

13.6.4 Sexual dysfunction

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section 13 Endocrine disorders 2408 percutaneous drainage under ultrasound guidance. In smokers, inflammation around the ducts, known as periductal mastitis, leads to an abscess formation if secondary infection occurs. This can progress to form a mammary fistula. Granulomatous mastitis is uncommon, the diagnosis being made after excluding infection, especially tuberculosis, and sarcoidosis. Mondor's disease is the name given to superficial thrombophlebitis of a chest wall vein. In the breast this results in a tender cord-like thickening running vertically and superficially. Benign conditions of the male breast Gynaecomastia, which is often unilateral, is hypertrophy of the ductal and stromal tissue of the male breast and is a common finding at puberty and in older men with failing testicular function. Pseudogynaecomastia, which is increase in the fat underlying the male breast, is more common than true gynaecomastia. This may be idiopathic, but a careful history of prescribed and recreational drugs may be fruitful. Common causes are digoxin, anabolic steroids, heroin, spironolactone, omeprazole, and methyldopa. Testicular tumours can cause gynaecomastia, which is also seen in Klinefelter's syndrome. Other benign conditions of the male breast are very rare. Male breast cancers account for less than 1% of all breast cancers in the United Kingdom, but suspicious features such as a hard, rugged lump in the breast should prompt triple assessment to exclude malignancy.

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13.6.4 Sexual dysfunction Ian Eardley ESSENTIALS Male sexual dysfunction, particularly erectile dysfunction, is common and becomes commoner with increasing age. It is often associated with cardiovascular disease and its risk factors. First line management should seek to identify and treat causative risk factors along with oral pharmacotherapy with a phosphodiesterase type 5 inhibitor. Premature ejaculation is a common but poorly understood condition that can be treated effectively by psychosexual therapy or by the use of selective serotonin reuptake inhibitors. Penile deformity is relatively uncommon and is usually due to Peyronie's disease. It is possible to treat established disease by injection of collagenase, but for most patients who actually require

treatment, surgical correction is the appropriate therapy. Prolonged erection or priapism is a rare medical emergency that requires urgent therapy or else erectile function may be lost. In women, sexual desire disorders are commoner in older postmenopausal women and a new treatment, flibanserin, has recently been licensed for this indication. Sexual arousal disorders in women also become more common postmenopausally and have a multifactorial aetiology. Treatment should be directed at the aetiological factor in the first instance; trials of oral pharmacotherapy have been disappointing. While true vaginismus is uncommon, dyspareunia is relatively common. Again, there may be a multifactorial aetiology, with vulvar vestibulitis being perhaps the most common aetiological condition. Treatment is often unsatisfactory.

Male sexual dysfunction Male sexual function is a complex neurovascular event which can be affected by several disease processes that result in problems of penile erection, ejaculatory difficulties, and disorders of desire. Of these, the commonest dysfunctions encountered in clinical practice are erectile dysfunction, penile deformity, prolonged erections, and rapid (or premature) ejaculation. Erectile dysfunction (ED) is defined as 'The inability to attain or maintain an erection satisfactory for sexual intercourse'. It is a very common symptom which affects around 50% of men over the age of 40 years. The prevalence increases with age, and there are associations with a range of conditions including diabetes, hypertension, dyslipidaemia, and depression. The first modern (and perhaps most important) epidemiological study was the Massachusetts Male Aging study which reported the age-related prevalence in North American men. A plethora of subsequent epidemiological studies have explored the issues. The physiology of penile erection is complex and involves co-ordinated interaction of the neurological, vascular, and endocrine systems. As such, there are many diseases of these systems that can lead to the development of ED, some of which are listed in Table 13.6.4.1. It is now recognized that in most men with ED, both psychogenic and organic risk factors coexist, albeit to varying degrees. Certainly secondary 'performance related anxiety' often complicates the pathophysiology of men who have a primary organic aetiology. The commonest cause of ED is that which arises secondary to vascular disease, with ED being a common coexisting symptom in men with hypertension, diabetes, hyperlipidaemia, and cardiovascular disease. Indeed, there is emerging evidence that ED may be one of the very earliest features of systemic vascular disease. Since ED is a self-declared symptom, the fundamental basis of diagnosis is the clinical history. Mild cases are characterized by a history of difficulty in maintaining an erection, while in more

13.6.4 Sexual dysfunction 2409 In severe cases, there may be difficulty in initiating an erection or even be loss of erections altogether. Assessment should seek to identify treatable causes (Tables 13.6.4.1, 13.6.4.2) and remediable risk factors. A predominantly psychogenic aetiology is suggested by the continued presence of nocturnal or early morning erections. Current guidelines suggest a focussed physical examination of the genitalia, assessment of secondary sexual characteristics, and assessment of blood pressure. More extensive genitourinary and vascular examination is only performed when clinically indicated. Baseline investigations should include a fasting blood sugar, a lipid screen, and an assessment of a serum testosterone. Further assessment is usually unnecessary, although in selected cases vascular investigations (including colour Doppler assessment of penile vasculature) and more complex endocrine investigations might be indicated. In a few patients, specific remedies can cure ED. For instance, in men with a predominantly psychogenic aetiology, psychosexual counselling is often valuable. In men with an endocrine cause (such as hypogonadism or hyperprolactinaemia), appropriate endocrine therapy is also helpful. Finally, in men who have developed ED following pelvic or perineal trauma,

reconstructive vascular surgery may be effective. In most men, however, treatment is symptomatic, rather than aiming to cure the underlying problem. Given the prevalence of secondary psychogenic problems, patient education and counselling are always valuable. Management of risk factors is an important part of patient management. Those with hypertension should have their blood pressure optimally controlled, with emerging evidence that the angiotensin receptor antagonists actually improve erectile function, while many other drugs used for hypertension have an adverse effect upon erections. Similarly, there is some evidence that improved diabetic control improves erections, and there are some studies suggesting that lifestyle interventions can also improve erectile function. However, the improvements seen are usually modest and some form of pharmacological treatment is usually required. The mainstay of therapy in most men who present with ED will be oral therapy using a phosphodiesterase type 5 inhibitor (PDE5i). While sildenafil was the first such licensed preparation, three other drugs—tadalafil, vardenafil, and avanafil—have been marketed in recent years in Europe and North America. There appears to be little difference in efficacy and tolerability between these drugs, with the major differences relating to pharmacokinetics. The drugs are usually used in an 'on demand' fashion, with ingestion 1–2 hours prior to sexual activity. Tadalafil can be used in a daily dosing regimen given its extended half-life. They are ineffective in the absence of sexual stimulation and a heavy meal may delay absorption.

Table 13.6.4.1 Common causes of erectile dysfunction

Systemic disease	Neurological disease	Multiple sclerosis	Spinal cord injury	Multiple system atrophy
Lumbar disc prolapse	Cauda equine syndrome	Peripheral autonomic neuropathy (e.g. diabetes)	Vascular disease	Atherosclerosis
Hypertension	Hyperlipidaemia	Diabetes	Endocrine disease	Diabetes mellitus
Hypogonadism	Hyperprolactinaemia	Thyrotoxicosis	Psychiatric disease	Depression
Anxiety states	Iatrogenic	Pelvic surgery (with nerve damage)	Pelvic radiotherapy	Drugs

(see Table 13.6.4.2) Multifactorial aetiologies Renal failure

Table 13.6.4.2 Drugs that can cause sexual dysfunction in men

Drug type	Drug or class of drug	Effect
Antihypertensive drugs	Diuretics	ED
β -blockers	Centrally acting antihypertensive agents, e.g. clonidine, methyl DOPA	ED
Centrally acting agents	Phenothiazines	ED
Butyrophenones	Serotonin reuptake inhibitors	ED
Tricyclic antidepressants	Phenytoin	ED, reduced libido, ejaculatory dysfunction
ED, reduced libido	ED, reduced libido	ED
Endocrine drugs	LHRH analogues	ED
Antiandrogens	Oestrogens	ED, reduced libido
ED, reduced libido	ED, reduced libido	ED
Recreational drugs	Alcohol	ED
Marijuana	Cocaine	ED
Opiates	Amphetamines	ED
Anabolic steroids	ED, reduced libido, ejaculatory dysfunction	ED
ED, reduced libido	ED, reduced libido	ED
Other drugs	Cimetidine	ED
Metoclopramide	Digoxin	ED, reduced libido
ED, reduced libido	ED	ED

section 13 Endocrine disorders 2410 70–80% of men will respond to such therapy, although the response rate is lower in certain patient groups, such as diabetics and in men who have undergone radical pelvic surgery. Side effects include headache, flushing, indigestion, and nasal congestion, although these are usually mild and well tolerated. Prolonged erections are extremely rare with these drugs. Extensive research has not identified any significant cardiac risk with this class of drugs, although they are contraindicated in men using nitrate medication, and sexual activity is inadvisable in men with unstable cardiac disease. A detailed summary of the management of ED in men with cardiac disease can be found in the 'Princeton Consensus Statements'. In men who fail to respond to oral PDE5i therapy, several approaches can be tried, although there is little evidence that use of a different PDE5i is beneficial. Regular (daily) dosing appears to help some patients, the rationale reflecting experimental evidence that a PDE5i might improve endothelial function when taken regularly. Alternatively, optimization of coexistent medical conditions is occa-

sionally helpful in this respect. There is developing evidence that in some men with a low serum testosterone, the response to a PDE5i can be improved by normalizing the testosterone levels. In men who fail to respond to these manoeuvres, alternative therapies such as vacuum erection devices or intracavernosal self-injection of alprostadil are often effective. In a few patients, notably those with severe diabetes, even these treatments are ineffective or poorly tolerated, and under these circumstances insertion of a penile prosthesis may be indicated. Rapid

(premature) ejaculation Premature ejaculation (PE) occurs when a man ejaculates before he or his partner want climax to happen. For some men, the problem starts with their first sexual experience (primary PE). For others, it happens after a period of normal sexual functioning (secondary PE). In recent years there have been consensus panels that have agreed a unified definition of both acquired and lifelong PE as a male sexual dysfunction characterized by ejaculation which always or nearly always occurs prior to or within about one minute of vaginal penetration from the first sexual experiences (lifelong PE), or a clinically significant and bothersome reduction in latency time, often to about 3 minutes or less (acquired PE), associated with the inability to delay ejaculation on all or nearly all vaginal penetrations, and negative personal consequences such as distress, bother, frustration, and/or the avoidance of sexual intimacy. The aetiology of primary PE is poorly understood with many potential pathophysiological mechanisms being proposed and none proven. Acquired PE is commonly associated with erectile dysfunction and there are known associations with thyroid disease, alcohol abuse, opiate withdrawal, and possibly with prostatitis. Many men with PE do not seek treatment, although there is evidence that there can be deleterious effects upon self-esteem, interpersonal relationships, and upon quality of life. When patients do seek medical attention, it is important to look for coexistent sexual dysfunctions (erectile dysfunction is commonly present) and to identify potential causes, while it is also important to assess the degree to which relationship issues are important. Therapeutic options include sex therapy and psychotherapy, selective serotonin reuptake inhibitors (SSRIs), and topical anaesthetics. While psychological approaches are commonly successful in the short term, they typically do not lead to long term cure. There is longstanding data that 'off-label' use of SSRIs is an effective treatment for PE, with both on-demand and daily dosing regimens in use. The latter provides a greater prolongation of the intravaginal ejaculatory latency time (IELT), but the potential risks of regular dosing with these drugs are well documented. Recently, a short acting SSRI has been licensed in Europe for this indication and appears to prolong the IELT two- to threefold, while improving control and distress. Local anaesthetic gels or sprays are also potential treatments, but none are currently licensed for clinical use. Penile deformity and Peyronie's disease While young men or adolescents can occasionally present with a penile deformity that develops during adolescence (congenital curvature of the penis), the commonest cause of penile deformity is Peyronie's disease, which is a localized connective tissue disease of the penis leading to fibrotic plaque formation in the tunica albuginea of the corpus cavernosum. It was first described by Fallopius in 1561, although the condition is named after Francois Gigot De La Peyronie, surgeon to King Louis XV of France, who described it in 1743. While in pathological studies there is subclinical disease in over 20% of men, the clinical prevalence is around 1%. In some cases the condition can be familial, and there are associations with Dupuytren's contracture, plantar fasciitis, and tympanosclerosis. The peak incidence occurs in the sixth decade, but cases have been reported throughout adult life. The pathophysiology is poorly understood, but the most commonly held view suggests that there is damage of the tunica albuginea associated with microvascular trauma. This leads to extravasation and perivascular inflammation with a round cell infiltrate, which in turn leads to fibrin deposition and subsequently fibrosis. Patients present with a palpable plaque within the

penis which is most commonly felt on the dorsum, and there is a curvature of the penis, most commonly in the dorsal direction. The clinical picture usually has two distinct phases, the 'acute phase', characterized by painful erections with progressive change in plaque size and deformity, which typically last 9–18 months, and the subsequent 'chronic' phase where the deformity stabilizes (and occasionally improves) and pain with erection disappears. Numerous medical treatments have been tried in men with Peyronie's disease. The oral agents that are most commonly used in clinical practice are vitamin E, tamoxifen, colchicine, and Potaba (potassium para-aminobenzoate, a form of vitamin B), but controlled studies have failed to demonstrate a consistent benefit for any agent. The most commonly used intralesional (injectable) agents are steroids, verapamil, and collagenase—and following formal randomized controlled trials, the latter has been licensed for this indication. In patients in the chronic phase of the disease, collagenase is injected into the plaque on several occasions with evidence that the deformity is improved in many, even if it does not disappear completely. The cost of the treatment and the variability of the response currently limit the clinical utility of collagenase. Surgical treatment is reserved for those patients whose penile deformity prevents sexual activity or those in whom sexual activity is painful either for the patient or his partner because of the deformity. The disease needs to be stable prior to surgical intervention. This point is particularly important, since disease progression after corrective surgery should be avoided. The most common surgical

13.6.4 Sexual dysfunction 2411 procedure is a Nesbit's Procedure which involves surgical excision of an ellipse of a tunica albuginea on the opposite side of the penis to the plaque. The edges of the ellipse are apposed. Occasionally a grafting procedure can be performed whereby the plaque is incised and grafted typically with a patch of saphenous vein. In severe cases associated with coexistent erectile dysfunction, a penile prosthesis is indicated. Priapism A priapism is a penile erection which is unduly prolonged and which persists in the absence of a sexual stimulus. The classification of priapism divides cases into those where the aetiology is arterial (so-called high flow priapism), and those cases where the aetiology reflects reduced venous drainage of the corpus cavernosum (so-called low flow priapism). Low flow priapism is by far the most common form of priapism and reflects stasis of blood within the penis, with sludging within the cavernosal sinusoids and subsequent thrombosis. Ischaemia, hypoxia, and acidosis develop as a consequence, and this prevents contraction of the penile smooth muscle which in turn exacerbates the condition. Low flow priapism is a medical emergency and requires urgent treatment to avoid irreversible damage to the vascular endothelium and smooth muscle of the penis and its erectile capacity. While comprehensive evidence is lacking, there are data to suggest that if treatment is successful within 24 hours, 56% of men will recover erectile function, while if treatment is delayed beyond that only 11% will recover potency. Causes of low flow priapism are shown in Table 13.6.4.3. The commonest are intracavernosal injections, sickle cell disease, other hyperviscosity syndromes, and the use of some psychotropic drugs. Men with low flow priapism typically present with a painful erection, in contrast to high flow priapism which is usually painless, but a careful assessment should seek to differentiate the low flow from the high flow condition (Table 13.6.4.4) and to identify the underlying cause. Aspiration of penile blood reveals thick dark venous blood, which on blood gas analysis shows hypoxia, hypercapnia, and acidosis. Doppler scanning confirms lack of blood flow within the penis. Treatment for low flow priapism initially involves aspiration of enough blood via a wide bore cannula to produce detumescence and, if necessary, irrigation with warm heparinized normal saline (Fig. 13.6.4.1). If the erection reappears then intracavernosal injection of a smooth muscle α -agonist such as phenylephrine is appropriate, with concurrent monitoring of the systemic

blood pressure. If this fails, then a surgical shunting procedure is appropriate. If treatment is delayed too long (see earlier) then even shunting procedures fail and the only hope for long term potency is a penile prosthesis. When there is a coexistent condition, such as sickle cell disease, specific treatment may be appropriate alongside treatment of the priapism. However, the traditional regime of oxygen, intravenous hydration, and analgesia is often unsuccessful, and only in selected cases is exchange transfusion indicated. High flow or nonischaemic priapism develops following perineal trauma, when the penile artery is lacerated, resulting in a fistula between the artery and the sinusoidal spaces of the penis. The priapism may appear at the time of injury, but more commonly it develops some time later, following spasm of the artery. Because the blood flowing through the penis is arterial (i.e. there is no ischaemia), there

Table 13.6.4.3 Causes of low flow priapism

Type	Example
Hypercoagulability disorders	Sickle cell disease, Myeloma, Leukaemia, Thalassemia
Total parenteral nutrition	Drugs
Intracavernosal agents (e.g. alprostadil)	Psychotropic agents (e.g. phenothiazines, butyrophenones, trazadone), Anticoagulants (e.g. warfarin, heparin), Antihypertensives (e.g. α -blockers, calcium channel blockers)
Miscellaneous	Lumbar disc disease, Solid tumours affecting the base of the penis, Genitourinary sepsis, Amyloidosis

Table 13.6.4.4 Features that help to differentiate low flow from high flow priapism

Findings	Low flow priapism	High flow priapism
History of perineal trauma	Rare	Common
History of coexistent blood disorder	Sometimes	Rare
History of recent intracavernosal injection	Sometimes	Rare
Corpora cavernosa rigid	Usually	Rare
Penile pain	Usually	Rare
Cavernosal blood gases	Hypoxia, hypercapnia, acidosis	Similar to arterial blood
Colour Doppler penis	Sluggish or absent flow	Normal flow with evidence of arterial turbulence

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Initial measures

Aspiration
Irrigation
Injection
Surgery

- Shunting procedure
 - Inject 200 µg phenylephrine directly into corpus cavernosum at the 3 o'clock or 9 o'clock position (monitor BP)
 - If fails consider repeat
 - If fails again, consider 500 µg
 - Irrigate by injecting and aspirating warm 0.9% saline
 - Compress penis
 - Use wide bore needle (18 G or 20 G) inserted either through the lateral penile shaft or through the glans penis to aspirate 50 ml blood from each corpus cavernosum
 - Specific measures (see text)
 - Oral drugs may help (Terbutaline, pseudoephedrine)
 - Analgesia
- Fig. 13.6.4.1 Treatment of low flow priapism.** a Phenylephrine ampoules are available as 10 mg in 1 ml; dilute in 49 ml 0.9% saline to make a solution containing 200 µg/ml

section 13 Endocrine disorders 2412 can be some circumspection in relation to treatment. The diagnosis is usually made by Doppler scanning and subsequent selective penile arteriography, which typically shows an arterial blush in the penile artery. The treatment of choice is embolization of the fistula with autologous blood clot. There is a rare variant of ischaemic priapism called 'stuttering priapism' where men present with recurrent episodes of painful erections, usually at night. These episodes often resolve spontaneously. The commonest cause is sickle cell disease, but

in some cases there is no obvious explanation. Treatment is difficult, with a variety of approaches including androgen suppression being used. Female sexual dysfunction The sexual problems of women have, until recently, received much less attention from the medical and scientific community than male sexual problems. This is reflected in a less well-developed understanding of physiological and pathophysiological processes, in a less well-defined classification of dysfunctions, and in a smaller range of therapeutic interventions. One classification of female sexual dysfunction is shown in Table 13.6.4.5. Epidemiological studies, although relatively immature, suggest that female sexual disorders are common and that they relate in part to age and to hormonal status. Comorbidities are common and should be identified, when present. The different sexual dysfunctions commonly coexist. One contentious area involves the recent DSM-5 classification, which introduces the diagnosis of female sexual interest/arousal disorder, and eliminates the previous separate diagnoses of hypoactive sexual desire disorder and female sexual arousal disorder. This reclassification has been controversial and for ease of presentation the conditions are described here in a traditional format, since that is how much of the clinical data has been developed.

Sexual desire disorders Hypoactive sexual desire disorder (HSDD) can be defined as 'absent or diminished feelings of sexual interest or desire, absent thoughts or fantasies, and a lack of responsive desire'. This lack of interest will be greater than that which is normally seen with ageing and with the length of a relationship. Epidemiological studies suggest that it affects up to 32% of women under the age of 50 years. It becomes commoner with increasing age and with the onset of the menopause, whether natural or surgically induced. Around half the women who have this condition are 'distressed' by it, with the degree of distress diminishing with increasing age. Women with a surgically induced menopause are more likely to be distressed than those who have undergone a natural menopause. The physiology and pathophysiology of desire is poorly understood, but probably resides biologically in the limbic system of the brain, where hormonal influences, including oestrogens and androgens, are important. Other hormonal abnormalities including hyperprolactinaemia and thyroid dysfunction can also result in sexual desire disorders. The dominant neurotransmitters include dopamine, which is important in the seeking-appetite-lust system, and oxytocin. Aetiology is often difficult to ascertain. There may be biological factors including natural or premature menopause, urinary incontinence, alcohol excess, and recreational drug abuse, while there are certain drug classes that can be associated with HSDD, including psychotropic agents, antihypertensive agents, drugs with an endocrine mechanism, and anticholinergics (Table 13.6.4.6). Psychological factors including a history of sexual abuse, anxiety and (especially) depression are also important, and their interaction with the biological issues makes this condition notoriously difficult to treat. Finally, the close relationship to other female sexual disorders and to male sexual dysfunction means that these aspects should be explored. Clinical assessment includes a sexual history and a full medical history. A careful history will seek to identify any psychological or physical issues, and it is important to find out whether the problem is universal or situational, and whether it is acquired or lifelong. A careful gynaecological assessment is important and a full endocrine evaluation may be appropriate, which will include measurement of serum testosterone, dehydroepiandrosterone sulphate, prolactin, 17- β oestradiol, and SHBG.

Table 13.6.4.5 A classification of female sexual dysfunction

Category	Subcategory	Disorders of desire	Interest	desire disorders	Sexual aversion disorders	Disorders of arousal	Subjective arousal disorders	Genital arousal disorders	Combined subjective and genital arousal disorders	Persistent arousal disorder	Orgasmic disorders	Pain disorders	Dyspareunia	Vaginismus
Table 13.6.4.6	Drugs that cause sexual dysfunction in women	Medication	Desire disorder	Arousal disorder	Orgasm disorder	Psychotropics	Antipsychotics	Benzodiazepines	Lithium					

SSRIs Venlafaxine + + + + + + + + + + + CV drugs Beta blockers Clonidine Digoxin
 Spironolactone + + + + + + Hormonal preparations Contraceptive pill Antiandrogens Tamoxifen
 GnRH analogues + + + + + + + + Other H2 receptor antagonists Odomethicin Ketoconazole
 Phenytoin Anticholinergics Antihistamines + + + + + +

13.6.4 Sexual dysfunction 2413 Treatment has been difficult and often unsuccessful, partly because the aetiology is commonly multifactorial; partly because of the complex interaction with relationship issues; but also because the motivation to be treated is often low. Where physical causes can be identified, they should be treated, and indeed outcomes are best in the group of patients who have definite endocrine abnormalities and who are highly motivated. Recently a new agent, flibanserin, has been licensed in the United States for the treatment of this condition. Flibanserin is a non- hormonal 5-HT1A receptor agonist and 5-HT2A receptor antagonist. In two 24-week clinical trials, treatment was associated with significant improvements in sexual desire, increased sexually significant experiences, and a decrease in sexual distress. The most commonly reported adverse events were dizziness, nausea, feeling tired, sleepiness and trouble sleeping, and drinking alcohol while on flibanserin resulted in low blood pressure in some patients. Although there is some data regarding the use of testosterone for this indication, the evidence is mixed and its use is not recommended. Nonpharmacological treatments include sex therapy and cognitive behavioural therapy, but the results are often disappointing. Sexual aversion disorders Defined as 'severe anxiety or disgust at the thought of sexual activity', sexual aversion disorder may arise as a result of incest, rape, molestation, and psychological abuse. It may coexist with other anxiety disorders, and psychosexual therapy is the mainstay of treatment. Sexual arousal disorders Female sexual arousal includes the physiological responses of increased blood flow to the clitoris, the labia, and the vagina, leading to vasocongestion and engorgement. Vaginal lubrication appears to be a purely hydrostatic event with transudation from the vaginal capillaries into the extracellular space. The physiology of these responses is poorly understood, but involves parasympathetic activation, with release of several neurotransmitters of which VIP and nitric oxide are almost certainly the most important. There is concurrent relaxation of the vaginal smooth muscle with lengthening and dilation of the vagina. Systemically, there is an increase in the heart rate, flushing, and erection of the nipples. There are three categories of arousal disorder. Firstly, there is the so-called subjective arousal disorder, associated with a reduction in the feelings of sexual arousal (including sexual excitement and sexual pleasure), but with normal vaginal lubrication still occurring. Alternatively, there may be genital sexual arousal disorder, when there is a definite and reduced degree of genital arousal in response to a sexual stimulus. Finally, the two may be found in combination. Sexual arousal disorders affect up to 24% of women. They are commoner in older women, especially after the menopause, and the aetiology may be multifactorial. Psychological factors include reduced desire, sexual inhibition, anxiety, lack of intimacy, and male erection problems. Physical factors that may be important include endocrine abnormalities (e.g. reduced oestrogens, hyperprolactinaemia), vascular disease (e.g. diabetes), neuropathy (e.g. diabetes, multiple sclerosis), drugs (Table 13.6.4.6), iatrogenic problems (e.g. posthysterectomy, postpelvic radiotherapy) and pain disorders (e.g. genital pain such as vaginismus, bladder pain due to recurrent urinary tract infection (UTI) or interstitial cystitis). Assessment should involve a careful history and examination, seeking to identify the range of risk factors that are present in an individual. Investigations might include an endocrine screen and, when indicated, Doppler assessment or plethysmography can evaluate vaginal and clitoral blood flow. Treatment should be directed towards the predominant aetiological issues. Psychosexual methods are important for

cases where 'subjective' problems predominate. Physical treatments that may be of value include local lubricants, topical oestrogens (when vaginal atrophy is present and oestrogen deficiency is present), and local physical devices analogous to the vacuum erection devices used in male erection difficulties. Systemic hormone replacement therapy may be appropriate in some, but there are no licensed non hormonal therapies for this condition. Given the physiological finding that nitric oxide was important in the control of vaginal blood flow, it was thought that the phosphodiesterase inhibitors such as sildenafil might have a role, but the results of clinical trials were disappointing. Persistent sexual arousal disorder This is a poorly documented but uncommon condition characterized by persistent genital arousal in the absence of a sexual stimulus. The pathophysiology is unknown, and there is no recognized therapy. Orgasmic disorders in women Anorgasmia is reported to occur in up to 37% of women, while in some there may be marked delay or diminution in the intensity of the orgasm. The female orgasm is characterized by a transient sensation of intense pleasure, associated with rhythmic contractions of the pelvic floor musculature, often associated with uterine and anal muscular contractions. Although the nature of the orgasm changes with age (becoming shorter and less intense), there is no evidence that the prevalence of orgasmic disorders becomes commoner with increasing age. The physiology of the female orgasm is poorly understood, but in most cases involves clitoral stimulation in association with an intact sacral reflex arc. While it would seem logical that intact ascending neural pathways are necessary for a woman to experience an orgasm, patients with complete spinal cord transection can, on occasions, experience them. It has been suggested therefore that ascending fibres within the vagus nerve may be important. There are multiple psychological and cultural influences on the ability of a woman to experience an orgasm. Similarly, there are number of physical and psychological problems that influence the ability of a woman to experience orgasm, including neuropathy, endocrine changes, thyroid disease, drugs (Table 13.6.4.6) and the presence of a weak pelvic floor. Psychological and social issues can be important: for instance, there is an inverse relationship between the degree to which a woman holds serious religious views and her ability to experience an orgasm. From a physical perspective, arousal disorders can result in delayed or absent orgasm. Therapy is primarily psychosexual, particularly when the problem is lifelong. When the problem is more recent in onset and associated with a physically induced sexual arousal disorder, then therapy should be directed at the arousal disorder. No pharmacological agents have shown any value in the treatment of this condition.

section 13 Endocrine disorders 2414 Sexual pain disorders in women Dyspareunia is pain associated with attempted or complete vaginal entry, while vaginismus indicates difficulties in the woman allowing entry of a penis (or any other object) into the vagina, despite her desire for this to happen. It is traditional to separate these two conditions, although in reality they may overlap, both in causality and in clinical presentation. Dyspareunia is reported by up to 15% of sexually active women, and it becomes much commoner in the postmenopausal population. Vaginismus is much less common, affecting less than 1% of sexually active women. Dyspareunia can be caused by several different conditions (see Table 13.6.4.7), and while these conditions can also cause vaginismus as well, there are cases where no clear organic aetiology appears to exist. Clinically, it is important to identify such causes when they are present, and to identify and treat any sexual comorbidities. Vulvar vestibulitis is one of the most important causes of vaginismus and is characterized by pain with penetration or attempted penetration, tenderness of the vestibular area to even light touch, and erythema of the vestibular area. Its aetiology is unknown, but there is evidence of chronic inflammation, although the mechanism by which this arises is unclear.

Treatment is often unsatisfactory, and involves analgesia (perhaps including tricyclic antidepressants and gabapentin), preventative hygienic measures, and sexual abstinence. There is preliminary evidence that vaginal electromyography (EMG) biofeedback, pelvic floor physiotherapy, and possibly vestibulectomy may have a role in treatment, although controlled studies are required for confirmation. FURTHER READING Basson R (2006). Clinical practice: sexual desire and arousal disorders in women. *N Engl J Med*, 354, 1497–506. Basson R, et al. (2004). Revised definitions of women's sexual dysfunction. *J Sex Med*, 1, 40–8. BAUS Section of Andrology Genitourethral Surgery (2018). BAUS consensus document for the management of male genital emergencies: priapism. *BJU Int*, 121, 835–9. Broderick GA, et al. (2010). Priapism: pathogenesis, epidemiology, and management. *J Sex Med*, 7, 476–500. Brotto LA, et al. (2010). Women's sexual desire and arousal disorders. *J Sex Med*, 7, 586–614. Buvat J, et al. (2011). Hypogonadal men nonresponders to the PDE5 inhibitor tadalafil benefit from normalization of testosterone levels with a 1% hydroalcoholic testosterone gel in the treatment of erectile dysfunction (TADTEST study). *J Sex Med*, 8, 284–93. Clayton AH, Valladares Juarez EM (2017). Female sexual dysfunction. *Psychiatr Clin North Am*, 40, 267–84. Ciocanel O, Power K, Eriksen A (2019). Interventions to treat erectile dysfunction and premature ejaculation: an overview of systematic reviews. *Sex Med*, 7, 251–69. Eardley I (2013). The incidence, prevalence, and natural history of erectile dysfunction. *Sex Med Rev*, 1, 3–16. Eardley I, et al. (2010). Pharmacotherapy for erectile dysfunction. *J Sex Med*, 7, 524–40. Feldman HA, et al. (1994). Impotence and its medical and psychosocial correlates: results of the Massachusetts Male Aging Study. *J Urol*, 151, 54–61.

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Table 13.6.4.7 Causes of dyspareunia Organic Superficial and introital Infections (vulvitis, vulvar vestibulitis, cystitis, vaginitis) Hormonal (vaginal atrophy) Anatomic (fibrous hymen, vaginal agenesis) Muscular (hyperactivity of levator ani) Iatrogenic (postsurgical, postradiation) Neuropathic Deep Endometriosis Pelvic inflammatory disease Chronic pelvic pain syndrome Iatrogenic (postsurgical, postradiation) Psychological Comorbidity with other disorders of female sexual function Previous sexual abuse or rape Depression and anxiety Couple related Inadequate foreplay Couple conflicts Sexual dissatisfaction Anatomic compatibility issues

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