31–52.
65. Valdimarsdóttir U, Helgason AR, Fürst CJ, Adolfsson J, Steineck G. Awareness of husband's impending death from cancer and long-term anxiety in widowhood: a nationwide follow-up. Palliat Med. 2004;18(5):432–43.
66. Campbell LC, Keefe FJ, McKee DC, Edwards CL, Herman SH, Johnson LE, Colvin OM, McBride CM, Donattuci CF. Prostate cancer in African Americans: relationship of patient and partner self-efficacy to quality of life. J Pain Symptom Manage. 2004;28(5):433–44.
67. Germino BB, Mishel MH, Belyea M, Harris L, Ware A, Mohler J. Uncertainty in prostate cancer. Ethnic and family patterns. Cancer Pract. 1998;6(2):107–13. Erratum in: Cancer Pract. 1998;6(4):205.
68. Phillips C, Gray RE, Fitch MI, Labrecque M, Fergus K, Klotz L. Early postsurgery experience of prostate cancer patients and spouses. Cancer Pract. 2000;8(4):165–71.
69. Ptacek JT, Pierce GR, Dodge KL, Ptacek JJ. Social support in spouses of cancer patients: what do they get and to what end? Pers Relat. 1997;4:431–49.

70. Boehmer U, Clark JA. Communication about prostate cancer between men and their wives. J Fam Pract. 2001;50(3):226–31.
71. EAU guidelines 2014 edition.
72. Khademi A, Alleyassin A, Amini M, Ghaemi M. Evaluation of sexual dysfunction prevalence in infertile couples. J Sex Med. 2008;5(6):1402–10.
73. Kucur Suna K, Ilay G, Aysenur A, Kerem Han G, Eda Ulku U, Pasa U, Fatma C. Effects of infertility etiology and depression on female sexual function. J Sex Marital Ther. 2016;42(1): 27–35. [Epub 2015 Jan 28].
74. Millheiser LS, Helmer AE, Quintero RB, Westphal LM, Milki AA, Lathi RB. Is infertility a risk factor for female sexual dysfunction? A case-control study. Fertil Steril. 2010;94(6): 2022–5.
75. Pakpour AH, Yekaninejad MS, Zeidi IM, Burri A. Prevalence and risk factors of the female sexual dysfunction in a sample of infertile Iranian women. Arch Gynecol Obstet. 2012;286(6): 1589–96.
76. Soltani M, Shairi MR, Roshan R, Rahimi CR. The impact of emotionally focused therapy on emotional distress in infertile couples. Int J Fertil Steril. 2014;7(4):337–44.
77. Lonnée-Hoffmann RA, Salvesen O, Mørkved S, Schei B. Male sexual function and pelvic floor surgery of their female partner: a one-year follow-up study. Post Reprod Health. 2014;20(2):55–61.

Female Sexuality in Chronic Pelvic Pain

14

Antonella Giannantoni

Abbreviations

BPS/IC	Bladder pain syndrome-interstitial cystitis
CEDS	Center for Epidemiologic Studies Depression Scale
DMSO	Dimethyl sulfoxide
FSFI	Female Sexual Function Index
ICPI	Interstitial Cystitis Problem Index
ICSI	Interstitial Cystitis Symptom Index
MCS	Mental Composite Scale
MGP	McGill Pain
QoL	Quality of Life
PSS	Perceived Stress Scale
PUF	Pelvic Pain and Urgency/Frequency Questionnaire
STAI	State-Trait Anxiety Inventory
UPOINT	Urinary Psychosocial, Organ Specific, Infection, Neurologic or Systemic, and Tenderness
VAS	Visual Analogue Scale

14.1 Introduction

BPS/IC is a chronic disorder diagnosed on the basis of chronic pelvic pain, pressure, or discomfort perceived to be related to the urinary bladder accompanied by at least one urinary symptom such as persistent urge to void or urinary frequency

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_14

[10, 45]. The etiology is still unclear, and different factors have been retained to be the cause of the disease, as infections, autoimmunity, mucosal abnormalities, urinary toxins, and local neuronal dysfunction [1]. None have persuasively explained the syndrome, and the lack of understanding of its pathogenesis has led to empiric and often inadequate therapies [47]. Due to many different clinical presentations, it is retained that patients with BPS/IC do not represent a homogenous group with perceived bladder pain associated with urinary frequency and urgency, but rather a group of individual patients with widely differing clinical phenotypes [22].

Sexual dysfunction symptoms have been reported for women with disorders related to BPS/IC, including lack of libido and of sexual interest, pain during intercourse, and vulvodynia [1].

The knowledge that sexual dysfunction may be present in patients with BPS/IC and can contribute to a reduced Quality of Life (QoL) has been only recently acquired.

14.2 Epidemiology

Research has found significantly worse sexual functioning among patients with BPS/IC than among the general population or age-matched control patients for several different sexual functioning domains, including a lack of desire, reduced arousal, lubrication difficulties, low orgasm frequency, dissatisfaction, and pain [25, 28].

The exact prevalence of sexual dysfunction among women with BPS/IC is unknown. When comparing prevalence rates of sexual dysfunction between women in the general population in the United States and those affected by the diseases, it was observed that about 40 % of women in the general population have reported at least one sexual dysfunction, such as a lack of interest (31 %), arousal difficulties (19 %), inability to achieve orgasm (25 %), performance anxiety (12 %), pain (15 %), and lack of pleasure (23 %) [7, 11, 14, 16, 39]. In convenient samples of women with BPS/IC, the range of dysfunction across studies has been found to range between 13 and 87 % ([21, 27, 29, 30, 42]). The results of the RICE study, the first population-based representative sample of women with BPS/IC symptoms, showed that women with the disease experience very high levels of sexual dysfunction compared with the general population.

In a previous study from Bogart et al., 88 % of women with a current partner complained of ≥1 general sexual dysfunction symptom in the previous 4 weeks, and 90 % of those with a current partner reported any BPS/IC -specific sexual dysfunction symptoms during the same period [2]. A lack of sexual interest and bladder pain during and/or after sex were the most prevalent symptoms, experienced by about two thirds of the women [2]. In a mailed survey sent to 5000 randomly selected women from the United States and 407 women with BPS/IC from a large referral center, a significantly greater proportion of patients (67.2 %) than controls (18 %)

reported having pelvic pain during intercourse [29, 30]. Fear of having pain during sexual intercourse was reported significantly more by 108 cases of 215 than controls 111 of 823 ($p=0.001$), and in those having intercourse, dyspareunia was reported more by the 132 cases of 177 than controls 193 of 646 ($p=0.001$) [29, 30]. Before the BPS/IC diagnosis, 86 % of cases recalled having moderate to high sexual desire compared with 78 % of the controls ($p=0.016$). After the BPS/IC diagnosis, sexual desire in cases with moderate-high severity of the disease declined from 86 to 40 %. The percentage of BPS/IC patients reporting orgasm as frequently or very frequently was lower (44 % among the cases). Interestingly, in this study, women with BPS/IC had significantly more pain and fear of pain during intercourse even in adolescence [29, 30].

Many studies reported that sexual pain in BPS/IC patients is a strong predictor of poor QoL [23] and that up to 54 % of these patients avoid intercourse with their partners most of the time [49]. In a recent case-control study, comparing 554 patients with BPS/IC and 131 controls, patients reported significant sexual dysfunction in all domains of the FSFI, with pain as the most significant finding [53]. Alterations in arousal, lubrication, and pain were most notable in patients younger than 30 years, while patients older than 50 experienced less distress in relation to their sexual dysfunction caused by BPS/IC [29, 30, 53]. In a survey of 565 patients with BPS/IC, more patients reported that their pain localized to the vaginal area (60.8 %), than to the lower abdominal (56.7 %) or suprapubic region (53.2 %) [13]. Over a third of patients also reported experiencing pain for days after intercourse [13].

14.3 Risk Factors

Sexual abuse. Almost no research has been conducted regarding the risk factors for sexual dysfunction among women with BPS/IC or whether women with BPS/IC receive medical help for sexual dysfunction issues. In a study of chronic pelvic pain and its relationship to prior sexual or physical abuse, subjects with a history of abuse reported more severe pain intensity compared to those without prior abuse even when there was no apparent objective medical basis for the difference in pain intensity [17]. In the study of Seth and Teichman [38], the subjects with BPS/IC with a sexual abuse history score worse on multiple FSFI domains and exhibited more tender areas on the abdominal and pelvic floor examination compared to those with PBS/IC without a history of abuse. Increased rates of adult chronic pain in patients with prior sexual abuse persist even after controlling for concurrent depression [3]. It is thought that sexual traumatization leads to dysregulation in the hypothalamic-pituitary-adrenal axis and increased autonomic activation. If prior sensitization of these central pathways occurs from sexual traumatization, patients with subsequent PBS/IC might have a more easily upregulated central nervous system compared to those without such prior sensitization [38]. A recent study reported that 18–33 % of patients with PBS/IC have a history of sexual abuse [29, 30].

14.3.1 Psychological Status: Anxiety and Depression

Several somatic and psychological predictors are associated with poorer BPS/IC QoL. In previous epidemiological studies, 55.8 % of BPS/IC patients presented with depression and 29.8 % showed feelings of worthlessness or helplessness. In these patients, depressive state has been found to be associated with maladjusted coping strategies for self-managing pain such as catastrophizing [43]. Catastrophizing is defined as the tendency to mentally focus on pain and evaluate one's ability to manage pain negatively [41]. Catastrophizing affects the QoL and reported pain severity of male and female patients with chronic pelvic pain [35]. Pain and reduced sexual functioning have been observed to be associated with poorer psychological functioning in other chronic pain conditions [6, 48]. Nickel et al. found that sexual functioning was the only predictor of poorer mental QoL in BPS/IC patients [23]. In a previous study from Tripp et al. [43], helplessness catastrophizing was associated with reduced MCS QoL more than the other independent variables. In addition, a high level of association between depression and MCS QoL was identified, suggesting that these constructs are basically assessing the same domain. From a medical perspective, focused assessments of psychological and medical predictors before treatment may be helpful. Identifying catastrophizing could help physicians in selecting the most efficient treatment, particularly the adjunctive treatment of patients with a cognitive behavioral intervention. From a neurophysiological point of view, chronic inflammation induces pain following chronic changes in neurotransmission, mechanisms of pain control, and tissue responses. These stimulatory factors affect the mechanisms associated with pain. A persistent presence of these stimulations converts the pelvic pain to neurological pain (which eventually leads to chronic pain syndrome) contributing to the impairment of sexual function [9].

14.3.2 Etiopathogenesis of Sexual Pain

Dyspareunia in association with BPS/IC may have a variety of causes. Resistance to penetration resulting from fear of pain may cause pelvic floor hypertonus, restricting vaginal entry and both dyspareunia and mechanical trauma of the vestibular mucosa and urethra [9]. Mechanical irritation of the urethra and/or bladder during intercourse may cause discomfort and exacerbate symptoms of PBS/IC. The pelvic floor and the bladder share common nerve pathways which may also result in shared symptoms. During sexual intercourse, physical irritation to the urethra and urinary bladder causes discomfort and increases the symptoms of BPS/IC.

14.4 Clinical Presentation

Sexual pain or dyspareunia, which is pain experienced during or after intercourse, may be due to an organic or psychological factor [37]. BPS/IC is a potential cause of sexual pain and should be considered in the differential diagnosis of dyspareunia.

Women with BPS/IC may present with the combination of dyspareunia and chronic pelvic pain as chief complains or the symptom of sexual pain can be discovered by the healthcare physician during the history. In a survey of 138 patients with BPS/IC, dyspareunia has been reported by 51 % with a larger proportion (79 %) indicating that sexual intercourse increased their pain [48]. Patients with BPS/IC and sexual dysfunction cannot be easily recognized, particularly in the early stages of the disease. The combination of chronic pelvic pain with urinary symptoms, as increased daytime and nighttime frequency and urgency, associated with vulvar pain or dyspareunia, are the typical presentation picture. Although patients with BPS/IC often present with only one symptom and may not develop the full spectrum of symptoms for several years, more typically symptoms present in a pattern of recurrences followed by remissions, with a gradual progression to a more severe condition along time [51]. In a previous study, 40.3 % of patients with BPS/IC presented with urinary incontinence during sexual intercourse [52]. Considering that BPS/IC patients present with different clinical phenotypes, multiple aspects of the disease should be considered in the clinical presentation and in the therapeutic program for these patients. Nickel et al. developed the UPOINT system to categorize the phenotype of BPS/IC patients into six clinically identifiable domains [22]. It was observed that the number of positive UPOINT domains significantly correlated with the severity of BPS/IC symptoms, as measured using the ICSI questionnaire and pain VAS scores. The UPOINT system does not include a sexual domain, although sexual pain is present in 88 % of patients with BPS/IC. Recently, Liu et al. demonstrated that sexual dysfunction is an important component of BPS/IC phenotype, and adding a sexual dysfunction domain to the UPOINT system improves the association with BPS/IC symptom severity [15]. BPS/IC may be the sole cause of symptoms or occur concurrently with other causes such as endometriosis or myofascial pain [4].

Endometriosis is a common cause of chronic pelvic pain in women and is diagnosed in up to 80 % of these patients. However, increasing evidence suggests that endometriosis in BPS/IC patients with chronic pelvic pain is more prevalent than was previously thought and may play a significant role in the cause of the disease. Symptoms commonly associated with endometriosis include perimenstrual lower abdominal pain and dyspareunia. In addition, patients with endometriosis can also present with dysuria, hematuria, urinary frequency, and painful voiding, particularly if the bladder is involved. When therapy for the symptoms of endometriosis is unsuccessful, it may be thought that BPS/IC may be an underlying cause [4].

Other common differential diagnoses for sexual pain due to BPS/IC are vaginismus, vulvar vestibulitis, vulvodynia, atrophic tissue or impaired lubrication, adnexal pathology, chronic cervicitis, pelvic inflammatory disease, and urethra disorders.

14.5 Diagnosis

The baseline evaluation includes history and physical and laboratory examination in order to assess the presence of BPS/IC and exclude other diseases with similar symptoms. Urinary symptoms and pain level should be adequately investigated and

assessed. Pain body maps can be used in patients whose presentation suggests a more global pain syndrome [10]. Most important, any eventual confusable disease should be diagnosed and excluded. In this respect, infection, bladder stone, bladder neck obstruction, overactive bladder, carcinoma of the bladder, endometriosis, urogenital prolapse, and cervical, uterine, and ovarian cancer have to be excluded with appropriate investigations [45]. One crucial point is an appropriate patient's symptom evaluation. It is important to have a uniform and reliable method to evaluate symptoms, and, in this regard, the use of standardized questionnaires is of extreme usefulness.

14.5.1 Condition-Specific Symptom Questionnaires

ICSI and ICPI questionnaires. These questionnaires have been designed to capture the most important voiding and pain symptoms and to assess how patients find them in the clearest and most concise manner possible [24]. Both instruments have been designed and validated for self-administration, so that trained interviewers are not needed to use them. Psychometric performance of both instruments is good, with the symptom index demonstrating excellent ability to discriminate characteristics between patients and controls [31].

MPQ questionnaire. Also in a more recent short form, this questionnaire provides quantitative information that can be used statistically, and it appears sufficiently sensitive to detect differences among different methods to relieve pain. It measures functional physical component status and mental component status [18, 19]. The MPQ has been used in the assessment of multiple types of acute and chronic pain, and its reliability and validity have been extensively documented.

PUF questionnaire. It is a symptom scale that gives balanced attention to urinary urgency/frequency, pelvic pain, and symptoms associated with sexual intercourse [26].

14.5.2 Psychosocial Parameters

Depressive symptoms can be assessed using the CES-D [32] and anxiety symptoms with the Trait Anxiety Scale of the STAI [40].

The PSS can be used to assess stress [5] and the FSFI is used to measure sexual functioning [34]. The Pain Catastrophizing Scale is used to measure catastrophizing cognitions concerning pain [41].

General questionnaires focused on sexual function include the FSFI and Sexual History Form 12, which were specifically designed to evaluate sexual function [34, 46]. These questionnaires have undergone validation and reliability testing in a general population. Although FSFI questionnaire provides overall metric and specific subscales of sexual functioning, including sexual pain, it is not condition specific and may not be sensitive enough to detect differences due to BPS/IC.

Quality-of-Life Questionnaires, such as the King's Health Questionnaire and the Incontinence Impact Questionnaire [33, 44], include a few questions addressing

sexual function but really deal with the overall impact of incontinence and/or prolapse on the patient's QoL or well-being and do not focus on sexual function.

14.6 Management Strategies for Patients with BPS/IC and Sexual Pain

According to some studies, treatment of patients with PBS/IC results in improved sexual function and quality of life. Nickel et al. reported that sexual function was the key factor in determining the quality of life in elderly patients with the disease [23]. The authors also suggested the goal of treatment should focus on sexual dysfunction and that the improvement of symptoms of PBS/IC should accompany improvement in sexual function. In patients with sexual dysfunction presenting with BPS/IC, treatment should be approached in many directions, including psychological and social aspects. Patients should be educated that BPS/IC is a chronic condition associated with relapsing and remission phases and that improvement usually occurs gradually. It is important to schedule regular follow-up visit thus giving the patient a sense of attention and more comprehensive cure. As the etiology of the disease appears to be multifactorial, a multimodal approach seems to be more appropriate.

Dietary modification may be useful to reduce symptoms in many patients. There are several "trigger foods" that can vary for each individual patients and a "food diary" is an important tool to use during an elimination diet. It will help the patient keep track of what he/she is eating and determine reaction to specific foods and beverages and whether there is an allergy or a sensitivity. Alcohol, artificial sweeteners (aspartame and saccharin), coffee, citrus juices, cranberry juice, hot peppers, soda, and spicy foods are the most bothersome foods for people with BPS/IC. Changes in diet and lifestyle can help reduce symptoms, and *bladder retraining* my reduce voiding frequency in some patients with the disease. When the patients' symptoms do not respond to nonpharmacological treatment, it is necessary to move toward pharmacological interventions, which often should be given as a combination of drugs. A combination of oral and intravesical therapies together with lifestyle intervention and physical therapy can gradually improve pain and urinary symptoms in patients affected by BPS/IC. Early treatment intervention may prevent the progression of the disease to more severe stages, when tissue damage is permanent and the BPS/IC became a chronic condition.

14.6.1 Self-Care for Patients with BPS/IC and Sexual Pain

There are many self-care approaches that can help women and their partners achieve some levels of sexual intimacy and give her a sense of control over her sexual life. Alternatives to sexual intercourse or different coital positions, assuming drugs to relax pelvic muscles before intercourse, may help to reduce pain during sexual activity. Patients should keep a bladder diary to record any triggers for symptom flares. In addition they should note any event which is linked to symptom

exacerbations. Vaginal lubricants could be useful in reducing dyspareunia due to friction to the urethra. Precoital or postcoital voiding or the use of ice packs to the suprapubic area or genitalia can alleviate pain symptoms during or after intercourse [51].

14.6.2 Pharmacological Treatment

Several pharmacological agents have been used in the treatment of patients with BPS/IC. Only few of these drugs have been approved by the Food and Drug Administration, as pentosan polysulfate sodium orally given or intravesical DMSO. Other oral or intravesical agents may be used "off label." Nickel et al. conducted a study in 128 patients who underwent medical treatment with oral pentosan polysulfate 300 mg/day for 32 weeks [21]. Patients reported statistically significant improvements in pain and in sexual functioning scores after treatment, and symptom improvement was moderately related with improvements in sexual dysfunction. Similar results were observed by Hung et al., who treated patients with BPS/IC and sexual dysfunction with intravesical hyaluronic acid solution [12]. In their study, the Pelvic Organ Prolapse/Urinary Incontinence Sexual Function Questionnaire total scores improved significantly from baseline after 1 month and 6 months of treatment [12].

Welk et al. treated 23 sexually active patients with BPS/IC with an intravesical solution constituted by lidocaine, bicarbonate, and heparin [50]. All patients completed the Pelvic Pain Urgency Frequency symptom scale, voiding diary and the pain domain (questions 17–19) of the FSFI questionnaire before and after therapy. Intravesical instillations were repeated 3 times weekly for 3 weeks. Of the 23 patients, 13 (57 %) reported resolution of dyspareunia. The results of this study demonstrated that an intravesical multidrug solution provides relief of voiding symptoms, pain, and dyspareunia in patients with BPS/IC. In a prospective case-control study conducted in a tertiary referral center, 69 patients with BPS/IC were treated applying determined therapeutical steps including tetracycline, bladder instillation consisting of heparin, local anesthetic and natrium-bicarbonate, prednisolone and antihistaminics, or instillation with DMSO [36]. Pain, nocturia, and urinary frequency and urgency were significantly reduced after treatment. The King's Health Questionnaire showed a significant improvement of all domains but emotions and sleep, and FSFI improved significantly in all domains but orgasm [36]. Recently, Nickel et al. proposed the use of flexible therapeutic strategy for patients referred to a specialized BPS/IC clinic using an individualized phenotype-directed treatment plan based on clinically based UPOINT categorization [20]. They found that almost 50 % of patients, regardless of the complexity or severity of condition, experienced clinically significant improvement using an individualized phenotype-directed therapeutic approach [20].

Other studies proposed many kinds of different approaches, but, because of the paucity of randomized placebo-controlled studies on different treatments, an evidence-based management approach has not yet been developed [8], and limited

evidence exists for the few treatments for BPS/IC. The lack of definitive conclusions is due to the great heterogeneity in methodology, symptom assessment, duration of treatment, and follow-up in both RCTs and nRCTs. In addition, few of the previously published studies considered specifically improvements in sexual activity after treatment.

Conclusions

Sexual dysfunction is a common symptom in women affected by BPS/IC, but it has been only recently recognized, although it highly affects the QoL of patients. Prompt recognition and intervention of BPS/IC are a determinant factor for a successful treatment in these women, in order to alleviate both pain and sexual dysfunction.

References

1. Bogart LM, Berry SH, Clemens JQ. Symptoms of interstitial cystitis bladder pain syndrome and similar diseases in women: a systematic review. J Urol. 2007;177:450–6.
2. Bogart LM, Suttorp MJ, Elliott MN, et al. Prevalence and correlates of sexual dysfunction among women with bladder pain syndrome/interstitial cystitis. Urology. 2011;77:576–80.
3. Brown J, Berenson K, Cohen P. Documented and self-reported child abuse and adult pain in a community sample. Clin J Pain. 2005;21:374–7.
4. Butrick CW. Patients with chronic pelvic pain: endometriosis or interstitial cystitis/painful bladder syndrome? JSLS. 2007;11:182–9.
5. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. J Health Soc Behav. 1983;24:385.
6. Flor H, Turk DC, Scholz OB. Impact of chronic pain on the spouse: marital, emotional and physical consequences. J Psychosom Res. 1987;31:63–71.
7. Gardella B, Porru D, Ferdeghini F, et al. Insight into urogynecologic features of women with interstitial cystitis/bladder pain syndrome. Eur Urol. 2008;54:1145–53.
8. Giannantoni A, Bini V, Dmochowski R, Hanno P, Nickel JC, Proietti S, Wyndaele JJ. Contemporary management of the painful bladder: a systematic review. Eur Urol. 2012;61:29–53.
9. Graziottin A. Sexual pain disorders: dispareunia and vaginismus. In: Porst H, Buvat J, editors. International society of sexual medicine standard committee book: standard practice in sexual medicine. 1st ed. Oxford: Blackwell; 2006. p. 342–50.
10. Hanno PM, Burks DA, Clemens JQ, Dmochowski RR, Erickson D, Fitzgerald MP, Forrest JB, Gordon B, Gray M, Mayer RD, Newman D, Nyberg Jr L, Payne CK, Wesselmann U, Faraday MM, Interstitial Cystitis Guidelines Panel of the American Urological Association Education and Research, Inc. AUA guideline for the diagnosis and treatment of interstitial cystitis/bladder pain syndrome. J Urol. 2011;185:2162–70.
11. Held PJ, Hanno PM, Wein AJ, Pauli MV, Cahn MA. Epidemiology of interstitial cystitis. In: Hanno PM, Staskin DR, Krane RJ, Wein AJ, editors. Interstitial cystitis. London: Springer; 1990. p. 29–48.
12. Hung MJ, Su TH, Lin YH, Huang WC, Lin TY, Hsu CS, Chuang FC, Tsai CP, Shen PS, Chen GD. Changes in sexual function of women with refractory interstitial cystitis/bladder pain syndrome after intravesical therapy with a hyaluronic acid solution. J Sex Med. 2014;11:2256–63.
13. Koziol JA. Epidemiology of interstitial cystitis. Urol Clin North Am. 1994;21:7–20.
14. Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. JAMA. 1999;281:537–44.

15. Liu B, Su M, Zhan H, Yang F, Li W, Zhou X. Adding a sexual dysfunction domain to UPOINT system improves association with symptoms in women with interstitial cystitis and bladder pain syndrome. Urology. 2014;84:1308–13.
16. Lutfey KE, Link CL, Rosen RC, et al. Prevalence and correlates of sexual activity and function in women: results from the Boston Area Community Health (BACH) Survey. Arch Sex Behav. 2009;38:514–27.
17. Meltzer-Brody S, Leserman J, Zolnoun D, Steege J, Green E, Teich A. Trauma and posttraumatic stress disorder in women with chronic pelvic pain. Obstet Gynecol. 2007;109:902–8.
18. Melzack R. The McGill Pain Questionnaire: major properties and scoring methods. Pain. 1975;1:277–99.
19. Melzack R. The short-form McGill Pain Questionnaire. Pain. 1987;30:191–7.
20. Nickel JC, Irvine-Bird K, Jianbo L, Shoskes DA. Phenotype-directed management of interstitial cystitis/bladder pain syndrome. Urology. 2014;84:175–9.
21. Nickel JC, Parsons CL, Forrest J, et al. Improvement in sexual functioning in patients with interstitial cystitis/bladder pain syndrome. J Sex Med. 2008;5:394–9.
22. Nickel CJ, Shoskes D, Irvine-Bird K. Clinical phenotyping of women with interstitial cystitis/painful bladder syndrome: a key to classification and potentially improved management. J Urol. 2009;182:155–60.
23. Nickel JC, Tripp D, Teal V, Propert KJ, Burks D, Foster HE, Hanno P, Mayer R, Payne CK, Peters KM, Kusek JW, Nyberg LM, Interstitial Cystitis Collaborative Trials Group. Sexual function is a determinant of poor quality of life for women with treatment refractory interstitial cystitis. J Urol. 2007;177:1832–6.
24. O'Leary MP, Sant GR, Fowler Jr FJ, Whitmore KE, Spolarich-Kroll J. The interstitial cystitis symptom index and problem index. Urology. 1997;49:58–63.
25. Ottem DP, Carr LK, Perks AE, et al. Interstitial cystitis and female sexual dysfunction. Urology. 2007;69:608–10.
26. Parsons CL, Dell J, Stanford EJ, Bullen M, Khan BS, Waxell T, Koziol JA. Increased prevalence of interstitial cystitis: previously unrecognized urologic and gynecologic cases indentified using a new questionnaire and intravesical potassium sensitivity. Urology. 2002;60:573–8.
27. Patel R, Calhoun EA, Meenan RT, et al. Incidence and clinical characteristics of interstitial cystitis in the community. Int Urogynecol Pelvic Floor Dysfunct. 2008;19:1093–6.
28. Peters KM, Carrico DJ, Ibrahim IA, et al. Characterization of a clinical cohort of 87 women with interstitial cystitis/painful bladder syndrome. Urology. 2008;71:634–40.
29. Peters KM, Kalinowski SE, Carrico DJ, Ibrahim IA, Diokno AC. Fact or fiction—is abuse prevalent in patients with interstitial cystitis? Results from a community survey and clinic population. J Urol. 2007;178:891.
30. Peters KM, Killinger KA, Carrico DJ, et al. Sexual function and sexual distress in women with interstitial cystitis: a case-control study. Urology. 2007;70:543–7.
31. Quaghebeur J, Wyndaele JJ. Comparison of questionnaires used for the evaluation of patients with chronic pelvic pain. Neurourol Urodyn. 2013;32:1074–9.
32. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. Appl Psych Meas. 1977;1:385–401.
33. Reese PR, Pleil AM, Okano GJ, Kelleher CJ. Multinational study of reliability and validity of the King's Health Questionnaire in patients with overactive bladder. Qual Life Res. 2003;12:427–42.
34. Rosen R, Brown C, Heiman J, Leiblum S, Meston C, Shabsigh R, Ferguson D, D'Agostino Jr R. The Female Sexual Function Index (FSFI): a multidimensional self-report instrument for the assessment of female sexual function. J Sex Marital Ther. 2000;26:191–208.
35. Rothrock NE, Lutgendorf SK, Kreder KJ. Coping strategies in patients with interstitial cystitis: relationships with quality of life and depression. J Urol. 2003;169:233–6.
36. Schmid C, Berger K, Müller M, Silke J, Mueller MD, Kuhn A. Painful bladder syndrome: management and effect on sexual function and quality of life. Ginekol Pol. 2011;82:96–101.

37. Schulz WW, Basson R, Binik Y, Eschenbach D, Wesselmann U, Van lankveld J. Women's sexual pain and its management. J Sex Med. 2005;2:301–16.
38. Seth A, Teichman JM. Differences in the clinical presentation of interstitial cystitis/painful bladder syndrome in patients with or without sexual abuse history. J Urol. 2008;180:2029–33.
39. Shifren JL, Monz BU, Russo PA, et al. Sexual problems and distress in United States women: prevalence and correlates. Obstet Gynecol. 2008;112:970–8.
40. Speilberger CD, Gorusch RL, Lushene R, et al. Manual for the State-Trait Anxiety Inventory (Form Y1). Palo Alto: Consulting Psychologists Press; 1983.
41. Sullivan MJL, Bishop SR, Pivik J. The pain catastrophizing scale: development and validation. Psychol Assess. 1995;7:524–53.
42. Tincello DG, Walker AC. Interstitial cystitis in the UK: results of a questionnaire survey of members of the interstitial cystitis support group. Eur J Obstet Gynecol Reprod Biol. 2005;118:91–5.
43. Tripp DA, Nickel JC, Fitzgerald MP, Mayer R, Stechyson N, Hsieh A. Sexual functioning, catastrophizing, depression, and pain, as predictors of quality of life in women with interstitial cystitis/painful bladder syndrome. Urology. 2009;73:987–92.
44. Uebersax JS, Wyman JF, Shumaker SA, McClish DK. Short forms to assess life quality and symptom distress for urinary incontinence in women: the incontinence impact questionnaire and the urogenital distress inventory. Neurourol Urodyn. 1995;2:131–9.
45. van de Merwe JP, Nordling J, Bouchelouche P, et al. Diagnostic criteria, classification, and nomenclature for painful bladder syndrome/interstitial cystitis: an ESSIC proposal. Eur Urol. 2008;53:60–7.
46. Ware JE, Kosinski M, Keller SA. 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. Med Care. 1996;34:220–33.
47. Warren JW. Bladder pain syndrome/interstitial cystitis as a functional somatic syndrome. J Psychosom Res. 2014;77:510–5.
48. Webster DC. Recontextualizing sexuality in chronic illness: women and interstitial cystitis. Health Care Women Int. 1997;18:575–89.
49. Webster DC, Brennan T. Use and effectiveness of sexual self-care strategies for interstitial cystitis. Urol Nurs. 1995;15:14–22.
50. Welk BK, Teichman JM. Dyspareunia response in patients with interstitial cystitis treated with intravesical lidocaine, bicarbonate, and heparin. Urology. 2008;71:67–70.
51. Whitmore K, Siegel JF, Kellogg-Spadt S. Interstitial cystitis/painful bladder syndrome as a cause of sexual pain in women: a diagnosis to consider. J Sex Med. 2007;4:720–7.
52. Yoon HS, Yoon H. Correlations of interstitial cystitis/painful bladder syndrome with female sexual activity. Korean J Urol. 2010;51:45–9.
53. Zaslau S, Riggs DR, Perlmutter AE, Jackson BJ, Osborne J, Kandzari SJ. Sexual dysfunction in patients with painful bladder syndrome is age related and progressive. Can J Urol. 2008;15(4):4158–62.

Female Sexual Function and Neurological Disease

15

Elena Andretta

Chronic neurological disease can have a tremendous impact on a woman's health, self-image, and consequently on self-esteem [1]. One of the most important areas that could be impaired after the onset of a neurological disease is sexual function.

Lesions in any level of the central nervous system (brain, spinal cord, and peripheral nerves) could impact on sexual function and could diminish a woman's libido and ability to become sexual aroused, finally preventing orgasm. Neurologic disease frequently negatively affects sexual experience in multiple ways. Nevertheless, because a woman's ability to become pregnant and carry and deliver a child is largely unaffected following such neurological disease, such as spinal cord injury or multiple sclerosis, it was assumed, for many years, that her sexuality was similarly unaffected [2]. Therefore, for many times, female sexuality was equalized with childbearing, and scientific interest was confined to pregnancy and its complications [3].

Nevertheless, sexual dysfunctions are common including decreased or loss of libido, painful or uncomfortable genital sensations (burning, tingling, numbness), and/or altered orgasmic response in both women and men. Women may experience decreased vaginal lubrication and dryness, anorgasmia, and low sex desire [4, 5].

Secondary sexual dysfunction arises as a consequence of disability caused by poor bladder and bowel control, fatigue, muscle weakness, spasticity, immobility, tremor, cognitive impairment, and sensory problems [6].

Muscular weakness, spasticity, or insensibility may prevent movements and reduce mobility and pleasure during intercourse. Moreover, psychological and social factors could contribute to cause further sexual dysfunction as hypoactive sexual disorder.

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_15

15.1 Neuroanatomy and Neurophysiology

The innervation of the female genital tract is mediated through the somatic and autonomic nervous systems. Somatic innervation is conducted through the pudendal nerve. The first branch of the pudendal nerve, called the dorsal nerve of the clitoris, is a sensory nerve, without any motor functions.

It carries somatosensory impulses from the clitoris [1].

The second branch, called the perineal nerve, provides sensory branches to the perineum, labia majora, labia minora, and distal third of vagina [1].

Moreover the perineal nerve provides motor innervations to pelvic floor muscles. Sympathetic innervation is provided from the sacral portion of the sympathetic chain.

The uterovaginal plexus supplies the uterus, salpinges, ovaries, vagina, erectile tissue of the clitoris and vestibular bulbs, urethra, and greater vestibular glands [7].

The neurophysiology of sexual response in women is not well understood even in nonneurological patients. In determining the neural pathways involved in the control of sexual response, the study of persons with spinal cord injuries (SCIs) allows to test hypotheses regarding the role of specific spinal mechanisms [8].

Sipski et al., with their laboratory-based, controlled studies in patients with SCI, demonstrated that preservation of sensory function in the T11–L2 dermatomes is associated with psychogenically mediated genital vasocongestion.

Less than 50 % of women with SCIs were able to achieve orgasm, compared with 100 % of able-bodied women ($p < 0.001$). Only 17 % of women with complete lower motor neuron dysfunction affecting the S2–S5 spinal segments were able to achieve orgasm, compared with 59 % of women with other levels and degrees of SCIs ($p = 0.048$). Time necessary to achieve orgasm was significantly increased in women with SCIs compared with able-bodied controls ($p = 0.049$) [8].

The impact of SCI on sexual response depends on the degree of injury and its location in the spinal cord [9]. Most of the information available about male sexual response is based on questionnaire studies, whereas most of the data available about women comes from laboratory-based research [10].

For women with complete upper motor neuron injuries affecting the sacral segments, the ability for reflex but not psychogenic lubrication should be maintained [8]. This hypothesis has been tested in a laboratory-based analysis [8], and the results, although not conclusive, supported the hypothesis that lubrication occurs reflexively [8].

For women with incomplete upper motor neuron injuries affecting the sacral segments, it is thought that they may retain the capacity for reflex lubrication and may maintain the capacity for psychogenic lubrication [8].

Reflex lubrication and orgasm are more prevalent in women with SCI who had preserved the sacral reflex (S2–S5). For those with complete SCI of the sacral segment, arousal and orgasm may be evoked through stimulation of other erogenous zones above the level of lesions such as the breasts, lips, and ears [11].

15.2 Psychological Change After Neurological Diseases

Although various health conditions can affect sexuality, the sexual consequences of suffering a spinal cord injury (SCI) can be substantial, especially because the majority of SCIs occur during the years when reproductive and sexual capacities are at their peak [12].

The SCI women reported that the neurological disease caused many changes in their life and affected many aspects of their sexuality negatively. Some changes were of a physical nature (decreased, lost, or changed sensation; difficulties to achieve orgasm; bladder or bowel problems; and difficulties to move and position), whereas other changes were of a psychological nature (feeling unattractive or less attractive, having less self-confidence, and difficulties to meet or find a partner).

The main difference in sexual functioning between women with disabilities and those without can be accounted for by the difficulties women with disabilities have in finding a partner. Their level of sexual desire may be the same, but the level of activity is generally less because fewer women with disabilities have partners.

There are no changes after paralysis that prevent women from engaging in sexual activity. Positioning can be an issue but could usually be accommodated. Autonomic dysreflexia could be anticipated and controlled. Although women's desire for sexual activity seems to decrease after injury, 46 % of women with SCIs indicated that sex was less important after injury, and 44 % of women with SCIs rated their level of desire as "none" to "low" after injury [13, 14]. Frequency of sexual activity is also known to decrease in women with SCIs.

Recent studies have tried to investigate the true incidence of sexual dysfunction in women affected by SCIs, with a special attention to the level and completeness of injury, time from injury, and age of the patients. Biering-Sørensen et al. have demonstrated, in a survey on 43 SCIs women, that nearly all (94 %) female participants reported not having problems with impaired vaginal lubrication [15]. Sixty-nine percent of the women reported being satisfied with their sexual life [15]. When comparing satisfaction with sex life and the amount of problems regarding bladder and bowel management, pressure ulcers, spasticity, or pain, there was no statistically significant difference ($p = 0.08$), although there was a tendency toward the more problems reported, the less satisfied the women were likely to be with their sexual life [15]. No statistically significant difference was found in satisfaction with sexual life when comparing tetraplegic and paraplegic women, or in relation to the Frankel classification [15].

When comparing satisfaction with sexual life with age, it was found that the mean age was statistically significantly lower for the satisfied women compared with the unsatisfied women (45 vs 53 years). Likewise, those who were satisfied were younger at the time of injury (24 vs 31 years). There was no significant difference in mean time since SCI in relation to satisfaction with sexual life (21 vs 22 years). There was no significant difference in satisfaction with sexual life between those who had given birth to a child after SCI and those who had not (87.5 % vs

65.4 %) [15]. Other studies found that women with tetraplegia were less likely to have sexual intercourse than other women with SCI, due to the possible presence of autonomic dysreflexia and urinary incontinence [16].

15.3 Pregnancy and Spinal Cord Lesion

Unlike men with SCIs, the ability of women with injury to conceive is thought to be unchanged. Of the 231 women studied by Charlifue et al., 60 experienced an average of 5 months of temporary amenorrhea after injury. After this time period, the women's fertility should have returned to normal levels [17]. Conception, pregnancy, and normal labor and delivery are possible virtually with little added risk [3].

Some of reported complications of pregnancy in SCI patients are autonomic hyperreflexia, when lesion is above T6 level, major urinary tract infections, anemia, respiratory problems, skin fragility, and increased risk of early delivery [18]. Some studies reported a high risk of assisted deliveries with forceps because of weakness of abdominal muscles during expulsive phase [19].

For this reason and other factors such as autonomic hyperreflexia, fear of higher incidence of cesarean deliveries was reported [19].

No other problems were reported, especially no problem in the babies, except lower average birth weights than the general populations [19, 20].

15.4 Therapeutic Options

There are no evidence-based therapeutic options to treat neurological women with sexual dysfunction.

Lombardi et al., recently, in a 2-year follow-up after permanent SNM, reported that 36.5 % of females with SCI and sexual dysfunction obtained positive effects on sexual response and showed a remarkable concomitant improvement through the Female Sexual Distress Scale (FSDS) questionnaire [6].

References

1. Yang CC. Female sexual function in neurologic disease. J Sex Res. 2000;37:205–12.
2. Axel SJ. Spinal cord injured women's concerns: menstruation and pregnancy. Rehabil Nurs. 1982;7(5):10–5.
3. Turk R, Turk M, Assejev V. The female paraplegic and mother-child relations. Paraplegia. 1983;21:186–91.
4. Sipski ML, Arenas A. Female sexual function after spinal cord injury. Prog Brain Res. 2006;152:441–7.
5. Harden CL. Sexual dysfunction in women with epilepsy. Seizure. 2008;17:131–5.
6. Lombardi G, Musco S, Kessler TM, Li Marzi V, Lanciotti M, Del Popolo G. Management of sexual dysfunction due to central nervous system disorders: a systematic review. BJU Int. 2015;Suppl 6:47–56.

7. Graziottin A, Giraldi A. Anatomy and physiology of women's sexual function. In: Porst H, Buvat J, editors. ISSM (International Society of Sexual Medicine) Standard Committee Book, standard practice in sexual medicine. Oxford: Blackwell; 2006. p. 289–304.
8. Sipski ML, Alexander CJ, Rosen R. Sexual arousal and orgasm in women: effects of spinal cord injury. Ann Neurol. 2001;49(1):35.
9. Bors E, Comarr EE. Neurological disturbances of sexual function with special reference to 529 patients with spinal cord injury. Urol Surv. 1960;110:191–221.
10. Berard EJ. The sexuality of spinal cord injured women: physiology and pathophysiology, a review. Paraplegia. 1989;27:99–112.
11. Sipski ML, Alexander CJ, Gomez-Marin O, Grossbard M, Rosen R. Effects of vibratory stimulation on sexual response in women with spinal cord injury. J Rehabil Res Dev. 2005;42: 609–16.
12. HA Fritz, H Dillaway, CL Lysack. "Don't think paralysis takes away your womanhood": sexual intimacy after spinal cord injury. Spinal Cord. 2011;49(1):154–60. doi:10.1038/sc.2010.51. Epub 2010 May 11.
13. Sipski ML, Alexander CJ. Sexual activities, response and satisfaction in women pre- and post-spinal cord injury. Arch Phys Med Rehabil. 1993;74:1025–9.
14. Kreuter M, Taft C, Siösteen A, Biering-Sørensen F. Women's sexual functioning and sex life after spinal cord injury. Spinal Cord. 2011;49(1):154–60.
15. Biering-Sørensen I, Rikke Bølling H, Biering-Sørensen F. Sexual function in a traumatic Spinal Cord Injured Population 10-45 years after injury. J Rehabil Med. 2012;44:926–31.
16. Lombardi G, Del Popolo G, Macchiarella A, Mencarini M, Celso M. Sexual rehabilitation in women with spinal cord injury: a critical review of the literature. Spinal Cord. 2010;48: 842–9.
17. Charlifue SW, Gerhart KA, Menter RR, Whiteneck GG, Scott Manley M. Sexual issues of women with spinal cord injuries. Paraplegia. 1992;30:192–9.
18. Johnston B. Pregnancy and childbirth in women with spinal cord injuries: a review of the literature. Matern Child Nurs J. 1982;11(1):41–5.
19. Verduyn WH. Spinal cord injured women, pregnancy and delivery. Paraplegia. 1986;24: 231–40.
20. Robertson DNS. Pregnancy and labor in the paraplegic. Paraplegia. 1972;16(3):209–12.

Female Genital Cosmetic Surgery

16

Sushma Srikrishna and Linda Cardozo

Abbreviations

BDD	Body dysmorphic disorder
CO_2	Carbon dioxide
COPS-L	Cosmetic Procedure Screening Scale modified for labia
Er:YAG	Erbium yttrium aluminium garnet
FGCS	Female genital cosmetic surgery
FGM	Female genital mutilation
GAS	Genital Appearance Satisfaction
GRISS	Golombok-Rust Inventory of Sexual Satisfaction
G-Spot	Grafenberg spot
MDT	Multidisciplinary team
Nd:YAG	Neodymium yttrium aluminium garnet
NHS	National Health Service
NICE	National Institute for Health and Care Excellence
PISQ	Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire
UK	United Kingdom

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_16

16.1 Introduction

Female genital cosmetic surgery (FGCS) is an overarching term that is used to describe a range of procedures aimed at 'enhancing' female genital appearance and/or function. The Royal College of Obstetricians and Gynaecologists defines FGCS as non-medically indicated cosmetic surgical procedures which change the structure and appearance of the healthy external genitalia of women or internally in the case of vaginal tightening [1]. This definition includes the most common procedure, labiaplasty, as well as others, such as hymenoplasty and vaginoplasty, also known as vaginal reconstruction and vaginal rejuvenation [2]. These interventions are performed mainly for cosmetic reasons although some procedures (principally labiaplasty) can be medically indicated.

Genital surgical alterations performed for aesthetic and/or sexual reasons were initially described around 30 years ago [3–5]; however the demand for FGCS has increased over the last decade [6]. This rise is difficult to quantify, as the majority of these procedures are performed in the private sector. However, this trend is also obvious within national health systems (NHS, UK), where the number of operations coded as 'excision of excess labial tissue' increased from 568 in 2002 to 1064 in 2008 [7]. Other published data suggests that the number of labiaplasty interventions performed in the UK has increased fivefold in 9 years, from 2001 to 2010 [8].

Worryingly, this trend towards increased demand for FGCS is not limited to adult alone, with the British Society for Paediatric and Adolescent Gynaecology reporting 266 labial reductions performed in the UK between 2008 and 2012 on girls under the age of fourteen [9], despite guidelines advising against this practice, given the immaturity of prepubescent genitalia.

A number of reasons could explain the increasing demand for FGCS. Younger women favour less pubic hair, which results in easier visualisation of the external genitalia [10]. Increased access to female body images on the Internet, television and magazines has raised awareness of genital appearance and skewed the perception of normality towards adolescent features, with flat vulvas without protrusion behind the labia majora [11]. Television programmes and articles in lifestyle magazines on FGCS might also influence the desire for a 'face lift of the vulva and vagina', together with the rising number of cosmetic procedures in general. Advertisement and aggressive marketing of FGCS could also inflate the demand for surgery. A recent Google search for 'cosmetic genital surgery' highlighted more than 2,000,000 items, whereas a PubMed search found only 234 items, demonstrating the huge increase in interest regarding this type of surgery by the general public with the medical profession being far less well informed.

The quality and quantity of clinical information in FGCS provider sites is poor, with erroneous information in some instances [12]. This field has been described like 'the old Wild, Wild West: wide open and unregulated' [13]. The majority of FGCS procedures are poorly defined and there is no standardised terminology. The use of non-medical terms, such as 'designer vagina', 'vaginal rejuvenation', 'laser vaginal rejuvenation®' or 'G-shot®', makes the scientific

assessment of these operations very difficult. The terminology needs to be standardised in an effort to avoid ambiguity and inconsistency [14]. Additionally, conflicts of interest prevent studying trademarked or proprietary treatments [15]. There is a well-recognised lack of robust evidence on the safety and benefits of these procedures [16].

16.2 Ethical Issues

The rising number of these operations has also heated a controversy about the relationship between FGCS and female genital mutilation (FGM). FGM comprises all procedures that involve 'partial or total removal of the external female genitalia or other injury to the female genital organs for non-medical reasons' [17] and is a criminal offence in most of the Western world countries. In the UK, the Female Genital Mutilation Act 2003 excludes surgical operations which are necessary for a woman's physical or mental health [18]. However, as there is limited evidence regarding the benefit of FGCS for physical and mental health, some campaigners suggest that medical practitioners should be prosecuted [19].

In addition, several gynaecological societies including the Royal College of Obstetricians and Gynaecology in the UK [20] and similar organisations in America, Canada, Australia and New Zealand [21–23] have all released documents expressing concerns about FGCS and discouraging the performance of any surgical intervention in the absence of robust scientific evidence. In addition, advice from these learned societies highlights the importance of sexual and psychological counselling; when offering FGCS and obtaining informed consent, only a full discussion of all the risks is associated with these procedures.

Another controversial issue is the funding of these procedures. The majority of the cosmetic operations performed for purely aesthetic reasons are carried out in the private sector and are self-funded. But a share of the demand for FGCS is being absorbed by publicly funded healthcare services [6]. Healthcare providers should discourage the use of public funds for performance of pure cosmetic procedures. Nevertheless, if FGCS procedures have physical or mental health benefits or are being performed for functional reasons, it might be justifiable to utilise public funding. A clear and strict selection process of appropriate candidates for FGCS is required to allow allocation of government funding.

16.3 Normal Anatomy and Patient Perception

A robust selection process would be impossible without understanding the normal anatomy of the female genitalia. A small cross-sectional study reporting on the normal dimensions and exact positioning of the vagina, urethra, clitoris and labia showed a wide natural variation of these features [24]. The mean labia minora width was 21.8 mm, but with a broad range of 7-50 mm, and their length was 60.6 mm with a range between 20 and 100 mm. These results have challenged a previous

definition of labia minora hypertrophy, which used a width of 40 mm as a cutoff [25]. Surprisingly, labial measurements of a cohort of 33 women requesting labial reduction surgery were all within the published normal range [26].

Measurements of genitalia	Range	Mean [SD]
Clitoral length (mm)	5–35	
Clitoral glans width (mm)	3–10	
Clitoris to urethra (mm)	16–45	
Labia majora length (cm)	7.0–12.0	
Labia minora length (mm)	20–100	
Labia minora width (mm)	7–50	
Perineum length (mm)	15–55	
Vaginal length (cm)	6.5–12.5	
Tanner stage (*n*)	IV	4
	V	46
Colour of genital area compared with surrounding skin (*n*)	Same	9
	Darker	41

A comparison of labial measurements between women seeking labiaplasty in an NHS gynaecology clinic and those in a private cosmetic clinic has shown that NHS patients appeared to have significantly greater labia minora width than the private patients (mean 40.27 vs 28.09 mm, $p<0.001$) [27]. Increased access to genital pictures may narrow the social definition of normal and increase the desire to emulate what is perceived as being attractive. In addition, many women who are worried about the appearance of their genitals search for information on the Internet as a 'first port of call'. Unfortunately, a lot of the information available online is published by the surgery provider's sites and has been reported to be misleading and erroneous [28]. In addition, the trend of Brazilian waxes which serve to emphasise visualisation of the external genital coupled with the concept promoted by online pornographic images that it is the norm to have completely hairless vulvae Internet pornography has set a new standard for vulvovaginal attractiveness where the labia minora do not protrude beyond the labia majora [8, 29]. Published data suggests that more women regularly self-examine their vulva, as they think its appearance is important [28, 30]. Considering the enormous natural variation of the topographical features of the female genitalia and the overlap between women seeking FGCS and those who do not, it is difficult to use anatomic measurements as objective criteria for surgery.

16.4 Body Dysmorphic Disorder and FGCS

Given the dichotomy between the wide range of normality of female external genitalia and the evolving concept of 'attractiveness', some women may experience significant emotional distress, such as feeling 'abnormal' or being ashamed based on the appearance of their genitalia [31]. These concerns may even be part of a wider mental health disorder such as body dysmorphic disorder (BDD). BDD is characterised by extreme preoccupation with minor or nonexistent defects or flaws

in physical appearance associated with intrusive thoughts, persistent distress, significant impairment in social and occupational functioning and repetitive behaviours, such as mirror checking, seeking reassurance from others and even requesting unnecessary cosmetic surgery [32, 33]. In one published study, nearly one fifth of all women seeking labiaplasty met the diagnostic criteria for BDD [34]. As the onset of BDD typically occurs during early adolescence [35], affected young women have to cope with the pressure of puberty and changes in self-identity which normally occur at this age [36]. Therefore, the assessment of the psychological health of adolescents seeking FGCS is even more important.

Finally, it is worth considering that the worry that an NHS doctor may not operate on a woman seeking FGCS for psychological reasons or that health insurance may not cover the procedure done privately often leads women to emphasise more functional reasons for considering FGCS, such as interference with physical exercise, sexual intercourse and wearing tight-fitting clothes [31]. Therefore psychological counselling should be strongly recommended to all healthy women who are considering FGCS for any reason.

16.5 Types of FGCS

There are a number of procedures included under the umbrella term FGCS (Table 16.1). It is of paramount importance to use descriptive and standardised terms such as those employed in the field of urogynaecology/female urology, rather than trademarked or commercial names. The procedures could be further subdivided based on the operating instruments such as laser, radiofrequency,

Table 16.1 FGCS procedures

FGCS procedures
1. Labiaplasty
I. Labia minora reduction
(a) Linear resection
(b) Wedge resection (V-wedge, Z-plasty)
(c) Deepithelised reduction
II. Labia majora augmentation
III. Labia majora reduction
2. Vaginoplasty
I. Anterior colporraphy
II. Posterior colporraphy (with or without levator ani plication)
III. Lateral colporraphy
IV. Vaginal bulking procedures
3. Perineoplasty
I. Perineorrhaphy
II. Fenton's procedure
4. Clitoral hood reduction
5. Hymenoplasty (hymenorrhaphy)
6. G-spot bulking (amplification)

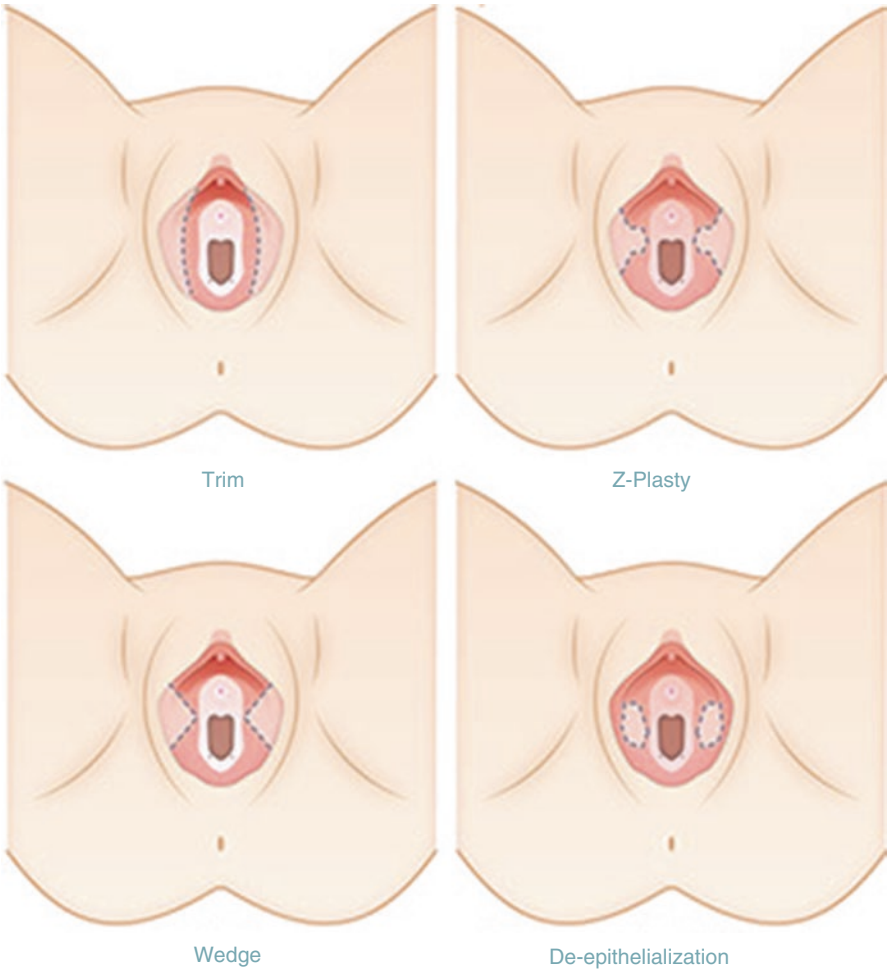

Fig. 16.1 Surgical techniques for labial reduction (Motakef et al. [49])

electrocautery or 'cold knife'. For laser-assisted procedures, different types of laser (CO_2, Er:YAG, Nd:YAG) have been used for excision or ablation [37]. For bulking procedures, the most commonly used materials are hyaluronic acid and autologous fat. Labiaplasty, clitoral hood reduction, hymenoplasty and G-spot amplification are described below in detail. Descriptions of vaginoplasty and perineoplasty techniques are available in most general gynaecology textbooks and are not elaborated on further in this chapter in interest of brevity.

Labia minora reduction: Several techniques exist to reduce the labia minora (Fig. 16.1).

Direct excision involves amputation of the labial tissue protruding through the labia majora and reapproximation of the edges. This technique creates a straight scar positioned along the labial edge and removes the labial border. However, direct excision may distort the labial edge and is not indicated for patients who wish to

retain the labial border. Moreover, the linear scar may contract and cause tight introitus or tension in the posterior fourchette.

Wedge resection A wedge or V-shaped resection can be centred over the protuberant region of the labia minora to reduce the size of the labia whilst preserving the labial border. However, this resection pattern can create an abrupt colour change in the labial border and leaves a linear scar that can contract and distort the natural contour of the labium. A modification to avoid linear contraction of the scar made by wedge resection is called Z-plasty, which adds 90° Z-plasties to each arm of the wedge.

Resection of the inferior wedge and reconstruction of the superior pedicle flap are indicated for patients with moderate to severe hypertrophy of the labia minora. To mark the tissue for resection, the middle portion of each labium is stretched inferiorly towards the posterior vaginal introitus. The resulting wedge of pinched tissue is resected. When the borders are reapproximated, the superior flap reconstructs the defect. This approach preserves the free edge of the labium and leaves a scar in the posterior, more hidden aspect of the labia minora. However, a mild bulging deformity of the contour may result because the superior flap is wider than the posterior edge of the labium.

Posterior wedge resection is a modification to inferior wedge resection and reconstruction of the superior flap. With this procedure, more of the anterior labial border is preserved, and most of the tissue is resected from the middle of the labium. These modifications reduce tension and decrease the risk of a bulging deformity but leave a narrow anterior flap with a potentially reduced vascular supply.

Deepithelialised reduction involves fusiform resection of the epidermis at the inner and outer sides of the labium. This technique reduces the lengths of the labia and preserves the border. Moreover, deepithelialisation reduces the number of sebaceous glands, thereby reducing the outflow of vaginal secretions.

The linear resection and modified wedge techniques appear to be the most widely utilised. The single study in the literature comparing the two most commonly performed procedures, modified V-wedge and linear resection, found little difference in short-term outcomes between the two procedures [38].

16.5.1 Labia Majora Augmentation

The labia may be augmented with fat injections. Fat is harvested by liposuction preferably from a site where liposuction has not been performed previously. Fat is collected into 5-mL syringes and is injected evenly into the labia majora, with 1.5-mm injection cannulae, until the desired size is achieved. Fat injections are technically less complex than other augmentation procedures and leave smaller scars. However, fat reabsorption may occur and may be variable and unpredictable (Fig. 16.2).

16.5.2 Labia Majora Reduction

If the source of the hypertrophy is fat rather than ptotic skin, liposuction may be performed by means of ≤3-mm cannulae. This procedure is simple and involves minimal scarring. However, contour irregularities may arise with time, and existing ptosis may become more severe.

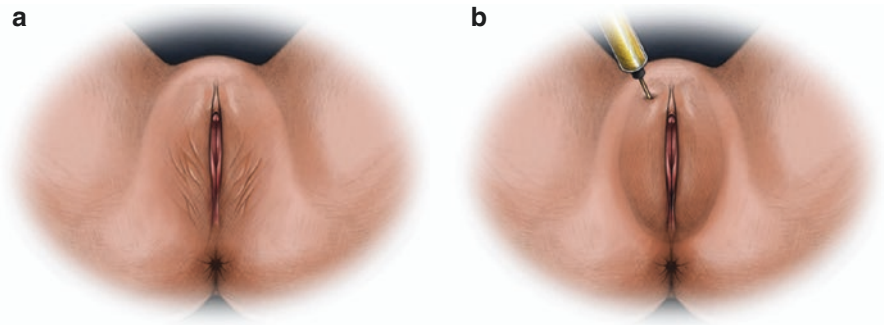

Fig. 16.2 Labia majora augmentation. *A* pre OP, *B* post OP

If excess skin is the cause of labia majora hypertrophy, resection of skin and fatty tissue may be carried out. Longitudinal resection of skin and subcutaneous tissue with scar placement in the vulva-thigh crease reduces the size of the labia without disturbing the labial edge. This method creates a scar in a high-tension area, increasing the likelihood of wound dehiscence and potentially opening the vaginal introitus. Excess skin and subcutaneous tissue also may be reduced by dermolipectomy of the longitudinal aspect of the labia. Alternatively, the final scar can be placed in the skin-mucosa transition, leaving it well concealed.

16.5.3 Clitoral Hood Reduction

In a technique involving hydrodissection with reverse V-plasty, the glans clitoris and hood are separated by hydrodissection, and a V-shaped incision is made in the clitoral hood to resect the contracted part and correct the phimosis. This modified V-plasty also can be applied to patients with excess tissue at the clitoral hood. For patients with asymmetric subdermal hypertrophy, subepithelial reduction 15 is recommended. This technique involves resection of hypertrophied subepithelial tissues to create a clitoral hood with symmetric thickness bilaterally (Fig. 16.3).

16.5.4 Hymenoplasty

Hymenoplasty is performed to narrow the vaginal opening and restore the hymen. Hymenoplasty may be requested by individuals in societies where virginity is culturally expected or by those who are not sexually active but have experienced genital trauma. Hymenoplasty generally is performed under local anaesthesia with sedation. The patient is placed in the lithotomy position, and caruncles are identified under a magnifying lens. The margins of each caruncle are freshened, and the caruncles are sutured together with absorbable stitches preserving an appropriate vaginal opening to allow for passage of vaginal and menstrual fluids (Fig. 16.4).

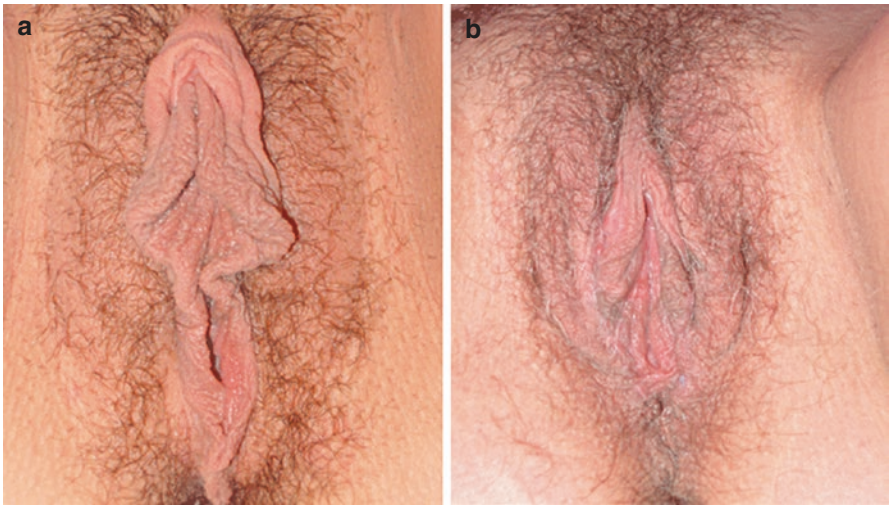

Fig. 16.3 Clitoral hood reduction. *A* pre OP, *B* post OP

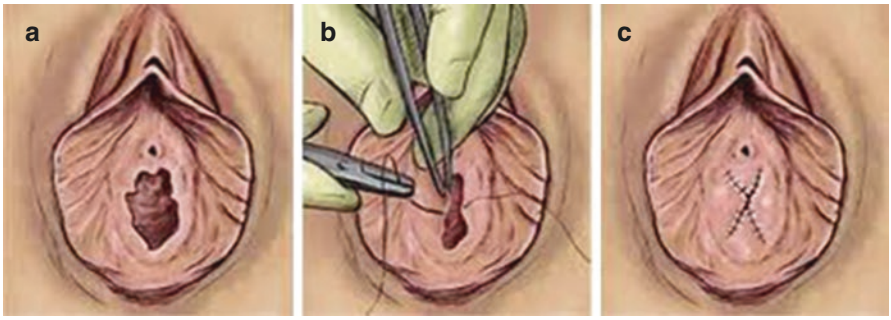

Fig. 16.4 Hymenoplasty. Continuing Medical Education (Triana and Robledo [50]. *A* pre OP, *B* post OP. Reprints and permission: journals.permissions@oup.com. www.aestheticsurgeryjournal.com)

16.5.5 G-Spot Amplification

G-Spot Amplification® was invented and developed by the gynaecologist David Matlock. The G-Shot® or G-Spot Amplification® is a patent-pending technique to enhance the Grafenberg spot with a bioengineered human collagen product. The G-Shot® (clinical description: G-Spot Amplification® or GSA®) is described online as a simple, nonsurgical, physician-administered treatment that can temporarily augment the Grafenberg spot (G-spot) in sexually active

women with normal sexual function, for a temporary period of time, around 4 months. No descriptions of the procedure can be found in any peer-reviewed journal, nor any studies scientifically evaluating its merits or potential complications.

16.6 Indications

Women seek FGCS for aesthetic, functional, sexual and cultural reasons. A number of physical complaints are described by these women, such as pain, discomfort or irritation associated with clothing, exercise, sexual intercourse as well as sensation of vaginal relaxation and lack of coital friction. In a retrospective study of 131 patients undergoing labiaplasty, 32 % were seeking surgery strictly for functional impairment, 37 % for cosmetic reasons only and 31 % for both functional and cosmetic reasons [39]. In a different multicentre cohort of 258 women undergoing FGCS, 64 % reported discomfort preoperatively, 48 % cosmetic reasons, 33 % wanted surgery for self-esteem and 30 % for sexual enhancement [40]. In the same study, the majority of women undergoing vaginoplasty and/or perineoplasty expected increase in their (58 %) or their partner's (54 %) sexual pleasure. Women referred to an NHS gynaecology clinic by their general practitioner are more likely to report functional reasons, compared to self-referred women attending a private cosmetic clinic [27].

The request for hymenoplasty is rather different and raises further ethical issues. It is usually made by a different group of patients. They often come from specific ethnic/religious groups especially African and Middle Eastern countries, where bleeding during postnuptial intercourse and vaginal tightness are considered proofs of virginity [41].

16.7 Outcomes

Despite the increasing interest in FGCS, only limited outcome data have been published in the literature. The majority of the studies are retrospective, single-centre case series with short-term follow-up (level of evidence III–IV), which report subjective, non-validated outcome measures. Every study shows success/satisfaction rate more than 80 % [42]. In a large multicentre study, 97 % of patients undergoing labiaplasty and 83 % of patients undergoing vaginoplasty/perineoplasty felt that they achieved their goals postoperatively. Improvement of their sexual function was reported by 64 % and 86 % of women, respectively [40].

The incidence of reported complications varies in the literature (3.8–23.8 %). A wide range of postoperative complications have been reported such as infection, haematoma, dyspareunia, localised pain, altered sensation, poor wound healing, scarring, wound separation and even bowel and bladder injury with fistula formation. The majority of these complications are considered as minors by the authors of the relevant papers, which do not apparently interfere with overall satisfaction [27].

16.8 Preoperative Considerations

Women should be adequately counselled and provide informed consent about FGCS procedures. They should be educated about the wide variety of normal female genital appearance and be reassured about their genital anatomy. Special attention should be paid to younger girls, as the development of external genitalia continues throughout adolescence. Ideally, any surgical intervention shall be delayed until the completion of the development. Women seeking FGCS should be made aware of the lack of robust scientific evidence about the safety and efficacy of these operations. Preoperative counselling regarding functional, sexual and cosmetic benefits should be balanced against potential risks to help patients develop realistic expectations. Physicians should declare any conflicts of interest for treatments, where commercial rights are involved.

The relevance of psychological issues in cosmetic surgery has been well established in the literature, and the increased prevalence of underlying psychopathology in patients requesting surgery has been recognised [43]. Women seeking labiaplasty do not differ from controls on measures of depression or anxiety, but they have a worse body image quality of life ($p=0.041$) and they are more likely to suffer from body dysmorphic disorder (BDD) (18 % vs 0 %, $p<0.001$) [18]. Consequently, screening for psychopathology during the preoperative interview is essential. Based on this, the surgeon could refer patients to a psychologist or psychiatrist and withhold FGCS from women with significant mental impairment. We recommend the routine use of the Genital Appearance Satisfaction (GAS) scale, an 11-item tool, and the Cosmetic Procedure Screening Scale modified for labia (COPS-L), a 9-item instrument, which are both validated in women seeking labiaplasty [44].

Another challenging group are patients with sexual dysfunction. Although the majority of women undergoing FGCS, mainly vaginoplasty and perineoplasty, expect improvement of their sexual function, caution in the preoperative counselling is required. There is a lack of good-quality evidence regarding sexual outcome after FGCS, and therefore patients should be aware that functional and cosmetic benefits are the basic indications for these operations. Validated instruments of sexual function/dysfunction such as the Golombok-Rust Inventory of Sexual Satisfaction (GRISS) [45] or Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire [PISQ] [46] structured screening questions should be used preoperatively. Women with significant sexual dysfunction should be referred to a qualified psychosexual therapist before any planned surgical intervention.

16.9 Service Provision

In the current unregulated environment of FGCS, gynaecologists, urogynaecologists, plastic surgeons and urologists perform these operations. Despite the absence of structured training programmes, the majority of the plastic surgeons in the USA (51 %) offer FGCS with a mean workload of 3.68 procedures per year [47]. These procedures are not considered gynaecologic or urologic and generally are not taught during residencies, as there are no training guidelines. In view of the increasing

demand of these operations, the relevant surgical professional bodies should provide some guidance on training and practice requirements. As with other surgical procedures, service accreditation should be based on education, training, experience and demonstrated competence, as well as an adequate workload and audit of outcomes.

FGCS should ideally be provided by a multidisciplinary team (MDT) of healthcare professionals, similar to the model recommended by the National Institute for Health and Care Excellence (NICE) for the management of urinary incontinence in women [48]. The MDT should include at least two operating surgeons, a psychologist/psychiatrist, a psychosexual therapist and a physiotherapist. Having representation from professionals with different experience would allow a wider choice for patients and better patient selection. An MDT review before all surgical procedures could save future problems arising from poor decision-making and potentially increase patient satisfaction. Furthermore, the MDT would also be able to make special clinical governance arrangements, audit interventions and continuously monitor and review outcomes. A national or international registry for FGCS procedures or an online database tool, similar to the one offered by the British Society of Urogynaecology for prolapse and urinary incontinence surgery, could create a regulatory mechanism in the field.

Conclusions

FGCS is a rapidly expanding and poorly regulated field of cosmetic surgery. Despite the controversies, the relevant surgical professional bodies should attempt to regulate training and performance of these procedures. The terminology should be standardised and descriptive in order to improve consistency and allow systematic review of the literature. Emotive terms such as 'designer vagina', 'laser vaginal rejuvenation®' or 'G-Shot®' should be abandoned in an effort to avoid confusion and develop realistic patient's expectations. Well-designed prospective randomised controlled trials or comparative studies with long-term follow-up, which use validated tools and patient-reported outcomes, are needed. Patients should be carefully counselled and selected preoperatively ideally by a multidisciplinary team. Clinical governance arrangements should be made to allow continuous monitoring of FGCS provision and to facilitate auditing of the service. The request for FGCS may be reasonable, and whilst we all respect the right to choose, there needs to be increased regulation in this controversial area of medicine.

Financial Disclaimer/Conflict of Interest *S Srikrishna*: Speaker Honorarium: Astellas

L. Cardozo: Consultant: Allergan, AMS, Astellas, Pfizer Speaker Honorarium: Allergan, Astellas, Trial Participation: Astellas, Pfizer. Research grant: Pfizer

References

1. RCOG: Ethical opinion paper Ethical considerations in relation to female genital cosmetic surgery (FGCS). 2013.
2. Goodman MP, Placik OJ, Benson RH 3rd, Miklos JR, Moore RD, Jason RA, Matlock DL, Simopoulos AF, Stern BH, Stanton RA, Kolb SE, Gonzalez F. A large multicenter outcome study of female genital plastic surgery. J Sex Med. 2010;7(4 Pt 1):1565–77. doi:10.1111/j.1743-6109.2009.01573.x. Epub 2009 Nov 12.

3. Honore LH, O'Hara KE. Benign enlargement of the labia minora: report of two cases. Eur J Obstet Gynecol Reprod Biol. 1978;8:61–4.
4. Hodgekinson DJ, Hait G. Aesthetic vaginal labiaplasty. Plast Reconstr Surg. 1984;74:414–6.
5. Chavis WM, LaFeria JJ, Niccolini R. Plastic repair of elongated hypertrophic labia minora. A case report. J Reprod Med. 1989;34:3737–45.
6. Liao LM, Creighton SM. Requests for cosmetic genitoplasty: how should healthcare providers respond? BMJ. 2007;334:1090–2.
7. Hospital Episode Statistics – Health and Social Care Information Centre. www.hscic.gov.uk. Last accessed 30 Sept 2013.
8. Moran C, Lee C. What's normal? Influencing women's perceptions of normal genitalia: an experiment involving exposure to modified and nonmodified images. BJOG. 2014;121:761–6.
9. British society for paediatric and adolescent gynaecology position statement, Labial reduction surgery (labiaplasty) on adolescents, October 2013
10. Yurteri-kaplan LA, Antosh DD, Sokol AI, Park AJ, Gutman RE, Kingsberg SA, et al. Interest in cosmetic vulvar surgery and perceptions of vulvar appearance. Am J Obstet Gynecol. 2012;207:428.e1–7.
11. Braun V. In search of (better) sexual pleasure: female genital cosmetic surgery. Sexualities. 2005;8:407–24.
12. Liao LM, Taghinejadi N, Creighton SM. An analysis of the content and clinical implications of online advertisements for female genital cosmetic surgery. BMJ Open. 2012;2(6). doi:pii: e001908. 10.1136/bmjopen-2012-001908.
13. Goodman MP. Female cosmetic genital surgery. Obstet Gynecol. 2009;113:154–9.
14. Mirzabeigi MN, Jandali S, Mettel R, Alter G. The nomenclature of "vaginal rejuvenation" and elective vulvovaginal plastic surgery. Aesthetic Surg J. 2011;31:723–4.
15. Cain JM, Iglesia CB, Dickens B, Montgomery O. Body enhancement through female genital cosmetic surgery creates ethical and rights dilemmas. Int J Gynaecol Obstet. 2013;122:169–72.
16. Liao LM, Michala L, Creighton SM. Labial surgery for well women: a review of the literature. BJOG. 2010;117:20–5.
17. World Health Organisation. Female genital mutilation. Fact sheet No 241. Updated February 2013.
18. Female Genital Mutilation Act 2003. www.legislation.gov.uk/ukpga/2003/31/pdfs/ukpga_20030031_en.pdf.
19. Berer M. It's female genital mutilation and should be prosecuted. BMJ. 2007;334:1335.
20. Royal College of Obstetricians and Gynaecologist. Ethical considerations in relation to female genital cosmetic surgery (FGCS). RCOG Ethics Committee, October 2013
21. Committee on Gynecologic Practice, American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 378: Vaginal "rejuvenation" and cosmetic vaginal procedures. Obestet Gynecol. 2007;110:737–8.
22. Clinical Practice Gynaecology Committee, Ethics Committee, Executive Council of the Society of Obstetricians and Gynaecologists of Canada. Female genital cosmetic surgery. J Obstet Gynaecol Can. 2013;35:e1–5.
23. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists. RANZCOG College Statement: C-Gyn 24. Vaginal 'Rejuvenation' and Cosmetic Vaginal Procedures. 2008.
24. Lloyd J, Crouch NS, Minto CL, Liao LM, Creighton SM. Female genital appearance: "normality" unfolds. BJOG. 2005;112:643–6.
25. Rouzier R, Louis-Sylvestre C, Paniel BJ, Haddad B. Hypertrophy of the labia minora; experience of 163 reductions. Am J Obstet Gynecol. 2000;182:35–40.
26. Crouch NS, Deans R, Michala L, Liao LM, Creighton SM. Clinical characteristics of well women seeking labial reduction surgery: a prospective study. BJOG. 2011;118:1507–10.
27. Veale D, Eshkevari E, Ellison N, Costa A, Robinson D, Kavouni A, et al. Psychological characteristics and motivation of women seeking labiaplasty. Psychol Med. 2013;10:1–12 [Epub ahead of print].

28. Liao LM, Taghinejadi N, Creighton SM. An analysis of the content and clinical implications of online advertisements for female genital cosmetic surgery. BMJ Open. 2012;2. doi:10.1136/bmjopen-2012-001908
29. Iglesia CB. Cosmetic gynecology and the elusive quest for the "perfect vagina". Obstet Gynecol. 2012;119:1083–4.
30. Koning M, Zeijlmans IA, Bouman TK, van der Lei B. Female attitudes regarding labia minora appearance and reduction with consideration of media influence. Aesthet Surg. 2009;29:65–71.
31. Zwier S. "What motivates her": motivations for considering labial reduction surgery as recounted on women's online communities and surgeons' websites. Sex Med. 2014;2:16–23.
32. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington, DC: American Psychiatric Association; 2013.
33. Crerand CE, Franklin ME, Sarwer, DB. Body dysmorphic disorder and cosmetic surgery. Plast Reconstr Surg. 2006;118:167e–80e.
34. Veale D, Eshkevari E, Ellison N, Costa A, Robinson D, Kavouni A, Cardozo L. Psychological characteristics and motivation of women seeking labiaplasty. Psychol Med. 2014;44:555–66.
35. Phillips KA. Body dysmorphic disorder: recognizing and treating imagined ugliness. World Psychiatry. 2003;3:12–7.
36. Facchin F, Margola D, Molgora S, Revenson TA. Effects of benefit-focused versus standard expressive writing on adolescents' self-concept during the high school transition. J Res Adolesc. 2014;24:131–44.
37. Iglesia CB, Yurteri-Kaplan L, Alinsod R. Female genital cosmetic surgery: a review of techniques and outcomes. Int Urogynecol J. 2013;22 [Epub ahead of print].
38. Goodman MP, Placik OJ, Benson III RH, Miklos JR, Moore RD, Jason RA, Matlock DL, Simopoulos AF, Stern BH, Stanton RA, Kolb SE, Gonzalez F. A large multicenter outcome study of female genital plastic surgery. J Sex Med. 2010;7:165–77.
39. Milkos JR, Moore RD. Labiaplasty of the labia minora: patients' indications for pursuing surgery. J Sex Med. 2008;5:1492–5.
40. Goodman MP, Placik OJ, Benson RH, Miklos JR, Moore RD, Jason RA, et al. A large multicentre outcome study of female genital plastic surgery. J Sex Med. 2010;7:1565–77.
41. van Moorst BR, van Lunsen RH, van Dijken DK, Salvatore CM. Backgrounds of women applying for hymen reconstruction, the effects of counselling on myths and misunderstandings about virginity, and the results of hymen reconstruction. Eur J Contracept Reprod Health Care. 2012;17:93–105.
42. Goodman MP. Female genital cosmetic and plastic surgery: a review. J Sex Med. 2011;8:1813–25.
43. Hasan JS. Psychological issues in cosmetic surgery: a functional overview. Ann Plast Surg. 2000;44:89–96.
44. Veale D, Eshkevari E, Ellison N, Cardozo L, Robinson D, Kavouni A. Validation of genital appearance satisfaction scale and the cosmetic procedure screening scale for women seeking labiaplasty. J Psychosom Obstet Gynaecol. 2013;34:46–52.
45. Rust J, Golombok S. The Golombok-Rust inventory of sexual satisfaction (GRISS). Br J Clin Psychol. 1985;24:63–4.
46. Rogers RG, Coates KW, Kammerer-Doak D, Khalsa S, Qualls C. A short form of the pelvic organ prolapse/urinary incontinence sexual questionnaire (PISQ-12). Int Urogynecol J Pelvic Floor Dysfunct. 2003;14(3):164–8.
47. Mirzabeigi MN, Moore Jr JH, Mericli AF, Bucciarelli P, Jandali S, Valerio IL, Stofman GM. Current trends in vaginal labioplasty: a survey of plastic surgeons. Ann Plast Surg. 2012;68:125–34.
48. National Institute for Health and Care Excellence (NICE) Clinical guideline 171. Urinary incontinence in women. 2013. http://www.nice.org.uk/nicemedia/live/14271/65144/65144.pdf. Assessed 30 Oct 2013.
49. Motakef S, Rodriguez-Feliz J, Chung MT, Ingargiola MJ, Wong VW, Patel A. Vaginal labiaplasty: current practices and a simplified classification system for labial protrusion. Plast Reconstr Surg. 2015;135(3):774–88.
50. Triana L, Robledo AM. Aesthetic surgery of female external genitalia. Aesthetic Surg J. 2015;35(2):165–77. doi:10.1093/asj/sju020.

Role of Physical Therapy in the Treatment of Female Dysfunction

17

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Abbreviations

B-FLUTS	Bristol Female Lower Urinary Tract Symptoms
FSD	Female sexual dysfunction
FSFI	Female Sexual Function Index
ICIQ-FLUTSsex	International Consultation on Incontinence Modular Questionnaire sexual matters module
ICIQ-VS	International Consultation on Incontinence Modular Questionnaire—Vaginal symptoms questionnaire
I-QOL	Incontinence Quality of Life Questionnaire
LA muscle	Levator ani muscle
PEDro	Physiotherapy Evidence Database
PFD	Pelvic floor dysfunctions
PFM	Pelvic floor muscles
PFMT	Pelvic floor muscle training
PISQ-12	Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire
POP	Pelvic organ prolapse

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5_17

PT	Physical therapist
QoLS-N	Quality of life scale
RCT	Randomized controlled trial
SAI	Sexual arousal inventory
SAI-E	Sexual arousal inventory-expanded form
SII	Sexual interaction inventory
SUI	Stress urinary incontinence
UI	Urinary incontinence
WSQ	Women's Sexuality Questionnaire

17.1 Introduction

Female sexual dysfunction (FSD) has been classified as sexual interest/arousal disorder, orgasmic disorder, and genitopelvic pain/penetration disorder [1]. It is commonly found among women of all ages [2]. Despite increasing demand for clinical services and the potential impact of these disorders on relationship satisfaction and quality of life [3], there is a lack of knowledge in how to approach women with FSD. It is an interlinked and complex disorder rooted in biological, social/relationship, and psychological aspects [4].

It has been postulated that the pelvic floor muscles' (PFM) tone, strength, and ability to contract are important factors for vaginal receptivity and responsiveness, for the pleasure during intercourse for both partners, and for the orgasmic muscular response [4]. Some studies have shown that strong PFM may be associated with better orgasmic and arousal potentials, desire, excitement, and vaginal lubrication [5, 6], in addition to improved vaginal sensation and tightness [7, 8]. However, studies have also shown contradictory results [9, 10]. On the other hand, sexual pain disorders have been linked to hypertonicity (overactivity) of the PFM [4, 11], and different treatment strategies including a psychosocial approach, relaxation, and stretching techniques of the PFM and perineal tissue especially in postpartum women have been postulated [12].

The PFM comprise a superficial layer: ischiocavernosus, bulbospongiosus, and the transverse perineal muscles and a deep layer including the levator ani muscles: (the puboviseralis (pubococcygeus), the puborectalis, and the iliococcygeus muscles) [13, 14]. It is assumed that in addition to the changes in muscle tone set by the emotional motor system, it is especially the levator ani muscles and the bulbocavernosus muscle that are thought to play an active part in the sexual response in human beings. Upon clitoral and cervical stimulation, a contraction of the levator ani muscle is seen, together with a simultaneous contraction of the bulbocavernosus muscle. This activity is mainly mediated through reflex contractions that may contribute to the achievement of the orgasmic stage as well as physical changes in the vagina preparing for penetration [15–17].

The theory behind the rationale for strength training of the PFM is that strength training may build up the structural support of the pelvic floor by elevating the

levator plate inside the pelvis to a higher location and by enhancing hypertrophy and stiffness of the PFM and adjacent connective tissue [18]. Braekken et al. [19] also showed that pelvic floor muscle training (PFMT) significantly reduced the levator hiatus area. Further, an increase in vascularization and neurogenic and musculogenic factors may potentially affect lubrication and sensation and enhancing orgasm [18, 20, 21]. However, FSD is multifactorial [4], and a positive effect of different physical therapy interventions, including PFMT in the treatment of sexual dysfunction may have several explanations, for example, psychosocial aspects such as improved self-esteem and self-acceptance, body awareness, and satisfaction.

The aim of this chapter is to give an overview of randomized controlled trials (RCTs) investigating the effect of different physical therapy interventions in the treatment of FSD [11, 22].

17.2 Methods

A systematic literature search was performed in the PubMed electronic database, the Cochrane systematic reviews, the PEDro database, and the AMED (Ovid) database in December 2014 (updated in January 2016) containing all relevant RCTs investigating the effect of different physical therapy interventions on FSD. Physical therapy was defined according to the Physiotherapy Evidence Database (PEDro) and the following treatments were included as relevant: acupuncture, education, electrotherapy/heat/cold, strength training, and stretching/mobilization/manipulation/massage. A broad search was performed to be able to find studies including relevant treatment methods and where FSD was not the primary aim of the study. The following combinations were used in the different databases: sexual function and pelvic floor, pelvic floor muscle training and postpartum, pelvic floor muscle training and female or sexual function, pelvic floor muscle and perineum or genitourinary system and pelvic and clinical trials, pelvic floor and female, female sexual dysfunction, and randomized controlled trial. Reference tracking of the eligible studies was also performed and included where appropriate. Chronic pain conditions of the pelvic floor were not included in this chapter. The methodological quality of the included trials was assessed using the PEDro quality score [23].

17.3 Results

The search revealed 640 articles, including duplicates. After an elimination process, we identified 14 RCTs on the effect of physical therapy interventions on FSD in either postpartum women [24–27], in women with pelvic organ prolapse (POP) [7, 28–30], in women with stress urinary incontinence (SUI) [31–33], in women with orgasmic disorders [34, 35], and in gynecological cancer patients [37]. Results from those trials are summarized in Table 17.1. There was a considerable variety in study

Table 17.1 Randomized controlled trials evaluating the effect of pelvic floor muscle training on female sexual function

Author/year	Design	Study population	Intervention period	Dosage	Follow-up	Loss to follow-up	Outcome measures	Results
Citak et al. (2010)	Single blinded RCT	118 primiparous women (mean age 22.6)	4th–7th month postpartum	*PFMT*: 10 reps increasing to 15 reps daily, 2 s contraction increasing to 5 and 10 s *Control*: no treatment	*PFMT*: telephone and meeting with nurse twice in the first month and once in the following months	Total: 43 (36.4 %) Training: 21 (17.8 %) Control: 22 (18.6 %)	FSFI. PFM strength assessed by digital palpation and manometer	Positive effect in favor of training group on arousal, lubrication, and orgasm. Improvement in PFM strength
Wilson and Herbison (1998)	RCT	230 incontinent primi/ multiparous women (mean age 28.4)	3–12 months postpartum	*PFMT*: 80–100 reps × 8–10 daily *Cones*: 20–100 g 15 min daily *PFMT and cones*: see above *Control group*: standard treatment as recommended at hospital, but no follow-up	*PFMT and cones*: PT meeting 3, 4, 6, and 9 months after delivery	Total: 85 (36.9 %) Intervention: 59 (25.7 %) Control:26 (11.3 %)	Postal questionnaire: pain, interest in and satisfaction with sex, arousal, ability to orgasm, adequacy of vaginal tone, and general feeling. PFM strength and endurance assessed by perinometer	No between-group difference
Bø et al. (2000)	RCT	59 women with SUI (mean age 50.7)	6 months	*PFMT*: 8–12×3 daily (near maximum PFM contractions) *Control*: no treatment	*PFMT*: weekly group training with PT	Total: 4 (6.8 %) Training: 4 (6.8 %) Control: 0	QoLS-N and B-FLUTS. PFM strength assessed with manometer	Less problems with sex life spoilt by urinary symptoms in favor of training group. Improved PFM strength

Brækken et al. (2014)	Partially blinded RCT	109 women with POP (mean age 48.9)	6 months	*PFMT*: 8–12 × 3 daily (near maximum PFM contractions) *Control*: no treatment	*PFMT*: individual training weekly/every other week with PT	Total: 2 (1.8 %) Training: 1 (0.9 %) Control: 1 (0.9 %)	POP-specific questionnaire, semi-structured interview, PFM function assessed by manometer	Improvement in sexual function in favor of training group. Improved PFM strength and endurance, not vaginal resting pressure
Liebergall-Wischnitzer et al. (2012)	RCT	187 women with SUI (mean age 46.7)	3 months	*PFMT*: 25 min exercises daily, rapid/prolonged/gradual contractions *Paula method*: 45 min exercises daily	*PFMT*: 6 group sessions with PT *Paula method*: weekly private session with instructor	Total: 61 (32.6 %) PFMT: 38 (20.3 %) Paula: 23 (12.3 %)	I-QOL and PISQ-12	Sexual function improved in both groups, no between-group differences
Mørkved et al. (2007)	Single blind RCT (Abstract)	280 primiparous women with and without UI followed up after 6 years (mean age at inclusion 27.5)	Btw 20th and 36th months of pregnancy for 12 weeks	*PFMT*: 6–8 s hold near maximum contraction with 3–4 fast contractions in 4 series. Resting period was about 6 s. Home training 8–12 reps × 2 daily *Control*: no treatment	*PFMT*: weekly group training led by PT	Total: 106 (38 %)	Postal questionnaire: satisfaction with sex after delivery	Improved satisfaction in favor of training group

(continued)

Table 17.1 (continued)

Author/year	Design	Study population	Intervention period	Dosage	Follow-up	Loss to follow-up	Outcome measures	Results
Yang et al. (2012)	RCT (pilot)	34 women with gynecological cancer (mean age 52.4)	4 weeks	*Biofeedback*: 20 min with 40 cycles with 10 s of maximum activity followed by 20 s of relaxation. 20 min with an intensive core exercise session. Home training 10 reps × 2 daily *Control*: no treatment	*Biofeedback*: 45-min exercise session and a 30-min counseling session per week for 4 weeks led by PT	Total: 10 (29.4%) Training: 5 (14.7%) Control: 5 (14.7%)	Australian pelvic floor questionnaire on sexual function PFM strength assessed with perinometer	Improved sexual function, numbers being sexually active and PFM strength increased in favor of the training group. Improved PFM strength
Hagen et al. (2014)	Parallel group, multicenter RCT	477 women with symptomatic POP (mean age 56.8 years)	16 weeks	*PFMT*: individualized: 10 s maximum hold × 10 and 50 fast contractions × 3 daily *Control*: no treatment	*PFMT*: 5 individualized sessions led by PT	6 months: Training: 36 (16%) Control: 36 (16%) 12 months: Training: 75 (33.3%) Control: 77 (34.7%)	PISQ-12	Sexual scores (interference of prolapse symptoms with sex life) improved at 6 months after intervention in favor of training group. 12 months after intervention no difference

Eftekhar et al. (2014)	RCT	90 women with grade <3 POP (mean age 36.5 years)	8 weeks	*Treatment*: vaginal and anal biofeedback, infrared, reinforcement exercises, and relaxation including PFMT. PFMT included 6–8 s of contractions with 6 s rest in between for 15 min × 3 daily *Surgery*: rectocele repair	*Treatment*: home training taught by PT	Total: 0 %	FSFI	Physical therapy, improved orgasm, and severe dyspareunia compared to surgical group. Libido and arousal improved in both groups with no between-group differences
Wiegersma et al. (2014)	RCT	287 women with symptomatic, mild POP (mean age 64.3 years)	3 months	*Treatment*: PFMT, myofeedback, electrical stimulation, the knack, lifestyle advice (diet, body weight, toilet habits), PFM relaxation, and general relaxation. PFMT: 2–3 times daily, 3–5 × per week *Control*: no treatment	*Treatment*: once a week with PT at first, then every 2nd and 3rd week with an average of 7 visits	Total: 48 (16.7 %) Training: 31 (10.8 %) Control: 17 (5.9 %)	PISQ 12 PFM function (ability to contract and relax) measured by digital palpation	No effect and no improvement of PFM function

(continued)

Table 17.1 (continued)

Author/year	Design	Study population	Intervention period	Dosage	Follow-up	Loss to follow-up	Outcome measures	Results
Chambless et al. (1984)	RCT	36 women with orgasmic disorder (mean age 27.4 years)	6 weeks	*PFMT*: 10 min daily *Placebo*: 10 nonsexual images concerning vaginal sensations 10 min daily *Control*: no treatment	*PFMT and placebo*: weekly mail and telephone contact	Total: 21 (58.3 %) PFMT: 8 (22.2 %) Placebo: 10 (27.7 %) Control: 3 (8.3 %)	SAI-E (arousal, anxiety, satisfaction), WSQ (orgasmic responsiveness, frequency of orgasm, stimulation), and expectancy due to treatment or assessment only. PFM strength assessed with perinometer	Higher expectancy scores in PFMT and placebo, but no between-group differences. All groups improved in orgasm, but no between-group differences. No improvement in strength
Trudel and Saint-Laurent (1983)	RCT	12 women with orgasmic disorder (mean age not stated)	8 weeks	*PFMT*: 3 s contract and 3 s relax with increasing intensity for 20 min daily *Sexual awareness*, *relaxation*, *breathing* (*SARB*): different exercises for 20 min daily	*Both groups*: home training with weekly phone contact with experimenter	0 %	SAI, SII, clinical questionnaire (sexual reactions, stimuli needed to reach orgasm)	No effect in main outcome: orgasmic responsitivity between groups. Higher scores for SARB group on sexual satisfaction, self-acceptance, and perceptual accuracy scale, but no between-group differences

Handa et al. (2011)	RCT	445 women with SUI, mean age 49.8 years	8 weeks (analysis done 3 months after intervention)	*Group 1*: continence pessary *Group 2*: behavioral therapy (pelvic floor muscle training and continence strategies) *Group 3*: combination therapy	*Group 1*: pessary fitted and used as desired to decrease UI *Group 2 and 3*: four visits over 8 weeks + home training with increasing difficulty over time	Total: 100 (22.5%)	Personal Experiences Questionnaire (SPEQ) (libido, arousal, and dyspareunia) Pelvic Organ Prolapse/Urinary Incontinence Sexual Function Questionnaire (PISQ-12) PFM function measured with palpation	Change in measured aspects of sexual function did not differ among treatment groups In those with improved SUI, the combined therapy group had improved sexual function compared to pessary group. In those with improved SUI, the behavioral therapy had improved sexual function compared to pessary group. In those with no improved SUI, groups were similar Change in PFM strength associated with improved SUI but not improved sexual function

(continued)

Table 17.1 (continued)

Author/year	Design	Study population	Intervention period	Dosage	Follow-up	Loss to follow-up	Outcome measures	Results
Tennfjord et al. (2015)	RCT	175 women (mean age 29.8) with and without symptoms	6 weeks postpartum to 6 months postpartum	*PFMT*: three sets of 8–12 close to maximum PFM contractions *Control*: standard treatment as recommended at hospital, but no follow-up	*PFMT*: weekly training with PT and daily PFMT at home	Total: 15 (8.6%) PFMT: 12 (13.8%) Control: 3 (3.4%)	ICIQ-FLUTSsex and ICIQ-VS PFM function assessed with manometer	Unadjusted subgroup analysis of women with a major defect of the levator ani muscle showed that women in the training group had 45% less risk of having the symptom "vagina feels loose or lax" compared with the control group. No association with this symptom and change in PFM variables was found

SUI stress urinary incontinence, *POP* pelvic organ prolapse, *PT* physical therapist, *UI* urinary incontinence, *RCT* randomized controlled trials, *FSFI* Female Sexual Function Index, *QoLS-N* Quality of life scale, *B-FLUTS* Bristol Female Lower Urinary Tract Symptoms, *I-QOL* Incontinence Quality of Life Questionnaire, *PISQ-12* Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire, *SAI-E* sexual arousal inventory-expanded form, *WSQ* Women's Sexuality Questionnaire, *SAI* sexual arousal inventory, *SII* sexual interaction inventory, *ICIQ-FLUTSsex* International Consultation on Incontinence Modular Questionnaire sexual matters module, *ICIQ-VS* International Consultation on Incontinence Modular Questionnaire—Vaginal symptoms questionnaire

population, type of intervention, training dosage, length and type of follow-up, outcome measures, and assessment methods of PFM. In a primary prevention study, Citak et al. [24] was the only study investigating effect of PFMT in a healthy population, making it difficult to compare and to generalize the findings from the published studies. The search strategy also revealed two nonrandomized trials in women with urinary incontinence (UI) [38, 39]. Since we aimed for randomized trials only in this chapter, the two latter studies are not included. The PEDro score of the included studies is summarized in Table 17.2.

Nine studies reported an overall positive effect of physical therapy for different aspects of FSD: improved arousal, lubrication and orgasm, satisfaction with sexual life, increased control and stronger PFM, "tighter vagina," increased libido, less pain, increased sensibility and awareness around the pelvic floor, improved confidence, and partner's sexual satisfaction [7, 24, 26–29, 31, 33, 37]. Of these, six studies included PFMT as the only treatment option [7, 24, 26–28, 31]. The other three studies combined PFMT with biofeedback [37]; PFMT with biofeedback, infrared, reinforcement exercises, and relaxation [29]; and PFMT with continence strategies [33]. However, five studies showed contradictory results [25, 30, 32, 34, 35].

Table 17.2 PEDro quality score of randomized controlled trials evaluating effect of pelvic floor muscle training on female sexual function

Study	E	1	2	3	4	5	6	7	8	9	10	Total score
Citak et al. (2010)	+	+		+	−	−	+	−	−	+	+	5
Wilson and Herbison (1998)	+	+	−	+	−	−	+	−	−	+	+	5
Bø et al. (2000)	+	+	+	+	−	−	+	+	−	+	+	7
Brækken et al. (2014)	+	+	+	+	−	−	−	+	+	+	+	7
Liebergall−Wischnitzer et al. (2012)	+	+	+	+	−	−	+	−	−	+	+	6
Mørkved et al. (2007) Abstract	+	+	+	+	−	−	+	−	−	+	+	6
Yang et al. (2012) Pilot	+	+	−	+	−	−	+	+	−	+	+	6
Hagen et al. (2014)	+	+	+	+	−	−	+	−	+	+	+	7
Eftekhar et al. (2014)	+	+	−	+	−	−	−	+	+	+	+	6
Wiegersma et al. (2014)	+	+	+	+	−	−	+	+	+	+	+	8
Chambless et al. (1984)	+	+	−	−	−	−	+	−	−	+	+	4
Trudel and Saint-Laurent (1983)	+	+	−	+	−	−	−	+	−	+	+	5
Handa et al. (2008) [36]	+	+	+	+	−	−	+	−	−	+	+	6
Tennfjord et al. (2015)	+	+	+	+	−	−	+	+	+	+	+	8

E eligibility criteria specified, *1* subjects randomly allocated to groups, *2* allocation concealed, *3* groups similar at baseline, *4* subjects blinded, *5* therapist administering treatment blinded, *6* assessors blinded, *7* measures of key outcomes obtained from >85 % of subjects, *8* data analyzed by intention to treat, *9* statistical comparison between groups conducted, *10* point measures and measures of group variability provided

+ criterion clearly satisfied, − criterion is not satisfied. Total score is determined by counting the number of criteria satisfied, except "eligibility criteria satisfied" score is not used to generate the total score. Total scores are out of 10

Points 4 and 5 are unable to meet satisfactory criteria in all studies as subjects allocated to PFMT and therapists administering the treatment are aware of the intervention

What characterizes these studies are a high dropout rate [25, 32, 34] and low adherence to the treatment protocol [30]. In addition, the studies reporting no effect of the intervention did not carry out an assessment (or provided information about doing so) of a correct PFM contraction by observation and palpation of the PFM before the intervention (Fig. 17.1) [40, 41]. Further, the studies by Liebergall-Wischnitzer et al. [32] and Chambless et al. [34] found a positive effect in both groups, but the results did not reach a significant between-group difference. Possible explanations could be differences in training dosage and follow-up between the two intervention groups [32], whereas the study by Chambless et al. [34] provided both intervention groups with the same information and encouragement to discuss their concerns which may have led to both groups being more aware and responsive.

Seven studies reported improved PFM strength after intervention compared with the control group [7, 24, 26, 27, 31, 33, 37], but only the studies by Braekken et al. [7] and Yang et al. [37] reported the improvement in PFM strength to be associated with improvement in sexual function. It is therefore difficult to conclude whether

Fig. 17.1 Through observation and vaginal palpation, the physical therapist instructs the woman in how to contract the pelvic floor muscles correctly, defined as an inward movement of the perineum and a squeeze around the pelvic openings. This contraction is palpated as a squeeze around the examiners' finger and an inward lift (Photo: private with permission from patient)

there is a link between improved sexual function and improved PFM function. Given the multidimensional etiology of FSD, it might be that the PFM indirectly play a part in female sexual function and dysfunction. Eight studies included questions related to sexual pain disorders (pelvic floor pain and dyspareunia) [7, 24, 25, 27, 29, 31–33], and two studies reported effect of PFMT (PFMT alone or combined) in women with POP [9, 29]. However, the comparison group with surgery may not have been appropriate in the study by Eftekhar et al. [29], as the surgery group reported worsening of dyspareunia following rectocele repair. Due to the nature and complexity of this symptom [4], more studies are needed and factors related to success of physical therapy interventions must be identified. Long-term follow-up was performed in two studies, showing contradictory results [26, 28].

17.4 Summary

This chapter reflects some positive results of physical therapy in the treatment of FSD. However, it must be kept in mind the complexity of FSD and that a multidisciplinary approach often is necessary. Physical therapy may therefore be an important part in the treatment of these women. Furthermore, an individual approach is crucial in the evaluation and treatment of each woman. It is an urgent need of more high-quality studies in this field of women's health and a standardization of study populations, interventions, outcome measures, and assessment methods of PFM function. Factors related to success of physical therapy interventions (Table 17.3) must be identified before we can make conclusions upon which approach is most effective.

Table 17.3 Suggestions for physical therapy in women with sexual dysfunction

A thorough anamnesis including relevant background factors: other types of pelvic floor dysfunctions and treatment of those, natural functions, hormonal contraceptives, parity, delivery mode, level of physical activity, sleep disturbances, depression, sexual activity, experience and function, social factors, partner, medication, history of illness/disease, abuse. What caused the problem, onset, duration, location, frequency, intensity, factors that trigger/relieve the problem. Keep in mind red and yellow flags. Own expectations for treatment. Use of pain body mapping and pain scale (NRS or VAS). Note that specific conditions requires additional attention. These questions are more general for all conditions
Referral to other professions and multidisciplinary team
Assessment of external musculature and joints, use of pain body mapping and pain scale (NRS or VAS) including lumbar spine, pelvis, and hips. Assess for posture, mobility, tension, strength, temperature, sweat, relevant scars pain
Observation of skin color (redness), scar tissue, ability to contract and relax. Palpation with assessment of superficial and deep pelvic floor musculature, obturator internus, sacrotuberale ligament. Assess for ability to contract, relax, tender points, muscle tone, including hypo -and hypertonicity. Assessment of strength, local muscular endurance and vaginal resting tone using appropriate devices (ultrasound, electromyography, manometer, dynamometer)
Summarize findings and set up a treatment plan related to the findings including suggested physical therapy interventions: life style advice, body awareness, education, electrotherapy/heat/cold, strength training, stretching/mobilization/manipulation/trigger point treatment/massage/acupuncture may be tried. Consider follow-up and home exercise

References

1. Ishak WW, Tobia G. DSM-5 changes in diagnostic criteria of sexual dysfunctions. Reprod Sys Sex Disord. 2013;2(2):122–4.
2. Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. JAMA. 1999;281(6):537–44.
3. Skinner EM, Dietz HP. Psychological and somatic sequelae of traumatic vaginal delivery: a literature review. Aust N Z J Obstet Gynaecol. 2015;55(4):309–14.
4. Graziottin A. Female pelvic floor dysfunction and evidence-based physical therapy. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence based physical therapy for the pelvic floor: bridging science and clinical practice. 2nd ed. Philadelphia: Churchill Livingstone Elsevier; 2015. p. 243–70.
5. Martinez CS, Ferreira FV, Castro AAM, Gomide LB. Women with greater pelvic floor muscle strength have better sexual function. Acta Obstet Gynaecol Scand. 2014;93:457–502.
6. Lowenstein L, Gruenwald I, Gartman I, Vardi Y. Can stronger pelvic muscle floor improve sexual function? Int Urogynecol J. 2010;21(5):553–6.
7. Braekken IH, Majida M, Ellstrom Engh M, Bo K. Can pelvic floor muscle training improve sexual function in women with pelvic organ prolapse? A randomized controlled trial. J Sex Med. 2015;12(2):470–80.
8. Tennfjord MK, Hilde G, Staer-Jensen J, Siafarikas F, Engh ME, Bo K. Coital incontinence and vaginal symptoms and the relationship to pelvic floor muscle function in primiparous women at 12 months postpartum: a cross-sectional study. J Sex Med. 2015;12(4):994–1003.
9. Chambless DL, Stern T, Sultan FE, Williams AJ, Goldstein AJ, Lineberger MH, et al. The pubococcygens and female orgasm: a correlational study with normal subjects. Arch Sex Behav. 1982;11(6):479–90.
10. Baytur YB, Deveci A, Uyar Y, Ozcakir HT, Kizilkaya S, Caglar H. Mode of delivery and pelvic floor muscle strength and sexual function after childbirth. Int J Gynaecol Obstet. 2005;88(3):276–80.
11. Messelink B, Benson T, Berghmans B, Bo K, Corcos J, Fowler C, et al. Standardization of terminology of pelvic floor muscle function and dysfunction: report from the pelvic floor clinical assessment group of the International Continence Society. Neurourol Urodyn. 2005;24(4):374–80.
12. Rosenbaum TY. An integrated mindfulness-based approach to the treatment of women with sexual pain and anxiety: promoting autonomy and mind/body connection. Sex Relation Ther. 2013;28(1–2):20–8.
13. Ashton-Miller JA, Howard D, DeLancey JO. The functional anatomy of the female pelvic floor and stress continence control system. Scand J Urol Nephrol Suppl. 2001;207:1–7; discussion 106–25.
14. Ashton-Miller JA, DeLancey JO. Functional anatomy of the female pelvic floor. In: Bø K, Berghmans B, Mørkved S, van Kampen M, editors. Evidence-based physical therapy for the pelvic floor: bridging science and clinical practice. 1st ed. Philadelphia: Churchill Livingstone Elsevier; 2007. p. 19–33.
15. Vodusek DB. Neuroanatomy and neurophysiology of the pelvic floor muscles. In: Bø K, Berghmans B, Mørkved S, van Kampen M, editors. Evidence-based physical therapy for the pelvic floor: bridging science and clinical practice. 2nd ed. Philadelphia: Churchill Livingstone Elsevier; 2015. p. 35–41.
16. Achtari C, Dwyer PL. Sexual function and pelvic floor disorders. Best Pract Res Clin Obstet Gynaecol. 2005;19(6):993–1008; quiz A1–8.
17. Shafik A. The role of the levator ani muscle in evacuation, sexual performance and pelvic floor disorders. Int Urogynecol J Pelvic Floor Dysfunct. 2000;11(6):361–76.
18. Bø K. Pelvic floor muscle training is effective in treatment of female stress urinary incontinence, but how does it work? Int Urogynecol J. 2004;15:76–84.

19. Braekken IH, Majida M, Engh ME, Bo K. Morphological changes after pelvic floor muscle training measured by 3-dimensional ultrasonography: a randomized controlled trial. Obstet Gynecol. 2010;115(2 Pt 1):317–24.
20. Berman J, Berman L, Kanaly K. Female sexual dysfunction: new perspectives on anatomy, physiology, evaluation and treatment. EAU Updat Ser. 2003;1(3):166–77.
21. Bø K, Aschehoug A. Strength training. In: Bø K, Berghmans B, Mørkved S, van Kampen M, editors. Evidence-based physical therapy for the pelvic floor: bridging science and clinical practice. 1st ed. Philadelphia: Churchill Livingstone Elsevier; 2007. p. 119–32.
22. Bo K, Frawley HC, Haylen BT, Abramov Y, Almeida FG, Berghmans B et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for the conservative and nonpharmacological management of female pelvic floor dysfunction. Int Urogynecol J. 2016. doi: 10.1007/s00192-016-3123-4.
23. Maher CG, Sherrington C, Herbert RD, Moseley AM, Elkins M. Reliability of the PEDro scale for rating quality of randomized controlled trials. Phys Ther. 2003;83(8):713–21.
24. Citak N, Cam C, Arslan H, Karateke A, Tug N, Ayaz R, et al. Postpartum sexual function of women and the effects of early pelvic floor muscle exercises. Acta Obstet Gynecol Scand. 2010;89(6):817–22.
25. Wilson PD, Herbison GP. A randomized controlled trial of pelvic floor muscle exercises to treat postnatal urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 1998;9(5):257–64.
26. Morkved S, Rommen K, Schei B, Salvesen KA, Bo K. No difference in urinary incontinence between training and control group six years after cessation of a randomized controlled trial, but improved sexual satisfaction in the training group. Neurourol Urodyn. 2007;26(5):667.
27. Tennfjord MK, Hilde G, Stær-Jensen J, Siafarikas F, Ellström Engh M, Bø K. Effect of postpartum pelvic floor muscle training on vaginal symptoms and sexual dysfunction—secondary analysis of a randomised trial. BJOG. 2016;123(4):634–42.
28. Hagen S, Stark D, Glazener C, Dickson S, Barry S, Elders A, et al. Individualised pelvic floor muscle training in women with pelvic organ prolapse (POPPY): a multicentre randomised controlled trial. Lancet. 2014;383(9919):796–806.
29. Eftekhar T, Sohrabi M, Haghollahi F, Shariat M, Miri E. Comparison effect of physiotherapy with surgery on sexual function in patients with pelvic floor disorder: a randomized clinical trial. Iran J Reprod Med. 2014;12(1):7–14.
30. Wiegersma M, Panman CM, Kollen BJ, Berger MY, Lisman-Van Leeuwen Y, Dekker JH. Effect of pelvic floor muscle training compared with watchful waiting in older women with symptomatic mild pelvic organ prolapse: randomised controlled trial in primary care. BMJ. 2014;349:g7378.
31. Bo K, Talseth T, Vinsnes A. Randomized controlled trial on the effect of pelvic floor muscle training on quality of life and sexual problems in genuine stress incontinent women. Acta Obstet Gynecol Scand. 2000;79(7):598–603.
32. Liebergall-Wischnitzer M, Paltiel O, Hochner Celnikier D, Lavy Y, Manor O, Woloski Wruble AC. Sexual function and quality of life of women with stress urinary incontinence: a randomized controlled trial comparing the Paula method (circular muscle exercises) to pelvic floor muscle training (PFMT) exercises. J Sex Med. 2012;9(6):1613–23.
33. Handa VL, Whitcomb E, Weidner AC, Nygaard I, Brubaker L, Bradley CS, et al. Sexual function before and after non-surgical treatment for stress urinary incontinence. Female Pelvic Med Reconstr Surg. 2011;17(1):30–5.
34. Chambless DL, Sultan FE, Stern TE, O'Neill C, Garrison S, Jackson A. Effect of pubococcygeal exercise on coital orgasm in women. J Consult Clin Psychol. 1984;52(1):114–8.
35. Trudel G, Saint-Laurent S. A comparison between the effects of Kegel's exercises and a combination of sexual awareness relaxation and breathing on situational orgasmic dysfunction in women. J Sex Marital Ther. 1983;9(3):204–9.
36. Handa VL, Cundiff G, Chang HH, Helzlsouer KJ. Female sexual function and pelvic floor disorders. Obstet Gynecol. 2008;111(5):1045–52.

37. Yang EJ, Lim JY, Rah UW, Kim YB. Effect of a pelvic floor muscle training program on gynecologic cancer survivors with pelvic floor dysfunction: a randomized controlled trial. Gynecol Oncol. 2012;125(3):705–11.
38. Beji NK, Yalcin O, Erkan HA. The effect of pelvic floor training on sexual function of treated patients. Int Urogynecol J Pelvic Floor Dysfunct. 2003;14(4):234–8; discussion 8.
39. Zahariou AG, Karamouti MV, Papaioannou PD. Pelvic floor muscle training improves sexual function of women with stress urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(3):401–6.
40. Kegel AH. Stress incontinence and genital relaxation; a nonsurgical method of increasing the tone of sphincters and their supporting structures. Ciba Clin Symp. 1952;4(2):35–51.
41. Bo K, Hagen RR, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of female stress urinary incontinence. II. Validity of vaginal pressure measurements of pelvic muscle strength and the necessity of supplementary methods for control of correct contraction. Neurourol Urodyn. 1990;9(5):479–87.

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E. Costantini et al. (eds.), *Female Sexual Function and Dysfunction*,
DOI 10.1007/978-3-319-41716-5