:
Title: *Professional Guide to Diseases, 9th Edition*

20

Sexual disorders

Introduction

Sexuality is an integral human function that's inevitably affected by many interrelated factors. Its expression reflects the interaction of all of the biological, psychological, and sociologic ingredients that affect a person's self-image and behavior.

Depending on these complex factors, human sexuality can be healthy and enriching, or it can be the source of mental and physical distress. A sexually healthy person is commonly defined as a person who:

- exhibits behavior that agrees with gender identity (persistent feeling of oneself as male or female)
- can participate in a potentially loving or committed relationship
- finds erotic stimulation pleasurable
- can make decisions about sexual behavior that are compatible with values and beliefs.

Hazards to sexual health

An important group of sex-related disorders results from infection that's transmitted through sexual contact. These disorders include human immunodeficiency virus infection, gonorrhea, syphilis, chlamydial infections, genital herpes, genital warts, trichomoniasis, chancroid, and lymphogranuloma

venereum. Sexually transmitted diseases (STDs) are among the most prevalent infections around the world; gonorrhea, chlamydial infections, and genital warts are approaching epidemic proportions in the United States.

Sexual dysfunction disorders, including arousal disorders, orgasmic disorders, and sexual pain disorders (dyspareunia and vaginismus), may be caused by a general medical condition, psychological factors, or a combination of factors, or they may be substance-induced. Other disorders have a definite physical etiology.

Gender identity disorders and paraphilias are sexual disorders whose diagnostic criteria are found in the *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition, Text Revision.

Physical assessment

Physical assessment, primarily a diagnostic tool, can also serve as an excellent opportunity for patient teaching.

- During examination of the female, evaluate breast development, pubic hair distribution, and the development of external genitalia. With gloved hands, use a speculum to examine internal genitalia, including the cervix and vagina. Palpate the uterus and ovaries.

ELDER TIP

Take special care when examining an older woman because atrophic changes of the vaginal mucosa may increase her discomfort during a pelvic examination. Use a small speculum because of the decreased vaginal size. To ease insertion, dampen the speculum with warm water; don't use a lubricant because it may alter Papanicolaou test results. Proceed slowly; abrupt insertion of the speculum may damage sensitive degenerating tissue.

- During examination of the male, check pubic and axillary hair distribution. With a gloved hand, palpate the penis, scrotum, prostate

gland, and rectum. Inspect the penis (shaft, glans, and urethral meatus) for lesions, swelling, inflammation, scars, or discharge. In the uncircumcised male, retract the foreskin to visualize the glans. Examine the scrotum for size, shape, and abnormalities, such as nodules or inflammation. Check for the presence of both testes (the left testis is typically lower than the right).

ELDER TIP

The testes of an older male may be slightly smaller than those of a younger male, but they should be equal in size, smooth, freely moveable, and soft without nodules.

- Inspect and palpate the inguinal canal; you shouldn't observe any bulging of tissues or organs. (See *Male sexual anatomy*.)

Sexual history

Careful assessment helps identify the cause of a sexual problem as psychological or physical. A sexual history provides the basis for prevention, diagnosis, and treatment.

- Ensure privacy, as for physical assessment. Allow sufficient time so that the patient doesn't feel rushed.
- Approach a sexual history objectively. Remember, sexual health is relative; avoid making assumptions or judgments about the patient's sexual activities.
- After listening to the patient, determine his level of sexual understanding and phrase your questions in language that he can understand. Avoid technical terms.
- Begin with the least threatening questions. Usually, a menstrual or urologic history helps lead into a sexual history.
- Inquire about what the patient accepts as normal sexual behavior. Ask about sexual needs and priorities and whether the patient can discuss them with a sex partner.
- Assess risk behavior concerning selection of sex partners and specific sexual practices.

- Ask about possible homosexual activity, which can influence the risk and treatment of some STDs.
- Ask the female patient if she has adequate lubrication during intercourse and if she has ever experienced orgasm or pain with sexual contact. Ask the male patient if he has ever had difficulties with erection or ejaculation.
- Ask about current or past contraceptive practices.
- Try to use the history therapeutically by encouraging the patient to express anxiety. Such fears may be alleviated simply by providing factual information and answering questions.

MALE SEXUAL ANATOMY

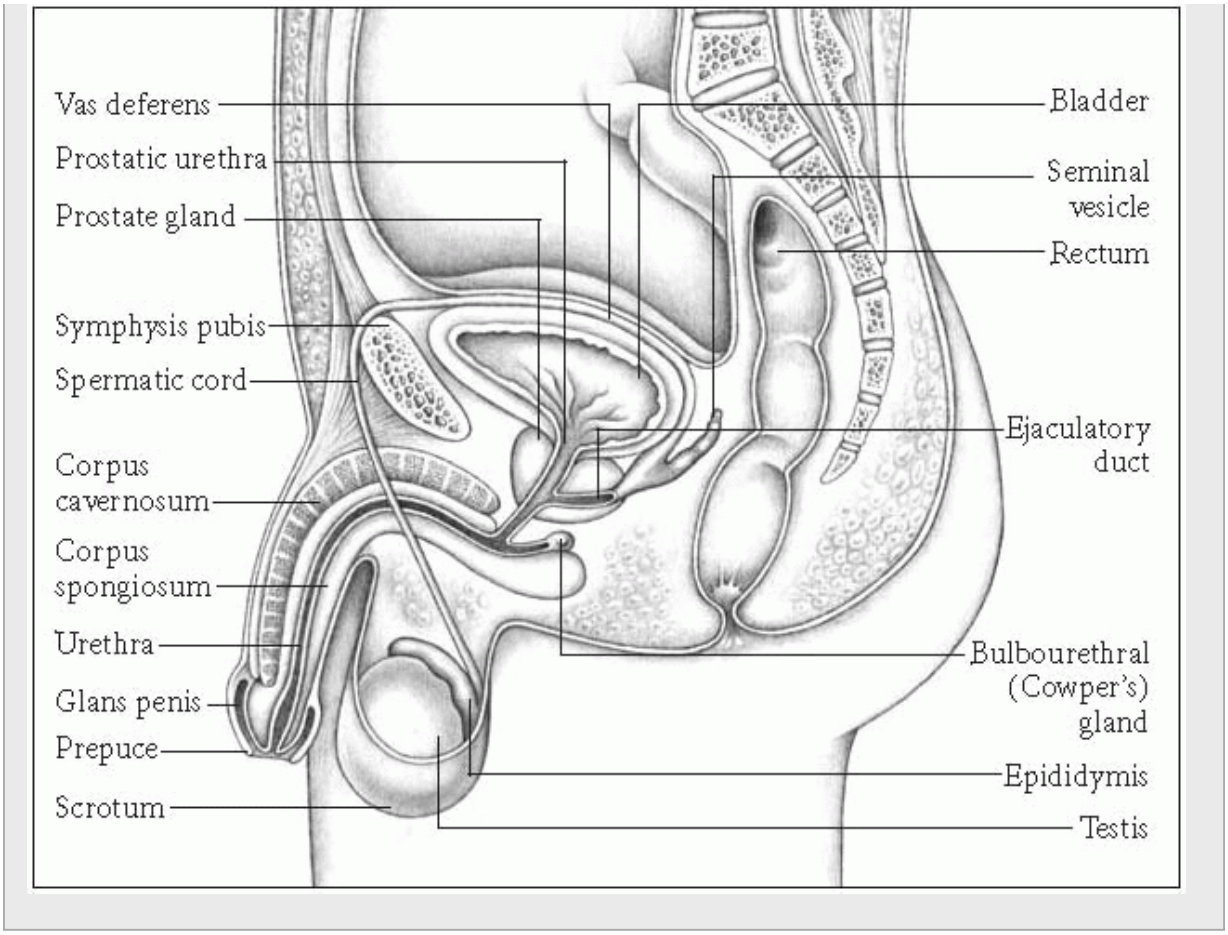
The *scrotum,* which contains the testes, epididymis, and lower spermatic cords, maintains the proper testicular temperature for spermatogenesis through relaxation and contraction. The penis consists of three cylinders of erectile tissue: two corpora cavernosa and the corpus spongiosum, which contains the urethra.

The *testes* (gonads, testicles) produce sperm in the seminiferous tubules, with complete spermatogenesis developing in most males by age 15 or 16. In the fetus, the testes form in the abdominal cavity and descend into the scrotum during the seventh month of gestation. The testes also secrete hormones, especially testosterone, in the interstitial cells (Leydig's cells). Testosterone affects the development and maintenance of secondary sex characteristics and sex drive. It also regulates metabolism, stimulates protein anabolism (encouraging skeletal growth and muscular development), inhibits pituitary secretion of the gonadotropins (follicle-stimulating hormone and interstitial cell-stimulating

hormone), promotes potassium excretion, and mildly influences renal sodium reabsorption.

The *vas deferens* connects the *epididymis,* in which sperm develop and mature for up to 6 weeks, and the *ejaculatory ducts.* (Vasectomy achieves sterilization by severing and interrupting the vas deferens, as both ends are tied off.) The *seminal vesicles,* two convoluted membranous pouches, secrete a viscous liquid of fructose-rich semen and prostaglandins that probably facilitates fertilization. The *prostate gland* secretes the thin alkaline substance that comprises most of the seminal fluid; this fluid also protects sperm from acidity in the male urethra and in the vagina, increasing sperm motility.

The *bulbourethral (Cowper's) glands* secrete an alkaline pre-ejaculatory fluid, probably similar in function to that produced by the prostate gland. The *spermatic cords* are cylindrical fibrous coverings in the inguinal canal, containing the vas deferens, blood vessels, and nerves.



Types of sex therapy

Sex therapy can be a vital therapeutic tool for treating sexual dysfunction. Before therapy begins, a history, a physical examination, and appropriate treatment must rule out organic causes of sexual dysfunction. The major forms of sex therapy include psychoanalysis, behavioral therapy, group therapy, classic (Masters and Johnson) therapy, and Kaplan's sex therapy. The type of therapy appropriate for the patient depends on his problems, needs, and finances.

SEXUALLY TRANSMITTED DISEASES

Gonorrhea

A common sexually transmitted disease, gonorrhea is an infection of the genitourinary tract (especially the urethra and cervix) and, occasionally, the rectum, pharynx, and eyes. Untreated gonorrhea can spread through the blood to the joints, tendons, meninges, and endocardium; in females, it can also lead to chronic pelvic inflammatory disease (PID) and sterility. After adequate treatment, the prognosis for both males and females is excellent, although reinfection is common. Gonorrhea is especially prevalent among young people and people with multiple partners, particularly those between ages 15 and 29. In these patients, suspect concomitant chlamydia infection.

Causes and incidence

Transmission of *Neisseria gonorrhoeae,* the organism that causes gonorrhea, usually follows sexual contact with an infected person. Children born of infected mothers can contract gonococcal ophthalmia neonatorum during passage through the birth canal. Children and adults with gonorrhea can contract gonococcal conjunctivitis by touching their eyes with contaminated hands.

The Centers for Disease Control and Prevention estimates that there are about 700,000 new cases of gonorrhea each year; only about half of these cases are reported to health care officials.

Complications

- Conjunctivitis
- Dermatitis
- Epididymitis
- Perihepatitis
- Proctitis
- Salpingitis
- Septic arthritis

Signs and symptoms

Although many infected males may be asymptomatic, after a 3- to 6-day incubation period, some develop symptoms of urethritis, including dysuria and purulent urethral discharge, with redness and swelling at the infection site. Most infected females remain asymptomatic but may develop inflammation and a greenish yellow discharge from the cervix—the most common gonorrheal symptoms in females. (See *What happens in gonorrhea*.)

Other clinical features vary according to the site involved:

- *urethra:* dysuria, urinary frequency and incontinence, purulent discharge, itching, and red and edematous meatus
- *vulva:* occasional itching, burning, and pain due to exudate from an adjacent infected area (symptoms tend to be more severe before puberty or after menopause)
- *vagina* (most common site in children older than age 1): engorgement, redness, swelling, and profuse purulent discharge
- *liver:* right upper quadrant pain in a patient with perihepatitis
- *pelvis:* severe pelvic and lower abdominal pain, muscle rigidity, tenderness, and abdominal distention. As the infection spreads, nausea, vomiting, fever, and tachycardia may develop in a patient with salpingitis or PID.

Other possible symptoms include pharyngitis, tonsillitis, rectal burning and itching, and bloody mucopurulent discharge.

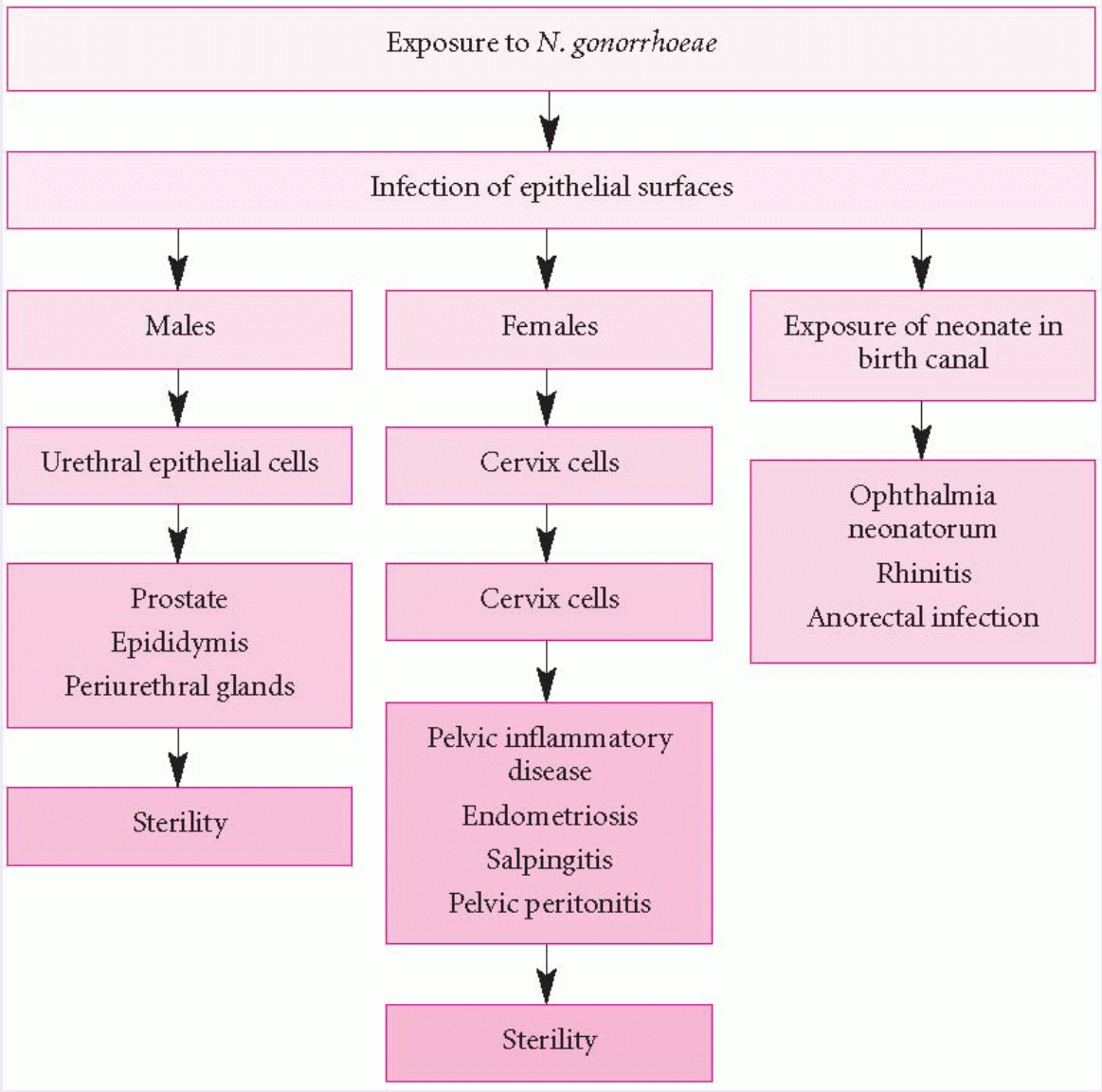
Gonococcal septicemia is more common in females than in males. Its characteristic signs include tender papillary skin lesions

on the hands and feet; these lesions may be pustular, hemorrhagic, or necrotic. Gonococcal septicemia may also produce migratory polyarthralgia and polyarthritis and tenosynovitis of the wrists, fingers, knees, or ankles. Untreated septic arthritis leads to progressive joint destruction.

PATHOPHYSIOLOGY

WHAT HAPPENS IN GONORRHEA

After exposure to *Neisseria gonorrhoeae,* the epithelial cells at the infection site become infected; then the disease begins to spread locally. The disease pattern depends on the individual infected and the infection site.



Signs of gonococcal ophthalmia neonatorum include lid edema, bilateral conjunctival infection, and abundant purulent discharge 2 to 3 days after birth. Adult conjunctivitis, most common in men, causes unilateral conjunctival redness and swelling. Untreated gonococcal conjunctivitis can progress to corneal ulceration and blindness.

Diagnosis

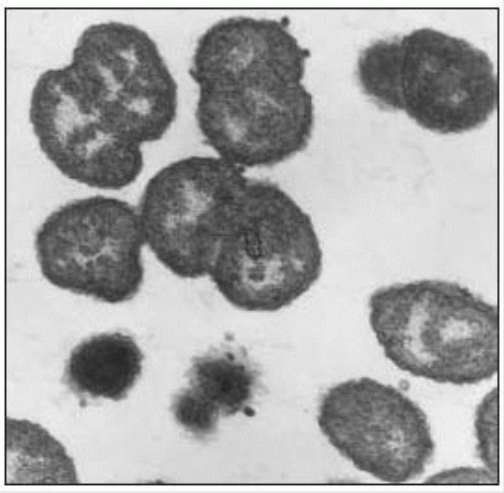
CONFIRMING DIAGNOSIS

A culture from the infection site (urethra, cervix, rectum, or pharynx), grown on a Thayer-Martin or Transgrow medium, usually establishes the diagnosis by isolating N. gonorrhoeae. (See Neisseria gonorrhoeae, *page 1222.) A Gram stain showing gram-negative diplococci supports the diagnosis*

and may be sufficient to confirm gonorrhea in males.

NEISSERIA GONORRHOEAE

In gonorrhea, microscopic examination reveals gram-negative diplococcus—*N. gonorrhoeae,* the causative organism.



Ligase chain reaction is an assay that can detect *N. gonorrhoeae* and *Chlamydia trachomatis* from urethral or cervical swabs. It allows for rapid diagnosis and offers improved sensitivity and specificity compared to swab specimen cultures.

Confirmation of gonococcal arthritis requires identification of gram-negative diplococci on smears made from joint fluid and skin lesions.

Complement fixation and immunofluorescent assays of serum reveal antibody titers four times the normal rate. Culture of conjunctival scrapings confirms gonococcal conjunctivitis.

Treatment

For adults and adolescents, the recommended treatment for uncomplicated gonorrhea caused by susceptible nonpenicillinase-producing *N. gonorrhoeae* is a single dose of ceftriaxone or cefixime; for presumptive treatment of concurrent *C. trachomatis* infection, doxycycline. Gonorrhea may also be treated with a single dose of azithromycin (Zithromax). Common alternative prescriptions may include cefuroxime, cefpodoxime proxetil, or erythromycin. A follow-up visit 7 days after treatment to recheck cultures and confirm the cure of infection is recommended, especially for women who are asymptomatic or may not have symptoms associated with the infection. A single dose of ceftriaxone and erythromycin is recommended for pregnant patients and those allergic to penicillin.

Treatment of gonococcal conjunctivitis requires a single dose of ceftriaxone, and lavage of the infected eye with saline solution once.

Routine instillation of 1% silver nitrate drops or erythromycin ointment into the neonate's eyes soon after delivery has greatly reduced the incidence of gonococcal ophthalmia neonatorum.

Special considerations

- Before treatment, establish whether the patient has any drug sensitivities, and watch closely for adverse drug reactions during therapy.
- Warn the patient that, until cultures prove negative, he's still infectious and can transmit gonococcal infection.
- If the patient has gonococcal arthritis, apply moist heat to ease pain in affected joints.
- Urge the patient to inform sexual contacts of his infection so that they can seek treatment, even if cultures are negative. Advise him to avoid sexual intercourse until treatment is complete.

- Report all cases of gonorrhea to local public health authorities for follow-up on sexual contacts. Examine and test all people exposed to gonorrhea as well as children of infected mothers.
- Routinely instill two drops of 1% silver nitrate solution or erythromycin ointment in the eyes of all neonates immediately after birth. Check the neonate of an infected mother for signs of infection. Take specimens for culture from the neonate's eyes, pharynx, and rectum.
- To prevent gonorrhea, tell patients to avoid anyone *suspected* of being infected. Tell them that abstinence is the only sure way to prevent gonorrhea. (See *Preventing gonorrhea*.)
- Report all cases of gonorrhea in children to child abuse authorities.

PREVENTION

PREVENTING GONORRHEA

To prevent gonorrhea, teach your patient the following:

- Tell the patient to avoid sexual contact until test cultures are negative and infection is gone.
- Advise the partner of an infected person to be treated even if the partner doesn't have a positive culture. Recommend that the partner avoid sexual contact with anyone until treatment is complete because reinfection is very common.
- Advise the patient and all sexual partners to be tested for human immunodeficiency virus and hepatitis B infection.
- Tell the patient to take anti-infective drugs for the length of time prescribed.
- To prevent reinfection, tell the patient to avoid sexual contact with anyone suspected of being infected, to use condoms during intercourse, to wash genitalia with

soap and water before and after intercourse, and to avoid sharing washcloths or using douches.
- Advise the patient to return for follow-up testing.

Chlamydial infections

Chlamydial infections—including urethritis in men and urethritis and cervicitis in women—are a group of infections that are linked to one organism: *Chlamydia trachomatis*. Trachoma inclusion conjunctivitis, a chlamydial infection that seldom occurs in the United States, is a leading cause of blindness in Third World countries. Lymphogranuloma venereum, a rare disease in the United States, is also caused by *C. trachomatis*. (See *Lymphogranuloma venereum,* page 1224.)

Untreated, chlamydial infections can lead to such complications as acute epididymitis, salpingitis, pelvic inflammatory disease (PID) and, eventually, sterility. Some studies show that a chlamydial infection in a pregnant woman is associated with spontaneous abortion and premature delivery.

Causes and incidence

Transmission of *C. trachomatis* primarily follows vaginal or rectal intercourse or orogenital contact with an infected person. Because symptoms of chlamydial infections commonly appear late in the disease's course, sexual transmission of the organism typically occurs unknowingly. Children born of mothers who have chlamydial infections may contract associated conjunctivitis, otitis media, and pneumonia during passage through the birth canal.

Chlamydial infections are the most common sexually transmitted diseases in the United States, affecting an estimated four million people in the United States each year.

Complications

- Epididymitis
- Neonatal death

- Pelvic inflammatory disease
- Premature rupture of membranes
- Preterm delivery
- Salpingitis
- Spontaneous abortion
- Sterility

Signs and symptoms

Both men and women with chlamydial infections may be asymptomatic or may show signs of infection on physical examination. Individual signs and symptoms vary with the specific type of chlamydial infection and are determined by the organism's route of transmission to susceptible tissue.

A woman with cervicitis may develop cervical erosion, mucopurulent discharge, pelvic pain, and dyspareunia.

LYMPHOGRANULOMA VENEREUM

A rare disease in the United States, lymphogranuloma venereum is caused by serovars L1, L2, or L3 of *Chlamydia trachomatis.* The most common clinical manifestation of LGV among heterosexuals, especially male patients, is enlarged inguinal lymph nodes (usually unilateral). These nodes may become fluctuant, tender masses. Regional nodes draining the initial lesion may enlarge and appear as a series of bilateral buboes. Untreated buboes may rupture and form sinus tracts that discharge a thick, yellow, granular secretion.

Women and homosexually active men may have proctocolitis or inflammatory involvement of perirectal or perianal lymphatic tissues, resulting in fistulas and strictures.

By the time most patients seek treatment, the self-limited genital ulcer that sometimes occurs at the inoculation site is no longer present. The diagnosis usually is made serologically and by excluding other causes of inguinal lymphadenopathy or genital ulcers.

The treatment of choice is doxycycline. Treatment cures infection and prevents ongoing tissue damage, although the patient may develop a scar or an indurated inguinal mass. Buboes may require aspiration or incision and drainage through intact skin.

A woman with endometritis or salpingitis may experience signs of PID, such as pain and tenderness of the abdomen, cervix, uterus, and lymph nodes; chills; fever; breakthrough bleeding; bleeding after intercourse; and vaginal discharge. She may also have dysuria.

A woman with urethral syndrome may experience dysuria, pyuria, and urinary frequency.

A man with urethritis may experience dysuria, erythema, tenderness of the urethral meatus, urinary frequency, pruritus, and urethral discharge. In urethritis, such discharge may be copious and purulent or scant and clear or mucoid.

A man with epididymitis may experience painful scrotal swelling and urethral discharge.

A man with *prostatitis* may have lower back pain, urinary frequency, dysuria, nocturia, and painful ejaculation.

A patient with *proctitis* may have diarrhea, tenesmus, pruritus, bloody or mucopurulent discharge, and diffuse or discrete ulceration in the rectosigmoid colon.

Diagnosis

A swab from the site of infection (urethra, cervix, or rectum) establishes a diagnosis of urethritis, cervicitis, salpingitis, endometritis, or proctitis. A culture of aspirated material establishes a diagnosis of epididymitis.

Antigen detection methods, including the enzyme-linked immunosorbent assay and the direct fluorescent antibody test, have long been used for identifying chlamydial infection. Tissue cell cultures, however, are more sensitive and specific. Newer nucleic acid probes using polymerase chain reactions are also commercially available and have become the diagnostic tests of choice.

Treatment

The recommended first-line treatment for adults and adolescents who have chlamydial infections is drug therapy with tetracycline, erythromycin, or azithromycin.

For pregnant women with chlamydial infections, erythromycin (stearate base) or azithromycin may be used.

Special considerations

- Practice standard precautions when caring for a patient with a chlamydial infection.
- Make sure that the patient fully understands the dosage requirements of prescribed medications for this infection.

- Stress the importance of completing the entire course of drug therapy even after the symptoms subside.
- Teach the patient to follow meticulous personal hygiene measures as recommended.
- Urge the patient to inform sexual contacts of his infection so that they can receive appropriate treatment.
- If required in your state, report all cases of chlamydial infection to the appropriate local public health authorities, who will then conduct follow-up notification of the patient's sexual contacts.
- Suggest that the patient and his sex partners receive testing for the human immunodeficiency virus.
- Tell the patient to return for follow-up testing.

- Check the neonate of an infected mother for signs of chlamydial infection. Obtain appropriate specimens for diagnostic testing.

PREVENTION

- *To prevent eye contamination, tell the patient to avoid touching any discharge and to wash and dry his hands thoroughly before touching his eyes.*
- *To prevent reinfection during treatment, urge the patient to abstain from sexual intercourse until he and his partner are free of infection.*

Genital herpes

Genital herpes is an acute inflammatory disease of the genitalia. The prognosis varies, depending on the patient's age, the strength of his immune defenses, and the infection site. Primary genital herpes is usually self-limiting but may cause painful local or systemic disease. (See *Understanding the genital herpes cycle,* page 1226.) In neonates and patients who are immunocompromised, such as those with acquired immunodeficiency syndrome, genital herpes is usually severe, resulting in complications and a high mortality.

Causes and incidence

Genital herpes is usually caused by infection with herpes simplex virus Type 2, but some studies report increasing incidence of infection with herpes simplex virus Type 1. This disease is typically transmitted through sexual intercourse, orogenital sexual activity, kissing, and hand-to-body contact. Pregnant women may transmit the infection to neonates during vaginal delivery if an active infection is present. Such transmitted infection may be localized (for instance, in the eyes) or disseminated and may be associated with central nervous system involvement.

An estimated 86 million people worldwide are thought to have genital herpes.

Complications

General

- Increased risk of contracting other STDs

During pregnancy

- Neonatal brain damage
- Neonatal blindness

Signs and symptoms

After a 3- to 7-day incubation period, fluid-filled vesicles appear, usually on the cervix (the primary infection site) and possibly on the labia, perianal skin, vulva, or vagina of the female and on the glans penis, foreskin, or penile shaft of the male. Extragenital lesions may appear on the mouth or anus. In both males and females, the vesicles, usually painless at first, will rupture and develop into extensive, shallow, painful ulcers, with redness, marked edema, tender inguinal lymph nodes, and the characteristic yellow, oozing centers.

Other features of initial mucocutaneous infection include fever, malaise, dysuria and, in females, leukorrhea. Rare complications (generally from extragenital lesions) include herpetic keratitis, which may lead to blindness, and potentially fatal herpetic encephalitis.

Diagnosis

Diagnosis is based on the physical examination and patient history. Helpful (but nondiagnostic) measures include laboratory data showing increased antibody titers, smears of genital lesions showing atypical cells, and cytologic preparations (Tzanck test) that reveal giant cells.

PATHOPHYSIOLOGY

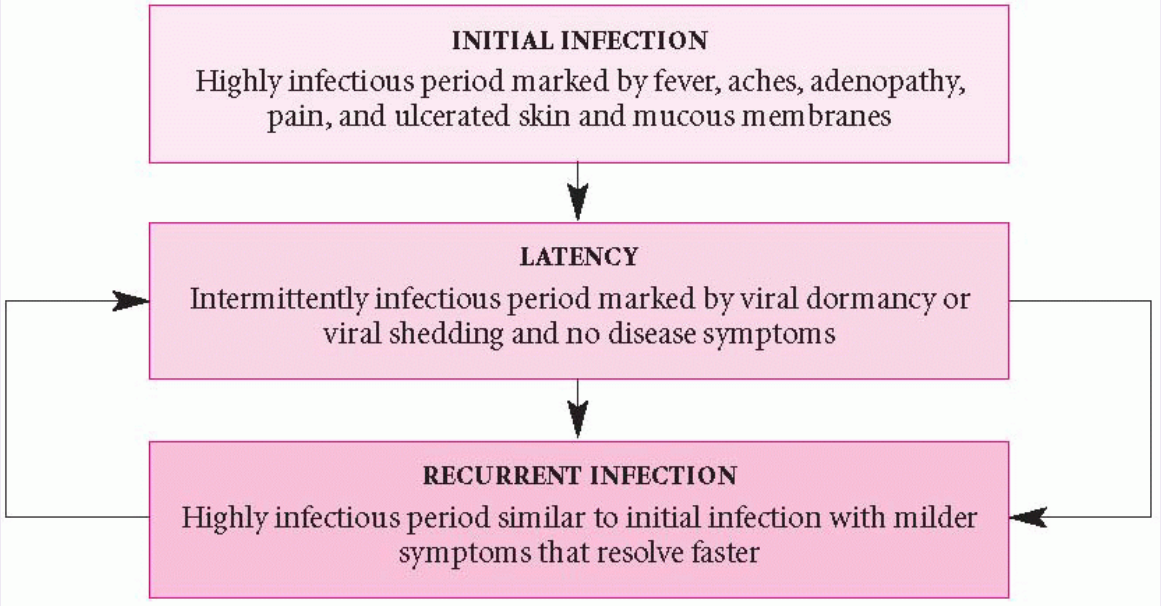
UNDERSTANDING THE GENITAL HERPES CYCLE

After a patient is infected with genital herpes, a latency period follows. The virus takes up permanent residence

in the nerve cells surrounding the lesions, and intermittent viral shedding may take place.

Repeated outbreaks may develop at any time, again followed by a latent stage during which the lesions heal completely. Outbreaks may recur as often as three to eight times yearly.

Although the cycle continues indefinitely, some people remain symptom-free for years.



CONFIRMING DIAGNOSIS

Diagnosis can be confirmed by demonstration of the herpes simplex virus in vesicular fluid, using tissue culture techniques, or by antigen tests that identify specific antigens.

Treatment

Acyclovir has proved to be an effective treatment for genital herpes. I.V. administration may be required for patients who are hospitalized with severe genital herpes or for those who are immunocompromised and have a potentially life-threatening herpes infection. Oral acyclovir may be prescribed for the patient with a first-time infection or recurrent

outbreak. Other agents include famciclovir, valacyclovir, and penciclovir; these drugs suppress symptoms but don't cure the infection. Daily prophylaxis with acyclovir reduces the frequency of recurrences by at least 50%, but this is only appropriate for a patient with frequent outbreaks and may not decrease transmission rate of the disease.

Foscavir, a powerful antiviral agent, is the treatment of choice for herpes strains that are severe in nature or have become resistant to acyclovir and similar drugs. Administered I.V., foscavir can have several toxic effects, such as reversible impairment of kidney function or induction of seizures. As with other antiviral drugs, this drug doesn't cure herpes.

Special considerations

- Encourage the patient to get adequate rest and nutrition and to keep the lesions dry.
- Tell the patient that warm baths may relieve the pain associated with genital lesions.
- Recommend gentle cleaning of the lesions with soap and water.

- Secondary infections of skin lesions by bacteria require a topical or oral antibiotic. Tell the patient to report worsening of lesions, indicating possible secondary infection, to the health care provider.
- Advise the patient to avoid sexual intercourse during the active stage of this disease (while lesions are present) and to use condoms during all sexual exposures. Urge him to have his sex partners seek medical examination.
- Advise the female patient to have a Papanicolaou test every 6 months.
- Refer patients to the Herpes Resource Center, which has local chapters nationwide, for support.

Genital warts

Genital warts (also known as *venereal warts* or *condylomata acuminata*) consist of papillomas with fibrous tissue overgrowth from the dermis and

thickened epithelial coverings. They're uncommon before puberty or after menopause. Certain types of human papillomavirus (HPV) infections have been strongly associated with genital dysplasia and, over a period of years (depending on the viral strain), with cervical neoplasia.

Causes and incidence

Infection with one of the more than 70 known strains of HPV causes genital warts, which are transmitted through sexual contact. The warts grow rapidly in the presence of pregnancy and commonly accompany other genital infections.

Each year, about 6 million people are infected with HPV in the United States.

Complication

- Genital tract dysplasia or cancer

Signs and symptoms

After a 1- to 6-month incubation period (usually 2 months), genital warts develop on moist surfaces: in males, on the subpreputial sac, within the urethral meatus and, less commonly, on the penile shaft; in females, on the vulva and on vaginal and cervical walls. In both sexes, papillomas spread to the perineum and the perianal area. These painless warts start as tiny red or pink swellings that grow (sometimes up to 10 cm) and become pedunculated. Typically, multiple swellings give them a cauliflower-like appearance. If infected, the warts become malodorous.

Most patients report no symptoms; a few complain of itching or pain.

Diagnosis

CONFIRMING DIAGNOSIS

Dark-field examination of scrapings from wart cells shows marked vascularization of epidermal cells, which helps to differentiate genital warts from condylomata lata associated with second-stage syphilis.

Applying 5% acetic acid (white vinegar) to the warts turns them white. Warts usually are diagnosed early by visual inspection; biopsy is indicated only when neoplasia is strongly suspected.

Treatment

Treatment is mostly for cosmetic reasons and should be guided by the patient's preference. Treatment aims to remove exophytic warts and to ameliorate signs and symptoms. Topical drug therapy (10% to 25% podophyllum in compound benzoin tincture, trichloroacetic acid, or dichloroacetic acid) removes small warts. (Podophyllum is contraindicated in pregnancy.) Warts larger than 2.5 cm are generally removed by carbon dioxide laser treatment, cryosurgery, or electrocautery. Other treatments include Podofilox, Imiquimod, interferon, and combined laser and interferon therapy. No therapy has proved effective in eradicating HPV; relapse is common.

Special considerations

- Tell the patient to remove the podophyllum with soap and water 4 to 6 hours after applying it.
- Encourage the patient's sex partners to be examined for HPV, human immunodeficiency virus, and other sexually transmitted diseases (STDs).
- Advise the female patient to have a Papanicolaou test every year.

PREVENTION

Recommend the use of condoms, and tell the patient that abstinence is the only sure way to avoid genital warts and other STDs.

Syphilis

A chronic, infectious, sexually transmitted disease, syphilis begins in the mucous membranes and quickly becomes systemic, spreading to nearby lymph nodes and the bloodstream. This disease, when untreated, is

characterized by progressive stages: primary, secondary, latent, and late (formerly called *tertiary*). Untreated syphilis leads to long-term health problems, but the prognosis is excellent with early treatment.

Causes and incidence

Infection from the spirochete *Treponema pallidum* causes syphilis. Transmission occurs primarily through sexual contact during the primary, secondary, and early latent stages of infection. Prenatal transmission from an infected mother to her fetus is also possible. (See *Prenatal syphilis*.)

Incidence is highest in people ages 20 to 29. In 2005, there were 3 new cases per every 100,000 people.

Complications

- Aortic regurgitation
- Aneurysm
- Central nervous system damage
- Meningitis

Signs and symptoms

Primary syphilis develops after an incubation period that generally lasts about 3 weeks. Initially, one or more chancres (small, fluid-filled lesions) erupt on the genitalia; others may erupt on the anus, fingers, lips, tongue, nipples, tonsils, or eyelids. These chancres, which are usually painless, start as papules and then erode; they have indurated, raised edges and clear bases. Chancres typically disappear after 3 to 6 weeks, even when untreated. They're usually associated with regional lymphadenopathy (unilateral or bilateral). In females, chancres are commonly overlooked because they usually develop on internal structures—the cervix or the vaginal wall.

The development of symmetrical mucocutaneous lesions and general lymphadenopathy signals the onset of *secondary syphilis,* which may develop within a few days or up to 8 weeks after onset of initial chancres. The rash of secondary syphilis can be macular, papular,

pustular, or nodular. Lesions are of uniform size, well defined, and generalized. Macules typically erupt between rolls of fat on the trunk and, proximally, on the arms, palms, soles, face, and scalp. In warm, moist areas (perineum, scrotum, vulva, and between rolls of fat), the lesions enlarge and erode, producing highly contagious, pink or grayish white lesions (condylomata lata).

Mild constitutional symptoms of syphilis appear in the second stage and may include headache, malaise, anorexia, weight loss, nausea, vomiting, sore throat and, possibly, slight fever. Alopecia may occur, with or without treatment, and is usually temporary. Nails become brittle and pitted.

Latent syphilis is characterized by an absence of clinical symptoms but a reactive serologic test for syphilis. Because infectious mucocutaneous lesions may reappear when infection is of less than 4 years' duration, early latent syphilis is considered contagious. About two-thirds of patients remain asymptomatic in the late latent stage; the rest develop characteristic late-stage symptoms.

Late syphilis is the final, destructive but noninfectious stage of the disease. It has three subtypes, any or all of which may affect the patient: late benign syphilis, cardiovascular syphilis, and neurosyphilis. The lesions of *late benign syphilis* develop on the skin, bones, mucous membranes, upper respiratory tract, liver, or stomach between 1 and 10 years after infection. The typical lesion is a gumma—a chronic, superficial nodule or deep, granulomatous lesion that's solitary, asymmetrical, painless, and indurated. Gummas can be found on any bone—particularly the long bones of the legs—and in any organ. If late syphilis involves the liver, it can cause epigastric pain, tenderness, enlarged spleen, and anemia; if it involves the upper respiratory tract, it can cause perforation of the nasal septum or the palate. In severe cases, late benign syphilis results in destruction of

bones or organs, which eventually causes death.

PRENATAL SYPHILIS

A woman can transmit syphilis transplacentally to her unborn child throughout pregnancy. This type of syphilis

is often called congenital, but prenatal is a more accurate term. About 50% of infected fetuses die before or shortly after birth. The prognosis is better for infants who develop overt infection after age 2.

Signs and symptoms

The neonate with prenatal syphilis may appear healthy at birth, but usually develops characteristic lesions—vesicular, bullous eruptions, often on the palms and soles—3 weeks later. Shortly afterward, a maculopapular rash similar to that in secondary syphilis may erupt on the face, mouth, genitalia, palms, or soles. Condylomata lata typically occur around the anus. Lesions may erupt on the mucous membranes of the mouth, pharynx, and nose. When the infant's larynx is affected, his cry becomes weak and forced. If nasal mucous membranes are involved, he may also develop nasal discharge, which can be slight and mucopurulent or copious with blood-tinged pus. Visceral and bone lesions, liver or spleen enlargement with ascites, and nephrotic syndrome may also occur.

Late prenatal syphilis becomes apparent after age 2; it may be identifiable only through blood studies or may cause unmistakable syphilitic changes: screwdriver-shaped central incisors, deformed molars or cusps, thick clavicles, saber shins, bowed tibias, nasal septum perforation, eighth nerve deafness, and neurosyphilis.

Diagnosis and treatment

In the neonate with prenatal syphilis, the Venereal Disease Research Laboratory titer, if reactive at birth, stays the same or rises, indicating active disease. The infant's titer drops in 3 months if the mother has received effective prenatal treatment. Absolute diagnosis

necessitates dark-field examination of umbilical vein blood or lesion drainage.

An infant with abnormal cerebrospinal fluid (CSF) may be treated with aqueous crystalline penicillin G. An infant with normal CSF may be treated with a single injection of penicillin G.

When caring for a child with prenatal syphilis, record the extent of the rash and watch for signs of systemic involvement, especially laryngeal swelling, jaundice, and decreasing urine output.

Cardiovascular syphilis develops about 10 years after the initial infection in about 10% of patients with late, untreated syphilis. It causes fibrosis of elastic tissue of the aorta and leads to aortitis, usually in the ascending and transverse sections of the aortic arch. Cardiovascular syphilis may be asymptomatic or may cause aortic insufficiency or aneurysm.

Symptoms of *neurosyphilis* develop in about 8% of patients with late, untreated syphilis and appear from 5 to 35 years after infection. These clinical effects consist of meningitis and widespread central nervous system damage that may include general paresis, personality changes, and arm and leg weakness.

Diagnosis

CONFIRMING DIAGNOSIS

Identifying T. pallidum *from a lesion on dark-field examination confirms the diagnosis of syphilis. This method is most effective when moist lesions are present, as in primary, secondary, and prenatal syphilis. (See* Treponema pallidum, *page 1230.)*

The fluorescent treponemal antibodyabsorption test identifies antigens of *T. pallidum* in tissue, ocular fluid, cerebrospinal fluid (CSF), tracheobronchial secretions, and exudates from lesions. This is the most sensitive test available for detecting syphilis

in all stages. Once reactive, it remains so permanently.

TREPONEMA PALLIDUM

In syphilis, a dark-field examination that shows spiral-shaped bacterial organisms—*T. pallidum*—confirms the diagnosis.

Other appropriate procedures include the following:

- Venereal Disease Research Laboratory (VDRL) slide test and rapid plasma reagin test (RPR) detect nonspecific antibodies. Both tests, if positive, become reactive within 1 to 2 weeks after the primary lesion appears or 4 to 5 weeks after the infection begins.
- CSF examination identifies neurosyphilis when the total protein level is above 40 mg/dl, the VDRL slide test is reactive, and the cell count exceeds five mononuclear cells/µl.

Treatment

Treatment of choice is administration of penicillin I.M. or I.V. depending on the infection's stage. After therapy, follow-up RPR tests are usually done to check for adequacy of treatment. The nonpregnant patient who

is allergic to penicillin may be treated with tetracycline or doxycycline. Nonpenicillin therapy for latent or late syphilis should be used only after neurosyphilis has been excluded. Tetracycline is contraindicated in the pregnant woman because it causes discoloration of the infant's teeth. If a pregnant woman with syphilis is allergic to penicillin, desensitization is recommended to permit the use of penicillin.

Special considerations

- Stress the importance of completing the full course of antibiotic therapy even after symptoms subside.
- Check for a history of drug sensitivity before administering the first dose.
- In secondary syphilis, keep lesions clean and dry. If they're draining, dispose of contaminated materials properly.
- In late syphilis, provide symptomatic care during prolonged treatment.
- In cardiovascular syphilis, check for signs of decreased cardiac output (decreased urine output, hypoxia, and decreased sensorium) and pulmonary congestion.
- In neurosyphilis, regularly check level of consciousness and monitor vital signs. Watch for signs of ataxia.
- Urge the patient to seek testing after treatment to determine the treatment's effectiveness. A patient treated for latent or late syphilis should be encouraged to continue follow-up care after treatment to determine its effectiveness.
- Be sure to report all cases of syphilis to local public health authorities. Urge the patient to inform sex partners of his infection so that they can also receive treatment.
- Refer the patient and his sex partners for human immunodeficiency virus testing as appropriate.

PREVENTION

- *Advise the patient to practice safe sex and consistently use condoms.*

- *Screen women who are pregnant for syphilis to lessen the risk of infection for an unborn baby.*

Trichomoniasis

A protozoal infection of the lower genitourinary (GU) tract, trichomoniasis affects about 15% of sexually active females and 10% of sexually active males. This infection,

which occurs worldwide, may be acute or chronic in females. The risk of recurrence is minimized when sex partners are treated concurrently.

Causes and incidence

Trichomonas vaginalis—a tetraflagellated, motile protozoan—causes trichomoniasis in females by infecting the vagina, the urethra and, possibly, the endocervix, bladder, Bartholin's glands, or Skene's glands; in males, it infects the lower urethra and, possibly, the prostate gland, seminal vesicles, or epididymis.

T. vaginalis grows best when the vaginal mucosa is more alkaline than normal (pH about 5.5 to 5.8). Therefore, factors that raise the vaginal pH—use of hormonal contraceptives, pregnancy, bacterial overgrowth, exudative cervical or vaginal lesions, or frequent douching, which disturbs lactobacilli that normally live in the vagina and maintain acidity—may predispose a woman to trichomoniasis.

Trichomoniasis is usually transmitted by intercourse; less commonly, by contaminated douche equipment or moist washcloths. In the United States, incidence is highest in women ages 16 to 35.

Complications

- Pelvic inflammatory disease
- Vaginal erosion

Signs and symptoms

About 70% of females—including those with chronic infections—and most males with trichomoniasis are asymptomatic. In females, acute infection may produce variable signs, such as a gray or greenish yellow and possibly profuse and frothy, malodorous vaginal discharge. Its other effects include severe itching, redness, swelling, tenderness, dyspareunia, dysuria, urinary frequency and, occasionally, postcoital spotting, menorrhagia, or dysmenorrhea.

Such symptoms may persist for 1 week to several months and may be more pronounced just after menstruation or during pregnancy. If trichomoniasis is untreated, symptoms may subside, although *T. vaginalis* infection persists, possibly associated with an abnormal cytologic smear of the cervix.

In males, trichomoniasis may produce mild to severe transient urethritis, possibly with dysuria and frequency.

Diagnosis

CONFIRMING DIAGNOSIS

Direct microscopic examination of vaginal or seminal discharge is decisive when it reveals T. vaginalis *(a motile, pearshaped organism) on wet prep. A Papanicolaou test may also detect the organism. Examination of clear urine specimens may also reveal* T. vaginalis.

Physical examination of symptomatic females shows vaginal erythema; edema; frank excoriation; a frothy, malodorous, greenish-yellow vaginal discharge and, rarely, a thin, gray pseudomembrane over the vagina. Cervical examination demonstrates punctate cervical hemorrhages, giving the cervix a strawberry appearance that's almost pathognomonic for this disorder.

Treatment

The treatment of choice for trichomoniasis is metronidazole given to both sex partners. Oral metronidazole hasn't been proven safe during the first trimester of pregnancy but can be considered for use if

symptoms are severe. In general, treatment during the first trimester should be avoided if possible. Effective alternatives aren't available for patients who are allergic to metronidazole. Sitz baths may be used to help relieve symptoms.

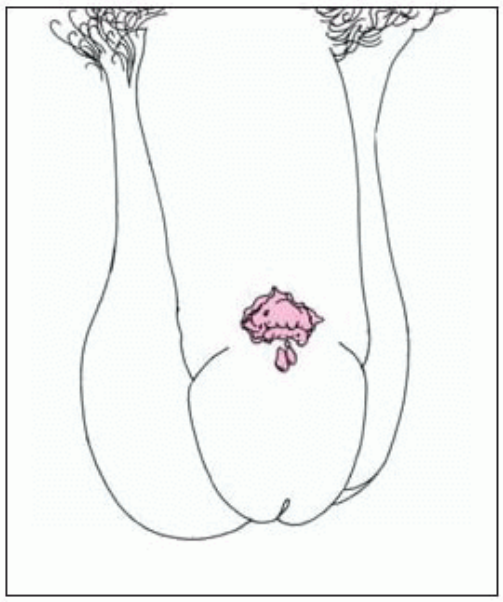
Special considerations

- Instruct the patient to refrain from douching before being examined for trichomoniasis.
- Warn the patient to abstain from alcoholic beverages while taking metronidazole because alcohol consumption may provoke a disulfiram-type reaction (confusion, headache, cramps, vomiting, and seizures). Also, tell her this drug may turn urine dark brown.
- Caution the patient to avoid over-the-counter douches and vaginal sprays because chronic use can alter vaginal pH.
- Advise the patient to scrub the bathtub with a disinfecting cleaner before and after sitz baths.

- Tell the patient she can reduce the risk of GU bacterial growth by wearing loose-fitting, cotton underpants, which allows ventilation; bacteria flourish in a warm, dark, moist environment.

CHANCROIDAL LESION

Chancroid produces a soft, painful chancre, similar to that of syphilis. Without treatment, it may progress to inguinal adenitis and formation of buboes (enlarged, inflamed lymph nodes).



PREVENTION

Advise abstinence from intercourse until treatment is completed. Refer partners for treatment. Tell a woman to avoid using tampons.

Chancroid

Chancroid, also known as *soft chancre,* is a sexually transmitted disease (STD) characterized by painful genital ulcers and inguinal adenitis. Chancroidal lesions may heal spontaneously and usually respond well to treatment in the absence of secondary infections. A high rate of human immunodeficiency virus (HIV) infection has been reported among patients with chancroid.

Causes and incidence

Chancroid results from *Haemophilus ducreyi,* a gram-negative Streptobacillus, and is transmitted through sexual contact. Poor hygiene may predispose males—especially those who are uncircumcised—to this disease.

This infection occurs worldwide but is particularly common in tropical countries; it affects more males than females.

Complications

- Phimosis
- Secondary infections
- Urethral fistulas

Signs and symptoms

After a 3- to 5-day incubation period, a small papule appears at the entry site, usually the groin or inner thigh; in the male, it may appear on the penis; in the female, on the vulva, vagina, or cervix. (See *Chancroidal lesion.*) Occasionally, this papule may erupt on the tongue, lip, breast, or navel. The papule rapidly ulcerates, becoming painful, soft, and malodorous; it bleeds easily and produces pus. It's gray and shallow, with irregular edges, and measures up to 2.5 cm in diameter. Within 2 to 3 weeks, inguinal adenitis develops, creating suppurated, inflamed nodes that may rupture into large ulcers or buboes. Headache and malaise occur in 50% of patients. During the healing stage, phimosis may develop.

Diagnosis

Gram stain smears of ulcer exudate or bubo aspirate are 50% reliable; blood agar cultures are 75% reliable. Biopsy confirms the diagnosis but is reserved for resistant cases or cases in which cancer is suspected. Dark-field examination and serologic testing rule out other STDs that cause similar ulcers. Testing for HIV infection should be done at the time of diagnosis.

Treatment

The treatment of choice is azithromycin, erythromycin, ceftriaxone, or ciprofloxacin. The safety of azithromycin for pregnant or lactating women hasn't been established. Aspiration of fluid-filled nodes may be indicated as well.

Special considerations

- Make sure the patient isn't allergic to any drug before giving the first dose.
- Instruct the patient not to apply lotions, creams, or oils on or near the genitalia or on other lesion sites.
- Tell the patient to abstain from sexual contact until healing is complete (usually about 2 weeks after treatment begins) and to wash the genitalia daily with soap and water. Instruct uncircumcised males to retract the foreskin for thorough cleaning.

PREVENTION

Advise the patient to avoid sexual contact with infected people, to use condoms during sexual activity, and to wash the genitalia with soap and water after sexual activity. Tell the patient that abstinence is the only sure way to prevent chancroid.

Nonspecific genitourinary infections

Nonspecific genitourinary (GU) infections, including nongonococcal urethritis (NGU) in males and mild vaginitis or cervicitis in females, are a group of infections with similar manifestations that aren't linked to a single organism. These sexually transmitted diseases (STDs) have become more prevalent since the mid-1960s. They're more widespread than gonorrhea and may be the most common STDs in the United States. The prognosis is good if sex partners are treated simultaneously.

Causes and incidence

Nonspecific GU infections are spread primarily through sexual intercourse. In males, NGU commonly results from infection with *Chlamydia trachomatis* or *Ureaplasma urealyticum*. Less commonly, infection may be related to pre-existing strictures, neoplasms, and chemical or traumatic inflammation. Some cases remain unexplained.

Although less is known about nonspecific GU infections in females, chlamydial organisms may also cause these infections. A thin vaginal

epithelium may predispose prepubertal and postmenopausal females to nonspecific vaginitis.

Signs and symptoms

NGU occurs 1 week to 1 month after coitus, with scant or moderate mucopurulent urethral discharge, variable dysuria, and occasional hematuria. If untreated, NGU may lead to acute epididymitis. Subclinical urethritis may be found on physical examination, especially if the sex partner has a positive diagnosis.

Females with nonspecific GU infections may experience persistent vaginal discharge, acute or recurrent cystitis for which no underlying cause can be found, or cervicitis with inflammatory erosion.

Both males and females with nonspecific GU infections may be asymptomatic but show signs of urethral, vaginal, or cervical infection on physical examination.

Diagnosis

In males, microscopic examination of smears of prostatic or urethral secretions shows excess polymorphonuclear leukocytes but few, if any, specific organisms.

In females, cervical or urethral smears also reveal excess leukocytes and no specific organisms. “Clue cells” (normal epithelial cells covered with bacteria that appear stippled) are diagnostic.

Treatment

Therapy for both sexes consists of azithromycin or doxycycline. If the infection recurs or persists, metronidazole with erythromycin is recommended.

Special considerations

- Tell the female patient to clean the pubic area before applying vaginal medication and to avoid using tampons during treatment.
- Make sure the patient clearly understands and strictly follows the dosage schedule for prescribed medications.

PREVENTION

- *Advise the patient to abstain from sexual contact with infected partners. Tell the patient to use condoms during sexual activity, to practice good hygiene afterward, and to void before and after intercourse.*
- *Encourage the patient to maintain adequate fluid intake.*

- *Advise the female patient to avoid routinely using douches and feminine hygiene sprays, wearing tight-fitting pants or pantyhose, and inserting foreign objects into the vagina.*
- *Suggest that the female patient wear cotton underpants and remove them before going to bed.*

MALE REPRODUCTIVE DISORDERS

Hypogonadism

Hypogonadism is a condition resulting from decreased androgen production in males, which may impair spermatogenesis (causing infertility) and inhibit the development of normal secondary sex characteristics. (See *Production of sperm*.) The clinical effects of androgen deficiency depend on the patient's age at onset.

Causes and incidence

Primary hypogonadism results directly from interstitial (Leydig's cell) cellular or seminiferous tubular damage due to faulty development or mechanical damage. This causes increased secretion of gonadotropins by the pituitary in an attempt to increase the testicular functional state and is therefore termed *hypergonadotropic hypogonadism*. This form of hypogonadism includes Klinefelter syndrome, Reifenstein's syndrome,

Turner syndrome, Sertoli-cell-only syndrome, anorchism, orchitis, and sequelae of irradiation.

Secondary hypogonadism is due to faulty interaction within the hypothalamic-pituitary axis, resulting in failure to secrete normal levels of gonadotropins, and is therefore termed *hypogonadotropic hypogonadism*. This form of hypogonadism includes hypopituitarism, isolated follicle-stimulating hormone deficiency, isolated luteinizing hormone deficiency, Kallmann's syndrome, and Prader-Willi syndrome. Depending on the patient's age at onset, hypogonadism may cause eunuchism (complete gonadal failure) or eunuchoidism (partial failure).

Medications, such as exogenous testosterone or anabolic steroids, can also cause of hypogonadism, resulting in infertility.

Hypogonadism is rare, and it has no racial predilection.

Complications

- Impotence
- Infertility
- Loss of sex drive
- Osteoporosis

Signs and symptoms

Although symptoms vary, depending on the specific cause of hypogonadism, some characteristic findings may include delayed closure of epiphyses and immature bone age; delayed puberty; infantile penis and small, soft testes; below-average muscle development and strength; fine, sparse facial hair; scant or absent axillary, pubic, and body hair; and a high-pitched, effeminate voice. In an adult, hypogonadism diminishes sex drive and potency and causes regression of secondary sex characteristics.

Diagnosis

Accurate diagnosis necessitates a detailed patient history, physical examination, and hormonal studies. Serum and urinary gonadotropin

levels increase in primary (hypergonadotropic) hypogonadism but decrease in secondary (hypogonadotropic) hypogonadism. Other relevant hormonal studies include assessment of neuroendocrine functions, such as thyrotropin, corticotropin, growth hormone, and vasopressin levels. Chromosomal analysis may determine the specific causative syndrome. Testicular biopsy and semen analysis determine sperm production, identify impaired spermatogenesis, and assess low levels of testosterone.

Treatment

Treatment depends on the underlying cause and may consist of hormonal replacement, especially with testosterone, methyltestosterone, estrogen, progesterone, or human chorionic gonadotropin (hCG) for primary hypogonadism, and with hCG for secondary hypogonadism. Fertility can't be restored after permanent testicular damage. However, eunuchism that results

from hypothalamic-pituitary lesions can be corrected when administration of gonadotropins stimulates normal testicular function.

PRODUCTION OF SPERM

Spermatogenesis, the production of male gametes within the seminiferous tubules of the testes, is basically a five-step process:

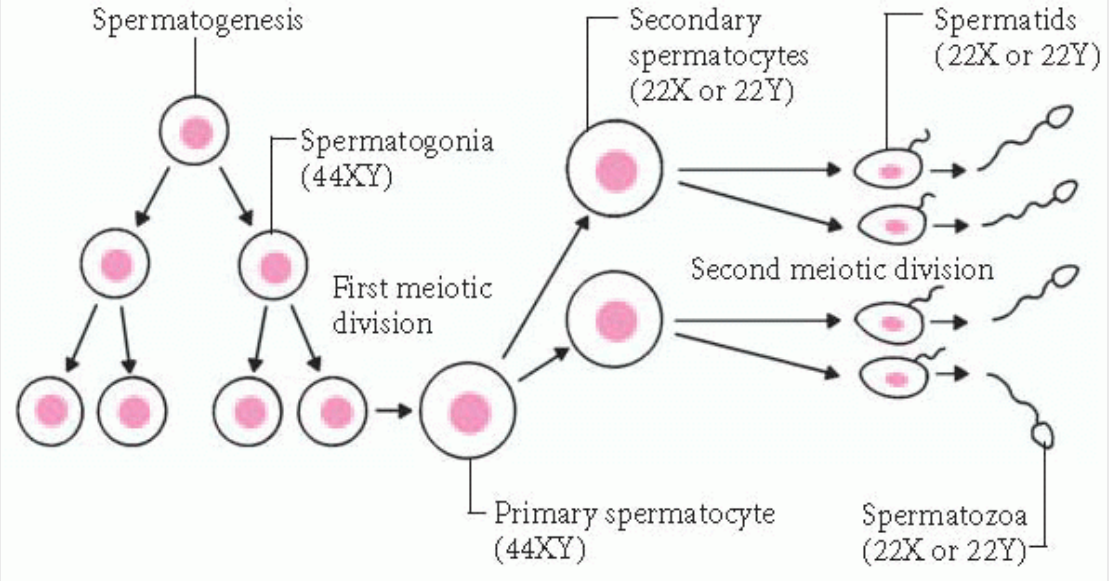
1. Diploid spermatogonia, the cells forming the tubule's outer layer, divide mitotically to generate new cells used in spermatozoa production.

2. Some of the spermatogonia move toward the lumen of the tubule and enlarge to primary spermatocytes.

3. Each primary spermatocyte divides meiotically, forming two secondary spermatocytes, one retaining the X chromosome and the other the Y chromosome.

4. Each secondary spermatocyte also divides meiotically, becoming spermatids.

5. After a series of structural changes, the spermatids develop into mature spermatozoa.



Special considerations

Because the patient with hypogonadism tends to have multiple associated physical problems, the care plan should be tailored to meet his specific needs.

- When caring for an adolescent boy with hypogonadism, make every possible effort to promote his self-confidence. If he feels sensitive about his underdeveloped body, provide access to a private bathroom. Explain hypogonadism to his parents. Encourage them to express their concerns about their son's delayed development. Reassure them and the patient that effective treatment is available.
- Make sure the parents and the patient understand hormonal replacement therapy fully, including expected adverse effects, such as acne and water retention.
- Encourage counseling as appropriate.

Undescended testes

Undescended testes is a congenital disorder in which one or both testes fail to descend into the scrotum, remaining in the abdomen or inguinal

canal or at the external ring. Although this condition, also known as *cryptorchidism,* may be bilateral, it more commonly affects the right testis. True undescended testes remain along the path of normal descent, whereas ectopic testes deviate from that path. If bilateral cryptorchidism persists untreated into adolescence,

it may result in sterility, make the testes more vulnerable to trauma, and significantly increase the risk of testicular cancer, presumably due to the higher temperature of the abdominal cavity.

Causes and incidence

The mechanism whereby the testes descend into the scrotum is still unexplained. Some evidence is available to implicate hormonal factors—most likely androgenic hormones from the placenta, maternal or fetal adrenals, or the immature fetal testis and, possibly, maternal progesterone or gonadotropic hormones from the maternal pituitary.

Researchers have linked undescended testes to the development of the gubernaculum, a fibromuscular band that connects the testes to the scrotal floor. In the normal male fetus, testosterone stimulates the formation of the gubernaculum. This band probably helps pull the testes into the scrotum by shortening as the fetus grows. Thus, cryptorchidism may result from inadequate testosterone levels or a defect in the testes or the gubernaculum.

Because the testes normally descend into the scrotum during the eighth month of gestation, cryptorchidism most commonly affects premature neonates. (It occurs in 30% of premature male neonates but in only 3% to 4% of those born at term.) In about 80% of affected infants, the testes descend spontaneously during the first year; in the rest, the testes may descend later.

Complications

- Infertility
- Testicular cancer

Signs and symptoms

In the young boy with unilateral cryptorchidism, the testis on the affected side isn't palpable in the scrotum, and the scrotum may appear underdeveloped. On the unaffected side, the scrotum occasionally appears enlarged as a result of compensatory hypertrophy. After puberty, uncorrected bilateral cryptorchidism prevents spermatogenesis and results in infertility, although testosterone levels remain normal.

Diagnosis

CONFIRMING DIAGNOSIS

Physical examination confirms cryptorchidism after the following laboratory tests determine sex:

- *Buccal smear determines genetic sex by showing a male sex chromatin pattern.*
- *Serum gonadotropin confirms the presence of testes by assessing the level of circulating hormone.*

Treatment

If the testes don't descend spontaneously by age 1 year, surgical correction may be indicated. Orchiopexy secures the testes in the scrotum and is commonly performed before the boy reaches age 4 (optimum age is 1 to 2). Orchiopexy prevents sterility and excessive trauma from abnormal positioning. It also prevents harmful psychological effects. Human chorionic gonadotropin (hCG) or testosterone may be given to stimulate descent. However, hormonal therapy with hCG is ineffective if the testes are located in the abdomen.

Special considerations

- Encourage parents of the child with undescended testes to express their concern about his condition. Provide information about causes, available treatments, and the ultimate effect on reproduction. Emphasize that, especially in premature neonates, the testes may descend spontaneously.

- If orchiopexy is necessary, explain the surgery to the child, using terms he understands. Tell him that a rubber band may be taped to his thigh for about 1 week after surgery to keep the testis in place. Explain that his scrotum may swell but shouldn't be painful.

After orchiopexy:

- Monitor vital signs and intake and output. Check dressings. Encourage coughing and deep breathing. Watch for urine retention.
- Keep the operative site clean. Tell the child to wipe from front to back after defecating. If a rubber band has been applied to keep the testis in place, maintain tension, but make sure it isn't too tight.
- Encourage parents to participate in postoperative care, such as bathing or feeding

the child. Also urge the child to do as much for himself as possible.

Testicular torsion

Testicular torsion is an abnormal twisting of the spermatic cord due to rotation of a testis or the mesorchium (a fold in the area between the testis and epididymis), which causes strangulation and, if untreated, eventual infarction of the testis. This condition is almost always (90%) unilateral in presentation, but the defect is bilateral, requiring both testicles to be surgically treated. Testicular torsion is most common between ages 12 and 18, but it may occur at any age. The prognosis is good with early detection and prompt treatment.

Causes and incidence

Normally, the tunica vaginalis envelops the testis and attaches to the epididymis and spermatic cord. In *intravaginal torsion* (the most common type of testicular torsion in adolescents), testicular twisting may result from an abnormality of the tunica, in which the testis is abnormally positioned, or from a narrowing of the mesentery support. In *extravaginal torsion* (most common in neonates), loose attachment of the tunica vaginalis to the scrotal lining causes spermatic cord rotation above the testis. Typically, there's no history of trauma, and the pain

occurs suddenly. A sudden forceful contraction of the cremaster muscle may precipitate this condition. (See *Extravaginal torsion*.)

Complications

- Complete testicular infarction
- Testicular atrophy

Signs and symptoms

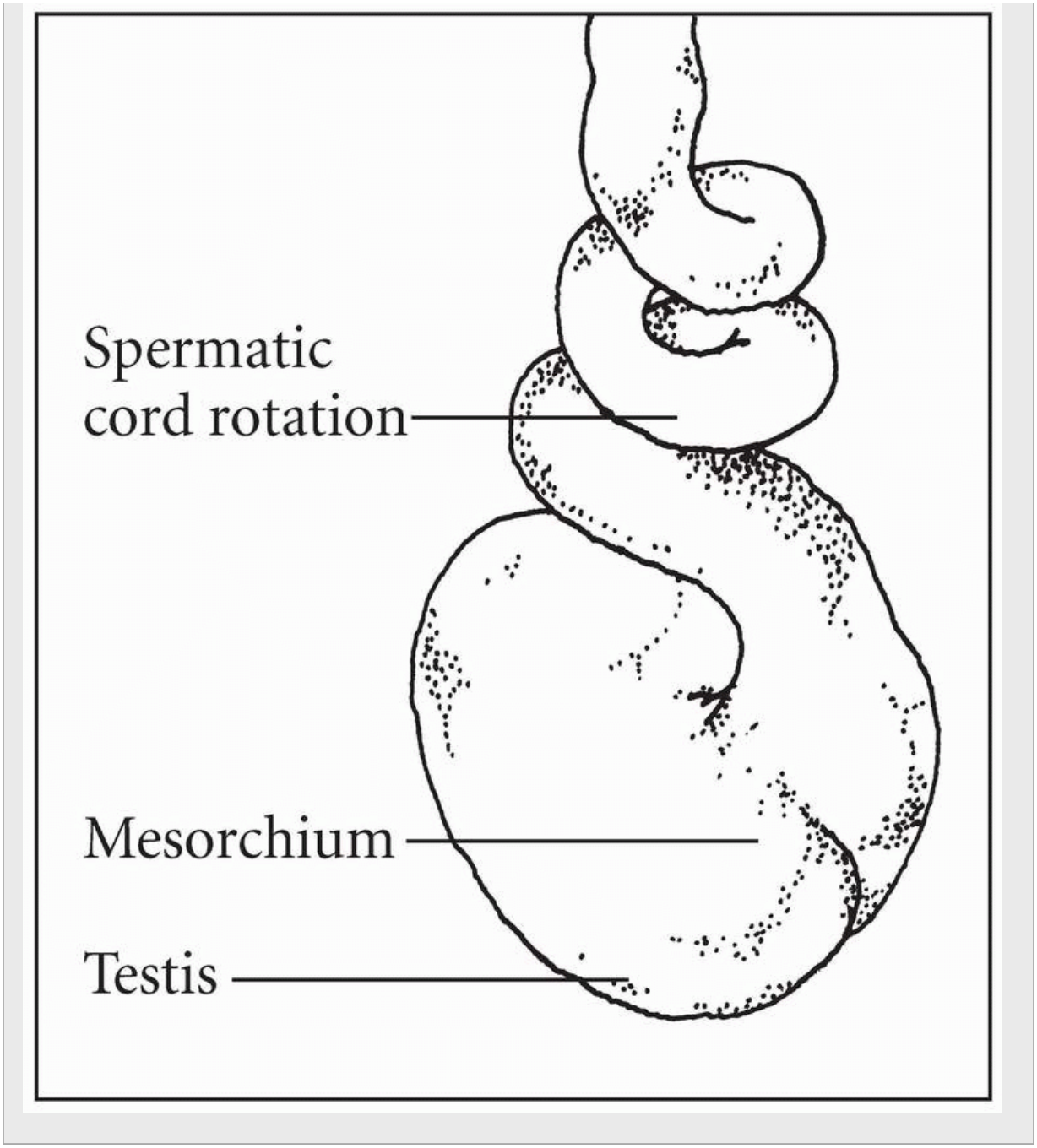
Torsion produces excruciating pain in the affected testis or iliac fossa. Nausea, vomiting, and light-headedness may also occur.

Diagnosis

Physical examination reveals tense, tender swelling in the scrotum or inguinal canal and hyperemia of the overlying skin. Doppler ultrasonography helps distinguish testicular torsion from strangulated hernia, undescended testes, or epididymitis.

EXTRAVAGINAL TORSION

In extravaginal torsion, rotation of the spermatic cord above the testis causes strangulation and, eventually, infarction of the testis.



Treatment

Treatment consists of untwisting the testes and immediate surgical repair by orchiopexy (fixation of a viable testis to the scrotum) or orchiectomy (excision of a nonviable testis). Both testes are usually anchored to the scrotum as a preventive measure. As with ovarian

torsion in the female, preservation of the organ is the preferred option. If surgery is performed within 6 hours, most testicles can be saved.

Special considerations

- Promote the patient's comfort before and after surgery.
- After surgery, administer pain medication as ordered. Monitor voiding, and apply an ice bag with a cover to reduce edema. Protect the wound from contamination. Otherwise, allow the patient to perform as many normal daily activities as possible.

Male infertility

Male infertility may be suspected whenever a couple fails to achieve pregnancy after about 1 year of regular, unprotected intercourse.

Causes and incidence

Some factors associated with male infertility include:

- varicocele, a mass of dilated and tortuous varicose veins in the spermatic cord
- semen disorders, such as volume or motility disturbances and inadequate sperm density
- proliferation of abnormal or immature sperm, with variations in the head's size and shape
- systemic disease, such as diabetes mellitus, neoplasms, hepatic and renal diseases, and viral disturbances, especially mumpsrelated orchitis
- genital infections, such as gonorrhea, tuberculosis, and herpes
- disorders of the testes, such as cryptorchidism, Sertoli-cell-only syndrome, and ductal obstruction (caused by absence or ligation of vas deferens or infection)
- genetic defects, such as Klinefelter's and Reifenstein's syndromes

- immunologic disorders, such as autoimmune infertility and allergic orchitis
- endocrine imbalances that disrupt pituitary gonadotropins, inhibiting spermatogenesis, testosterone production, or both (as in Kallmann's syndrome, panhypopituitarism, hypothyroidism, and congenital adrenal hyperplasia)
- chemicals and drugs that can inhibit gonadotropins or interfere with spermatogenesis, such as arsenic, methotrexate, medroxyprogesterone, nitrofurantoin, monoamine oxidase inhibitors, and some antihypertensives
- sexual problems, such as erectile dysfunction, ejaculatory incompetence, and low libido.

Age, occupation, and traumatic injury to the testes can also contribute to male infertility. About 30% to 40% of infertility problems in the United States are attributed to the male.

Complication

- Emotional or psychological distress

Signs and symptoms

The obvious indication of male infertility is failure to impregnate a fertile woman. Clinical features may include atrophied testes; empty scrotum; scrotal edema; varicocele or anteversion of the epididymis; inflamed seminal vesicles; beading or abnormal nodes on the spermatic cord and vas; penile nodes, warts, plaques, or hypospadias; prostatitis, which may be acute or chronic; and prostatic enlargement, nodules, swelling, or tenderness. In addition, male infertility commonly induces troublesome negative emotions in a couple—anger, hurt, disgust, guilt, and loss of self-esteem.

Diagnosis

A detailed patient history may reveal abnormal sexual development, delayed puberty, infertility in previous relationships, and a medical history of prolonged fever, mumps, impaired nutritional status, previous

surgery, or trauma to genitalia. After a thorough patient history and physical examination, the most conclusive test for male infertility is semen analysis. The specimen is collected after 2 to 3 days of complete abstinence to determine volume and viscosity as well as sperm count, motility, swimming speed, and shape.

Other laboratory tests include gonadotropin assay to determine the integrity of the pituitary gonadal axis, serum testosterone levels to determine end organ response to luteinizing hormone (LH), urine 17-ketosteroid levels to measure testicular function, and testicular biopsy to help clarify unexplained oligospermia and azoospermia. Vasography and seminal vesiculography may be necessary.

Treatment

When anatomic dysfunction or infection causes infertility, treatment consists of correcting the underlying problem. A varicocele requires surgical repair or removal. For patients with sexual dysfunction, treatment includes education, counseling or therapy (on sexual techniques, coital frequency, and reproductive physiology), and proper nutrition with vitamin supplements. Decreased follicle-stimulating hormone levels may respond to vitamin B therapy; decreased LH levels, to human chorionic gonadotropin (hCG) therapy. Normal or elevated LH level requires low dosages of testosterone. Decreased testosterone levels, decreased semen motility, and volume disturbances may respond to hCG.

A patient with oligospermia who has a normal history and physical examination, normal hormonal assays, and no signs of systemic disease requires emotional support and counseling, adequate nutrition, multivitamins, and selective therapeutic agents, such as clomiphene, hCG, and low dosages of testosterone. Obvious alternatives to such treatment are adoption and artificial insemination.

Special considerations

- Educate the couple, as necessary, about reproductive and sexual function and about factors that may interfere with fertility such as the

use of lubricants and douches.

- Urge men with oligospermia to avoid habits that may interfere with normal spermatogenesis by elevating scrotal temperature, such as wearing tight underwear and athletic supporters, taking hot tub baths, or habitually riding a bicycle. Explain that cool scrotal temperature is essential for normal spermatogenesis.
- When possible, advise the infertile couple to join group programs to share their feelings and concerns with other couples who have the same problem.

PREVENTION

- *Encourage the aging patient to have regular physical examinations.*
- *Advise the patient to protect his gonads during athletic activity.*
- *Advise the patient to receive early treatment for sexually transmitted diseases and surgical correction for anatomic defects.*

Precocious puberty in males

In precocious puberty, boys begin to mature sexually before age 10. This disorder can occur as *true precocious puberty,* which is most common, with early maturation of the hypothalamic-pituitary-gonadal axis, development of secondary sex characteristics, gonadal development, and spermatogenesis, or as *pseudoprecocious puberty,* with development of secondary sex characteristics without gonadal development. Boys with true precocious puberty reportedly have fathered children as early as age 7.

In most boys with precocious puberty, sexual characteristics develop in essentially normal sequence; these children function normally when they reach adulthood.

Causes and incidence

True precocious puberty may be idiopathic (constitutional) or cerebral (neurogenic). In some patients, idiopathic precocity may be genetically transmitted as a dominant trait. Cerebral precocity results from pituitary or hypothalamic intracranial lesions that cause excessive secretion of gonadotropin.

Pseudoprecocious puberty may result from testicular tumors (hyperplasia, adenoma, or carcinoma) or from congenital adrenogenital syndrome. Testicular tumors produce excessive testosterone levels; adrenogenital syndrome produces high levels of adrenocortical steroids.

Complications

- Emotional disturbances
- Increased intracranial pressure
- Pituitary tumors
- Stunted adult stature
- Vision disturbances

Signs and symptoms

All boys with precocious puberty experience early bone development, causing an initial growth spurt, early muscle development, and premature closure of the epiphyses, which results in stunted adult stature. Other features are adult hair pattern, penile growth, and bilateral enlarged testes. Symptoms of precocity due to cerebral lesions include nausea, vomiting, headache, vision disturbances, and internal hydrocephalus.

In pseudoprecocity caused by testicular tumors, adult hair patterns and acne develop. A discrepancy in testis size also occurs;

the enlarged testis may be hard or may contain a palpable, isolated nodule. Adrenogenital syndrome produces adult skin tone, excessive hair (including beard), and deepened voice. A boy with this syndrome appears stocky and muscular; his penis, scrotal sac, and prostate are enlarged (but not the testes).

Diagnosis

Assessing the cause of precocious puberty requires a complete physical examination. A detailed patient history can help evaluate the patient's recent growth pattern, behavior changes, a family history of precocious puberty, or ingestion of hormones.

In true precocity, laboratory results include the following:

- Serum levels of luteinizing and follicle-stimulating hormones and corticotropin are elevated.
- Plasma tests for testosterone demonstrate elevated levels (equal to those of an adult male).
- Evaluation of ejaculate reveals the presence of live spermatozoa.
- Brain scan, skull X-rays, and EEG can detect possible central nervous system tumors. Abdominal scans can detect testicular tumors.

A child with an initial diagnosis of idiopathic precocious puberty should be reassessed regularly for possible tumors.

In pseudoprecocity, chromosomal karyotype analysis demonstrates an abnormal pattern of autosomes and sex chromosomes. Elevated levels of 24-hour urinary 17-ketosteroids and other steroids also indicate pseudoprecocity.

Treatment

Boys with idiopathic precocious puberty generally require no medical treatment and suffer no physical complications in adulthood. Supportive psychological counseling is the most important therapy.

When precocious puberty is caused by tumors, the outlook is less encouraging. Brain tumors necessitate neurosurgery but may resist treatment and prove fatal. Testicular tumors may be treated by removing the affected testis (orchiectomy). Malignant tumors require chemotherapy and lymphatic radiation therapy. The prognosis is generally good, depending on tumor histology and degree of differentiation.

Adrenogenital syndrome that causes precocious puberty may respond to life-long therapy with maintenance doses of glucocorticoids (cortisol) to inhibit corticotropin production.

Special considerations

- Emphasize to parents that the child's social and emotional development should remain consistent with his chronological age, not with his physical development. Advise parents not to place unrealistic demands on him.
- Reassure the child that, although his body is changing more rapidly than those of other boys, eventually they will experience the same changes. Help him feel less self-conscious about his changing body. Suggest clothing that de-emphasizes sexual development.
- Provide sex education for the child with true precocity.
- If the child must take glucocorticoids for the rest of his life, explain the medication's adverse effects (cushingoid symptoms) to the family.

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