Significant proportion of the modern practice of adult gynecology and urology is devoted to the prevention, diagnosis, and treatment of sexually transmitted diseases (STDs), because of both the high prevalence of these diseases and their almost exclusive involvement of the reproductive systems in both sexes.

A sexually transmitted disease is any infectious disease that is transmitted from one person to another through sexual contact, taking that phrase in its broadest sense. Venereal disease (VD), a synonymous term, has now largely fallen out of use, as has the euphemism social disease. It is worth emphasizing that the only thing all STDs have in common is their mode of transmission. In other respects they vary widely among themselves. The common tendency to lump them all together yields a biologically invalid concept that invites confusion and misunderstanding.

Although any infection that is capable of being transmitted through sexual contact may be called an STD, that need not be its only mode of transmission. While it is true, for example, that AIDS and hepatitis B are often acquired through sexual contact, nonsexual transmission through oral-genital contact and anal intercourse, with resulting oropharyngeal, anorectal, or systemic infection. STDs affecting the skin (genital warts, genital herpes) or transmitted through the skin (AIDS, syphilis) can be acquired during intimate contact even though genital exposure is avoided or a condom is used.

All of the classically recognized STDs can be transmitted through vaginal intercourse. Most of them can also be transmitted through oral-genital contact and anal intercourse, with resulting oropharyngeal, anorectal, or systemic infection. STDs affecting the skin (genital warts, genital herpes) or transmitted through the skin (AIDS, syphilis) can be acquired during intimate contact even though genital exposure is avoided or a condom is used.

STDs are statistically more likely to occur in persons under 25 years of age, in members of ethnic minorities, in persons of low socioeconomic status, in persons with many sexual partners (especially prostitutes), and in sexually active gay men (but not in sexually active lesbians). The incidence of STDs is higher in urban areas.

The only absolute protection against acquiring an infection through sexual contact is lifelong celibacy or maintenance of a permanently and mutually monogamous sexual relationship. In this context, the term sexual promiscuity is rigorously defined as “having more than one sexual partner in a lifetime.” Some degree of protection against STDs is afforded by practicing “safe (or safer) sex”—which basically means using condoms and avoiding high-risk behaviors such as anal intercourse—and by limiting the number of sex partners.

The overall incidence of sexually transmitted infections has increased substantially during the past generation, and some diseases have shown a marked increase. Several factors have contributed to these changing statistics. The discovery in the 1940s that penicillin could cure syphilis and gonorrhea and the development during the 1950s of safe and effective oral contraceptives paved the way for the sexual revolution of the 1960s. Against a background of civil unrest, widespread drug abuse, and radical feminism, that revolution led to social acceptance of sexual promiscuity, popularization of oral and anal sex, and definition of overt homosexuality as normal behavior.

Other factors favoring sexual promiscuity have been the legalization of abortion on demand, the decline of the family as the basic unit of society, the widespread rejection of traditional faith-driven morality, and the increasing freedom and mobility of young people. Sex education in elementary and secondary schools, designed to reduce illegitimate pregnancies and STDs, has in the opinion of some observers had the opposite result.

The diagnosis and treatment of STDs are rendered more difficult by the reluctance of most people to discuss their sexual behavior with health professionals and by the refusal of many patients to believe that a sexual partner has become infected by some third person. Diagnosis often demands alertness and a high degree of suspicion on the part of the healthcare worker. History-taking must be searching but nonthreatening and nonjudgmental. Often the most suggestive point in the history is exposure to a new sexual partner within a month or so before the appearance of symptoms.

In treating any patient with an STD, the physician must reckon with two epidemiologic realities: the fact that at least one of the patient’s sexual partners (and possibly all of them) is also infected, and the statistical probability that a person with one STD has other STDs. Failure to treat sexual partners prophylactically will lead to eventual reinfection of most patients. Moreover, unless both partners in a relationship are treated at the same time, they may keep reinforcing each other, a phenomenon known as “ping-ponging.” Screening tests—that is, tests for certain STDs in persons who do not have symptoms of those STDs—are routinely performed on sexually active patients in a variety of healthcare settings.
Because a woman infected with gonorrhea may have only mild, nonspecific genital symptoms, or none at all, most cases of gonorrhea go untreated. As many as 20% of women with untreated gonorrhea will eventually develop acute salpingitis, also called pelvic inflammatory disease (PID), due to spread of infection to one or both uterine tubes.

Urethritis and Pelvic Inflammatory Disease: Gonorrhea and Chlamydia

Gonorrhea is an infection of genital mucous membranes or other tissues in either sex caused by Neisseria gonorrhoeae, a gram-negative diplococcus. This disease has been known for centuries and goes by the colloquial name of “clap.” Physicians often refer to the causative organism as the gonococcus, GC for short, and that abbreviation frequently stands for the disease itself in medical slang. After an incubation period (interval between acquisition of infection and appearance of symptoms) of just 3-5 days, gonorrhea attacks the mucosal lining of the urethra in men and the urethra and endocervical canal in women.

In men, gonococcal urethritis typically presents as a thick yellowish-green discharge with severe pain on urination. Complications of untreated gonorrhea in men include acute epididymitis (infection of the epididymis, part of the spermatic duct system adjacent to the testicle) and chronic scarring of the urethra resulting in stricture formation. Even asymptomatic inflammation of the male genitourinary tract associated with gonorrhea can increase susceptibility to HIV infection.

Because a woman infected with gonorrhea may have only mild, nonspecific genitourinary symptoms, or none at all, most cases of gonorrhea go untreated. As many as 20% of women with untreated gonorrhea will eventually develop acute salpingitis, also called pelvic inflammatory disease (PID), due to spread of infection to one or both uterine tubes. An acute attack of PID is more likely to occur during menstruation. The symptoms of pelvic pain and fever are nonspecific, but severe tenderness on manipulation of the cervix and on palpation of the uterine adnexa during pelvic examination are highly suggestive of the diagnosis.

PID can progress to tubo-ovarian abscesses (an abscess formed between a uterine tube and its adjacent ovary) or to perihalcyritis (Fitz-Hugh and Curtis syndrome, a local peritonitis in the region of the liver). A more common consequence of PID is scarring of the uterine tubes with resulting infertility (relative unlikelihood of conceiving) or sterility (impossibility of conceiving) and heightened risk of ectopic (tubal) pregnancy.

In both sexes gonorrhea occasionally develops into a disseminated form with focal or widespread skin lesions, arthritis, meningitis, or endocarditis. Oropharyngeal and rectal forms of infection result from oral and anal sex. Gonorrhea can cause severe eye infection, resulting in blindness, in an infant born to an infected mother.

The diagnosis of gonorrhea can be made by identification of gram-negative intracellular diplococci in a stained smear of urethral discharge or of material obtained from the pharynx, endocervix, or rectum; by culture of such material on Thayer-Martin agar, which is designed to favor the growth of gonococci; or by DNA probe, whose sensitivity is markedly enhanced by polymerase chain reaction (PCR) and ligand chain reaction (LCR) technology. The sensitivity of laboratory diagnosis, particularly in women, can be improved by doing rectal as well as cervical cultures, because even without a history of anal intercourse, the organism often migrates to the rectum from a genit al site of infection.

The technique of obtaining material for testing is crucial. Because organisms are intracellular, specimens must contain mucosal cells, not merely mucus or pus. The presence of blood, including menstrual discharge, impairs the sensitivity of molecular biology testing and requires culturing. Thayer-Martin plates must be refrigerated for storage but warmed to room temperature before inoculation, and incubated in an atmosphere of carbon dioxide. Screening for gonorrhea is routine in STD clinics, in pregnancy, and in high-risk female patients undergoing pelvic examination for any reason.

At one time gonorrhea could easily be eradicated with penicillin. During the 1970s, penicillin-resistant strains brought back from Asia by Vietnam War veterans became epidemiologically significant in the U.S. These organisms, called PPNG (penicillinase-producing Neisseria gonorrhoeae), are similar to resistant strains of staphylococcus in being able to break down some penicillins (and some cephalosporins) by producing the enzyme penicillinase (now more often called beta-lactamase because it inactivates the penicillin molecule by breaking it at its beta-lactam ring).

During the 1980s a second type of penicillin-resistant gonococcus was recognized in several geographic clusters in the U.S. Called chromosomally-mediated-resistant Neisseria gonorrhoeae (CMRNG), these organisms do not produce penicillinase (beta-lactamase) but are able to resist destruction by penicillin because of a genetic mutation. Because of widespread resistance to tetracyclines and fluoroquinolones, once standard alternative treatments for gonorrhea, these classes of antibiotic are also no longer approved for use.

The treatment currently recommended for uncomplicated gonococcal infection of the urethra, cervix, or rectum is ceftriaxone 250 mg in a single intramuscular dose (preferred) or cefixime 400 mg in a single oral dose. Oral cefixime is not approved for treatment of pharyngeal gonorrhea. All patients treated for gonorrhea are also treated prophylactically for chlamydia because of the high frequency with which these diseases occur together. All sexual contacts of patients with
Infection due to *chlamydia* is the most common of all bacterial STDs, with an estimated 1 million persons currently infected in the U.S. Although, strictly speaking, *chlamydia* is the name of the causative organism, in clinical parlance genital infections due to this organism are often called simply “chlamydia.” *Chlamydia trachomatis*, a bacterium that replicates inside human epithelial cells somewhat like a virus, was identified many years ago as the cause of trachoma, an important cause of blindness in tropical latitudes. Certain genetic subtypes of this organism are now recognized as the causes of various STD syndromes.

Two factors have favored the wide prevalence of chlamydial infections. First, the organism is highly contagious: at least 50% of sexual partners of persons with chlamydia are also infected. Second, 20% of men and 50% of women with the disease have no symptoms and do not know that they are infected (and infectious).

In men, chlamydia typically causes urethritis, with urinary burning and urethral discharge, after an incubation period of 1-4 weeks. In the days when most urethritis in men was due to gonorrhea, the term *nongonococcal urethritis* (NGU) or *nonspecific urethritis* (NSU) was applied to those cases in which smear and culture failed to confirm that diagnosis. Nowadays nongonococcal urethritis is more common than gonorrhea, and at least half of it is due to chlamydia. (Other organisms responsible for some cases of NGU are *Ureaplasma urealyticum* and *Trichomonas vaginalis*.)

As a complication of chlamydial urethritis, some patients (mostly males) develop a condition called *Reiter’s syndrome*, consisting of arthritis (particularly in the sacroiliac joints, knees, ankles, and feet) and conjunctivitis, occasionally accompanied by lesions of the skin and oral mucous membranes. This is an autoimmune response triggered by chlamydial infection. Like other autoimmune disorders such as rheumatoid spondylitis, it occurs almost exclusively in persons possessing the HLA-B27 antigen.

Some men who acquire chlamydial infection never develop symptoms. In any event, the symptoms are typically milder than those of gonorrhea, and they usually fade away within a few days, even without treatment. The patient can remain infectious for 1-3 years, however, and may eventually suffer some scarring of the urethra. Chlamydia is currently the principal cause of acute epididymitis and, in gay men, an important cause of proctitis (infection of the rectum).

In women the most frequent form of chlamydial infection is mucopurulent cervicitis, . . . *Chlamydia can also cause acute urethral syndrome in women, . . . As many as 20% of women with untreated chlamydial infection will eventually develop acute PID.* PID can cause scarring of the uterine tubes, tubo-ovarian abscess, or Fitz-Hugh and Curtis syndrome. At present half of all ectopic pregnancies in the U.S. occur in women who have had chlamydial PID. About 25% of women recovering from a first episode of PID are sterile, and the risk increases with each subsequent episode.

At least half of the infants born to mothers with active genital chlamydia become infected. As with gonorrhea, the most common form of congenital infection is conjunctivitis, which can result in visual impairment. Chlamydial pneumonia also occurs in newborns.

Before reliable culture methods were available for isolating chlamydia, the diagnosis was made by excluding gonorrhea in patients (chiefly men) with urethritis. Because chlamydia is an intracellular parasite, it can only be grown in tissue culture, that is, in a medium consisting of living cells. Early culture methods were expensive and yielded many false negatives. Currently most testing for chlamydial infection, including virtually all screening of asymptomatic persons, is performed by DNA probe on swab specimens collected from the urethra or cervix or on voided urine. PCR and other gene amplification technologies greatly augment the sensitivity and specificity of DNA probe testing.

The availability of urine testing, which requires no disrobing or genital exposure, has improved the accessibility and effectiveness of screening, especially of asymptomatic women. Some authorities currently recommend screening at intervals of 6 months for all sexually active persons who are not in mutually monogamous relationships. All authorities agree that pregnant women and persons at increased risk of contracting chlamydia infection because of lifestyle should be tested at regular intervals.

Chlamydia is treated with either azithromycin in a single dose of 1 g orally or with doxycycline 100 mg twice a day for 7-14 days. Oral erythromycin and amoxicillin are acceptable alternatives in pregnancy. All sexual partners must be treated at the same time as the patient, and concomitant treatment for gonorrhea, with or without previous screening, may also be recommended.

*Lymphogranuloma venereum* (LGV) is an uncommon STD syndrome caused by serovars (genetic variants that can be distinguished by serologic testing) of *C. trachomatis* other than those that cause urethritis and PID. LGV occurs in two or three stages. It usually begins as a solitary papule or ulcer at the site of infection. On the genitals this is usually painless,
Congenital syphilis is associated with a 40% perinatal mortality rate, and survivors display various deformities and developmental stigmata of the bones (saber shins, frontal bosses), teeth (mulberry molars, Hutchinson’s teeth), eyes (interstitial keratitis), and nervous system (neurosensory deafness, paresis).

but anorectal infection can lead to painful ulceration and bloody diarrhea. In the second stage, which follows the first within a few weeks, the patient experiences chills, fever, muscle and joint pains, and inguinal lymph node enlargement. The nodes may become extremely large, and suppurative formation often occurs within them. Spread of infection to deeper tissues may result in fistulas of the bladder or rectum. In about 5% of patients a third stage occurs, with progressive deterioration due to fistula formation, anal strictures (narrowing due to scarring), and edema caused by fibrosis of lymphatic channels. Diagnosis is confirmed by finding antibody to the infecting organism in the patient’s serum. As with other genital forms of chlamydial infection, treatment is with azithromycin or doxycycline.

Genital Ulcers: Syphilis and Herpes Simplex

Before the antibiotic era, syphilis was the most prevalent and most serious STD. Although at present the incidence of syphilis in the general population is low, it has been gradually rising in the U.S., U.K., Europe, and Australia since the year 2000. Most of the increase has occurred among male homosexuals. About 90% of syphilis cases worldwide occur in developing nations, where it is responsible for thousands of stillbirths and perinatal deaths annually.

Syphilis is caused by infection with Treponema pallidum, a spirochete that can enter the body through abraded skin or intact mucous membrane. A primary lesion (chancre) forms at the site of inoculation 2-12 weeks after infection. This is a firm papule or nodule, 0.5-2 cm or more in diameter, which gradually crusts over and ulcerates and is usually painless. Most chancre occur on the genitals, but any skin or mucous surface can be the site of invasion. Vaginal or cervical lesions often go unnoticed by the patient. A chancre is usually solitary, but sometimes two or more appear together. The surrounding skin becomes indurated (hardened), and regional lymph nodes may become swollen and tender. The chancre is an infectious lesion; contact with it is a principal means of spread of syphilis. Serum expressed from a chancre and examined by darkfield microscopy (which uses oblique rather than direct lighting of the specimen) shows motile spirochetes. The chancre heals spontaneously in a few weeks, sometimes leaving a scar.

This local and self-limited primary stage is, however, only the first manifestation of a wide-ranging and life-threatening systemic disease. Within a short time after inoculation, and even before the chancre appears, spirochetes are carried by the blood to every organ and tissue in the body. Some weeks after the appearance of the chancre, a secondary stage may occur, heralding the onset of systemic disease. The most common manifestation of secondary syphilis is a rash, which may be local or generalized and is highly variable in appearance. The palms and soles are often involved.

Condylomata lata (singular, condyloma lata) are soft moist papules or plaques that form on genital or inguinal skin. Ulcers or mucous patches may appear in the mouth or throat or on the cervix. Patchy hair loss and enlargement of spleen and lymph nodes may also occur. Cutaneous and mucosal lesions are infectious and yield positive results on darkfield examination. This secondary stage may not occur or may go unobserved. Occasionally secondary stage symptoms recur one or more times over a period of weeks.

After the secondary stage there may be no further overt symptoms, at least for many years, although spirochetes continue to cause progressive damage to many tissues, particularly in the cardiovascular and central nervous systems. This latent stage persists throughout life for as many as 75% of persons with untreated syphilis.

Late or tertiary syphilis occurs when signs or symptoms of organ damage appear. The principal pathologic change induced by spirochetes is an obliterative endarteritis of small arteries. Clinically this is manifested as cardiovascular syphilis (aortic valvular insufficiency, aortic aneurysm), neurosyphilis (general paresis, tabes dorsalis), or both. Paresis is a syndrome of generalized paralysis with dementia. In tabes dorsalis, damage to nerve tracts in the spinal cord leads to loss of position sense, abnormal reflexes, and disturbances of movement and coordination. Gummata (singular, gumma) are rubbery masses of fibrous tissue that form as a hypersensitivity reaction to T. pallidum. They can occur almost anywhere in the body, but are particularly common in the skin, bones, and liver. Their symptoms depend on their size and location.

A pregnant woman with untreated syphilis usually transmits the infection to the fetus. Congenital syphilis is associated with a 40% perinatal mortality rate, and survivors display various deformities and developmental stigmata of the bones (saber shins, frontal bosses), teeth (mulberry molars, Hutchinson’s teeth), eyes (interstitial keratitis), and nervous system (neurosensory deafness, paresis). Some of these may be evident at birth, while others do not develop until puberty or later.

Because of the broad variety of tissues that can be affected by syphilis, and the range of clinical effects, the disease is highly variable in its manifestations. To make a correct diagnosis the physician must often be particularly alert and must remember that syphilis has long been known as the “great imitator” because its signs and symptoms are so often mistaken for those of other diseases.

Demonstration of T. pallidum on darkfield examination of material from a lesion conclusively establishes the diagnosis.
Chancroid is yet another uncommon STD causing genital ulceration that must be differentiated from syphilis. This is an acute local infection manifested by one or more painful pustules that break down to form soft ulcers. Local lymph node involvement follows.

During the 1980s, a sharp rise in the incidence of genital herpes caused that disease to assume iconic status, chiefly through the perfervid zeal of the communications media. By the 1990s, herpes had been thrust completely into the shade by the horrifying advance of AIDS.

Herpes simplex is a local infection of skin or mucous membranes caused by either of two related viruses. In the 1980s, Type 1 herpes simplex virus (HSV-1) was typically associated with lesions of the lips and face (orofacial herpes, herpes labialis, cold sore, fever blister), while type 2 (HSV-2) was responsible for most lesions of the genitals (genital herpes, herpes genitalis). According to statistics published by the Centers for Disease Control and Prevention (CDC), up to 50% of first episodes of genital herpes are now caused by HSV-1.

Transmission of herpes simplex is by direct contact. Because, in theory, intact skin or mucous membrane is impermeable to the virus, infection implies at least superficial trauma at the inoculation site. Persons with latent infection (no active lesions) can spread the disease to others, at least in certain circumstances. The incubation period may be as short as one week, but sometimes the virus remains latent for months or years before causing symptoms.
Herpes simplex virus, once acquired, remains in the body for the life of the patient.

A person with orofacial herpes can shed infectious viral particles in respiratory secretions, including aerosolized droplets produced by coughing or sneezing, and in saliva. Before latex gloves became mandatory in dentistry, dentists and dental hygienists sometimes acquired herpes infections of the fingers, so-called herpetic whitlow.

Genital herpes is virtually always spread through sexual activity, including oral-genital contact. The virus can be transmitted through friction between any bare skin surfaces, as in wrestling. Infection acquired in this way is called herpes gladiatorum.

Regardless of its location, herpes simplex appears as a small cluster of thin-walled vesicles surrounded by a reddened zone of skin or mucous membrane. Itching or burning is often intense, and may precede the appearance of lesions. Within a day or two the vesicles slough and become shallow, painful ulcers. A first attack of herpes simplex may be accompanied by swelling and inflammation of regional lymph nodes and fever.

Herpes lesions heal after 1-2 weeks, as antibody formed by the immune system suppresses viral activity. Because antibody production continues throughout life, serologic testing can confirm a past history of herpes simplex infection and can distinguish between types 1 and 2. However, antibody does not destroy the virus or protect against recurrence of symptoms. Herpes simplex virus, once acquired, remains in the body for the life of the patient. After causing an orofacial eruption, the virus lies dormant in the trigeminal ganglion (containing cell bodies of sensory neurons in the fifth cranial nerve); after genital herpetic, in ganglia of sacral sensory nerves.

A recurrence of herpes simplex at the same site as the original eruption can be triggered by various physical or emotional stresses, including fever, sunburn, menses, and fatigue. Because of continuing antibody production, recurrent herpes simplex is usually milder than the primary attack and of shorter duration, and fever and lymph gland involvement seldom occur. Recurrences can come at intervals of days, weeks, months, or years. Many patients never experience any recurrences at all. Both recurrences and periods of viral shedding without overt symptoms are much less frequent with HSV-1 infection than with HSV-2. Susceptibility to first infection, severity of symptoms, and the likelihood of recurrences are all greater in immunosuppressive persons, including those with AIDS. About 90% of HIV-positive persons have antibody to HSV-1, and about 77% have antibody to HSV-2.

In men genital herpes usually affects the skin of the penis. In women, severely painful vulvar lesions typically occur, but when the cervix is the site of the eruption symptoms may be absent. Anorectal lesions result from anal intercourse.

Uncommonly, either type of herpesvirus can cause a severe, destructive ulceration of the cornea (herpetic keratitis, dendritic keratitis) or life-threatening encephalitis.

Neonatal infection, acquired at birth by a child born to a mother with active genital herpes, often leads to disseminated disease with a high mortality rate. Approximately 70% of cases of neonatal HSV occur when the mother is asymptptomatically shedding virus near the time of delivery. Because the risk of neonatal transmission approaches 100% if vaginal delivery occurs during acute maternal infection, cesarean delivery is the rule in such circumstances.

The diagnosis of herpes simplex is usually obvious from the patient’s history and the physician’s findings on direct examination. The diagnosis can be confirmed by either culture or serologic testing. Viable herpesvirus inoculated into a culture medium consisting of live cells causes cytopathic (cell-damaging) effects in 1-10 days. Culture is most reliable when the specimen is taken from a fresh lesion (vesicle), less so from an evolving one (ulcer). A negative culture does not rule out the presence of herpesvirus.

Detection of antibody to herpesvirus in the serum is firm evidence of infection and can distinguish between herpesvirus types 1 and 2, but not between active (recent) and prior (remote) infection. Moreover, antibody may not be detectable until several weeks after the infection has resolved.

Rapid diagnostic tests currently available for herpes simplex infection suffer from both poor specificity and poor sensitivity. In the Tzanck test, a smear of material from a herpes lesion stained with orthotoluidine may show balloon cells (multinucleated giant cells) containing viral inclusion bodies, but this test cannot distinguish between herpes simplex viruses and the closely related varicella-zoster virus. Antigen detection tests are not currently recommended for testing material from genital lesions. Polymerase chain reaction (PCR) can detect minute quantities of virus and distinguish between viral types, but because of expense it is usually reserved for examination of spinal fluid in encephalitis.

Screening of asymptomatic patients for herpesvirus infection is not currently recommended, with the exception of persons at high risk (partners of persons with herpes, prostitutes). However, patients diagnosed with genital herpes are screened for other sexually transmitted diseases.

A first episode of genital herpes is treated with an orally administered antiviral drug of the nucleoside analogue class (acyclovir, famciclovir, or valacyclovir) in high dosage. Dosage is reduced for recurrences. Patients with frequent recurrences may take a still lower, prophylactic dose daily for extended periods.

Genital Papules: Genital Warts and Molluscum Contagiosum

A wart is a benign tumor induced by infection with the human papillomavirus (HPV). More than 80 HPV types can be distinguished on the basis of their DNA structure. Certain
types tend to involve certain tissues—the soles of the feet (plantar warts, types 1, 2, and 4); the extremities, particularly the hands (common warts, verrucae vulgares, types 2, 4, 26, 27, 29, and 57); the external genitals (genital or venereal warts, condylomata acuminata, types 6 and 11). Some cause lesions in the mouth (focal epithelial hyperplasia, types 13 and 32) or respiratory tract (recurrent respiratory papillomatosis, types 6, 11, and 30).

Warts are transmitted from person to person by direct skin contact. Plantar warts are spread indirectly by surfaces such as bathroom and locker room floors. The incubation period is usually 1-2 months, but it may be much longer. Latent (asymptomatic) infection is commoner than was once thought. Although a wart may remain solitary indefinitely, most patients have several, and some have hundreds, by the time they seek treatment.

Warts are variable and unpredictable in their behavior. Most of them eventually regress and disappear spontaneously, probably because of an antibody response in the patient. But warts can persist for long periods, reappear after apparent regression, or become extremely large. In recent years the recognition of a causal connection between some types of HPV and cervical cancer has vastly altered the way in which that disease is viewed and treated.

Genital warts (also called venereal warts), which occur on the skin and mucous membranes of the genitals, perineum, and anus, are spread almost exclusively through sexual contact. Perianal spread may result from anal intercourse but is often due to migration of virus from the patient’s own genital lesions. Genital warts are highly contagious: 60 to 90% of sexual partners of persons with genital warts also have genital warts. Genital warts are more likely to develop during pregnancy and in persons with impaired immunity.

HPV infection is the most common STD in the U.S. Over 6 million people acquire genital HPV infections every year in the U.S., about 50% of them between the ages of 15 and 25. It is believed that 50-75% of the reproductive-age population has been infected with sexually-transmitted HPV at some point in life. HPV infection is the most common STD in the U.S. Over 6 million people acquire genital HPV infections every year in the U.S., about 50% of them between the ages of 15 and 25. It is believed that 50-75% of the reproductive-age population has been infected with sexually-transmitted HPV at some point in life.
Although bacterial vaginosis is usually just a nuisance, symptomatic infection occurring during pregnancy is associated with increased risks of preterm labor, premature rupture of the membranes, low birth weight, and postpartum infection.

Sexual partners of patients with HPV infection are examined by colposcopy or androscopy after application of acetic acid to identify visible genital warts, and in women a Pap smear is done.

Two vaccines produced by recombinant DNA technology have been approved by the U.S. Food and Drug Administration for the prevention of infection due to certain types of HPV. The target of Cervarix (GlaxoSmithKline), which stimulates immunity to HPV types 16 and 18, is cervical cancer. Cervarix is recommended only for prepubertal girls. Gardasil (Merck & Co.), a quadrivalent HPV vaccine protecting against cervical dysplasia and cancer due to types 16 and 18 and also against the 90% of genital warts caused by types 16 and 18, is recommended for young persons of both sexes.

Molluscum contagiosum is a viral eruption of the skin and occasionally of mucous membranes that is of little clinical importance except that it must be differentiated from genital warts. Molluscum is spread by direct contact. Lesions often occur on the upper trunk and extremities in children and wrestlers, and epidemics have occurred among mentally retarded persons in institutions. Lesions of the genitals and surrounding skin in adults are virtually always sexual in origin. In some studies, two-thirds of patients with molluscum contagiosum had at least one other STD.

The lesion of molluscum is a round raised papule, 1-5 mm in diameter, with a characteristic pearly pink color. Lesions are often umbilicated—that is, they have a central pit or dell. Typically 10-25 lesions are found scattered over the pubic and inguinal skin by the time a patient seeks treatment. The microbiology of this disorder is not fully understood, but it appears to be due to an abnormal mingling and interaction of normal flora and certain circumstances. Other typical findings are a vaginal and occasionally of mucous membranes that is of little clinical importance except that it must be differentiated from genital warts. Molluscum is spread by direct contact. Lesions often occur on the upper trunk and extremities in children and wrestlers, and epidemics have occurred among mentally retarded persons in institutions. Lesions of the genitals and surrounding skin in adults are virtually always sexual in origin. In some studies, two-thirds of patients with molluscum contagiosum had at least one other STD.

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Diagnosis is generally obvious on inspection. It can be confirmed by examining a stained or unstained smear of material from a lesion for molluscum bodies (characteristic viral inclusion bodies). Lesions are readily removed with a ring curet, separating cleanly from normal skin with so little pain that anesthesia is rarely needed. Liquid nitrogen and topical applications or podophyllin or trichoracic acid are also used.

Vaginitis

Vaginitis is inflammation of the vagina (and usually also of the vulva) as manifested by vulvovaginal pain or itching and vaginal discharge that is abnormal in volume, consistency, color, or odor. Three forms of vaginitis are associated with sexual transmission of microorganisms.

Candida albicans, a yeastlike fungus, is present on the skin and in the digestive tract of many normal persons. Under certain circumstances Candida can overgrow and invade epithelial surfaces, causing dermatitis, oropharyngeal infection (thrush), esophageal infection (especially in AIDS), or colitis.

Candidal vaginitis (vaginal candidosis or candidiasis) causes intense vulvar itching and a thick, white, curdy discharge. Like most other forms of candidal infection, candidal vaginitis is more likely to occur in diabetics, pregnancy, and immunodeficiency, and after antibiotic treatment, which suppresses the normal bacterial flora of the vagina. It is also more common in women taking oral contraceptives because of changes in the biochemistry of vaginal secretions. While this condition is probably not usually acquired by sexual contact, the sexual partner of a woman with candidal vaginitis may have a candidal dermatitis of the genitals, and may reinfect the woman after treatment.

Diagnosis is made by finding fungal elements (hyphae) on microscopic examination of vaginal secretions that have been treated with potassium hydroxide (KOH) to destroy human cellular material. Diagnosis may be confirmed by culture. Topical treatment with over-the-counter antifungal creams or suppositories containing butoconazole, clotrimazole, miconazole, or tioconazole often suffices to suppress candidal overgrowth and relieve symptoms. A single oral dose of fluconazole, available only by prescription, is curative.

Recurrences of candidal vaginitis are common, particularly in the continuing presence of risk factors. Prophylactic doses of oral or topical antifungals can reduce the frequency of recurrences.

Although traditionally classed as a form of vaginitis, bacterial vaginosis (BV, also called nonspecific vaginitis or Gardnerella vaginitis) causes little if any inflammation or itching. Usually the only symptom is a copious, gray, malodorous discharge. Asymptomatic infection is relatively common. The microbiology of this disorder is not fully understood, but it appears to be due to an abnormal mingling and interaction of anaerobic bacteria and Gardnerella vaginalis, a gram-negative coccobacillus. Like candidal vaginitis, this condition can arise with no prior sexual contact, but it is much more common in sexually active women.

BV is diagnosed by excluding other causes of abnormal vaginal discharge and by finding “clue cells” on microscopic examination of a wet preparation of vaginal secretions. Clue cells are vaginal epithelial cells studded with numerous Gardnerella organisms. Other typical findings are a vaginal

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pH higher than 4.5 and the release of a fishy or amine odor when potassium hydroxide is added to secretions in preparation for examination for *Candida*. Treatment with oral or vaginal preparations of metronidazole or clindamycin is promptly curative, but recurrences are common.

Although bacterial vaginosis is usually just a nuisance, symptomatic infection occurring during pregnancy is associated with increased risks of preterm labor, premature rupture of the membranes, low birth weight, and postpartum infection. Although eradication of symptomatic BV in pregnancy is therefore mandatory, there is no evidence that treating asymptomatic infection alters the risk of complications. Screening of pregnant women without symptoms for bacterial vaginosis is not recommended.

**Trichomonal vaginitis** is due to a protozoan parasite, *Trichomonas vaginalis*, which causes itching and a frothy green discharge. Unlike the other forms of vaginitis, trichomonal infection is exclusively an STD. Infrequently it causes urethritis in infected men. Diagnosis is made by finding the motile parasites in a wet preparation of vaginal secretions. The treatment of choice for this infection is metronidazole. Sexual partners are treated prophylactically.