Today, in the United States as well as elsewhere, sexually transmitted diseases (STDs) have reached epidemic levels. Many of these STDs have skin and mucous membrane manifestations. Changes in sexual mores, including wider acceptance of sexual activities with multiple partners and of homosexuality, may contribute to the problem. New transmission routes of pathogenic agents (eg, in amebiasis, giardiasis, and hepatitis) are definitely occurring. The social and financial costs of STDs are staggering. A sexually transmitted disease is defined as one for which sexual contact is epidemiologically significant but need not be the only mode of transmission. STDs include viral infections such as warts, molluscum contagiosum, herpes simplex, hepatitis, cytomegalovirus disease, and infectious mononucleosis; arthropod infestations such as scabies, body lice, and pubic lice; yeast infections; and the "classic" venereal diseases—syphilis, gonorrhea, lymphogranuloma venereum, chancroid, and granuloma inguinale. Additional STD problems include contact dermatitis, traumatic skin ulcers, and acquired immune deficiency syndrome (AIDS).

Warts

Like all human warts, genital warts, or condylomata acuminata, represent infection with the human papilloma virus (HPV). Warts may be transmitted by indirect or direct contact, including autoinoculation. Affected patients often lack circulating antibodies against the wart virus. Immunosuppressive diseases, recurrent infections of other kinds, and intraepithelial neoplasms of the genital tract afflict women with condylomata more frequently than controls.1,2

The typical condyloma acuminatum is a fleshy, nonhorny wart most commonly seen on the genitalia (Figure 1A), the mouth, or the perianal area (Figure 1B). When large, the wart's surface may resemble a piece of cauliflower! These viral tumors must be distinguished from condylomata lata, a form of secondary syphilis.

Warts may spread from the hands to the vulva and the vagina. Intraoral warts (Figure 1C) are most
commonly contracted via oral sex. Perianal warts may arise from episodes of anal intercourse.

The treatment of warts usually involves some destruction of the host cells. It is undertaken because viral spread and multiplication can cause discomfort as well as persistent reinfection. However, such treatment should be conservative and nonscarring since the disease is relatively benign.

While podophyllin resin and electrocautery have previously constituted standard methods, topical application of liquid nitrogen is now usually the treatment of choice. Such cryosurgical ablation is relatively painless, quick, and effective. All patients should be seen three weeks after initial therapy to identify and destroy recurrent or new warts.

As in all sex-related skin disorders, patients should alert their sexual partners to examine themselves for any evidence of this highly contagious disease.

**Molluscum Contagiosum**

Molluscum contagiosum is caused by a pox virus. Though one of the largest viruses to infect man, it has never been isolated in vitro. (Man is its only natural host.)

The clinical lesion is a discrete, pearly-hued papule with a characteristic central umbilication (Figure 1D). Lesions are often multiple. This disease has gradually changed from being an atypical infection to being an STD. In some studies as many as 67% of affected individuals had additional venereal disease.

Autoinoculation is common, accounting for the common clinical appearance of multiple lesions in a linear pattern along the site of a scratch. Untreated disease may persist for months or even years, sometimes forming giant lesions the size of a dime.

Treatment modes vary. Surgical removal of the central core, while often done, may be painful to the patient and permit recurrence. Topical application of liquid nitrogen is usually less painful and usually curative after only one treatment. Healing occurs without scarring since the lesions are superficial.

**Herpes Simplex**

Infections caused by herpes simplex virus (HSV) (Figure 2) are commonly divided into primary and recurrent disease. An estimated 20% of the United States population has had a herpes infection, making it one of the most common viral infections, if not the most common. The incidence of herpes seems much higher on college campuses; while the ratio of gonorrhea to herpes in the general population is 10:1, that incidence ratio was found to be reversed among students of at least one California university.

People with no circulating antibody to the virus are presumably susceptible to primary lesions. Primary lesions are acquired by close personal contact, including sexual intercourse. Recurrences may occur with physical or emotional trauma or stress, which includes sunburn, fever, sexual activity, menstruation, and psychological upset.
SKIN LESIONS SEEN WITH STDs: CONDYLOMATA ACUMINATA, MOLLUSCUM CONTAGIOSUM, HERPES SIMPLEX

A. Penile warts
B. Perianal warts
C. Wart on labial mucous membrane
D. Molluscum contagiosum on areola
E. Penile herpes simplex
F. Herpes simplex on buttock

Figure 1
The family Herpesviridae may be divided into 3 subfamilies on the basis of their biologic properties. Viruses that infect humans are distributed among these subfamilies as follows:

- **Alpha**: HSV-1, HSV-2, and the varicella-zoster virus
- **Beta**: Cytomegalovirus
- **Gamma**: Epstein-Barr virus

**Structure of the Herpesviridae virion**

- **Double-stranded DNA core**
  - Long segment—82%
  - Short segment—18%
- Each segment has a unique nucleotide sequence bounded by repeated and inverted patterns.
- **Capaeid**—a 20-faceted (icosahedral) structure, consisting of 162 capsomeres, which encases the DNA
- **Tegument**—composed of globular material
- **Envelope**—a bilayered membrane with surface projections
The virus (A) enters the host cell (B) either by pinocytosis or by fusion of the virion envelope with the cell plasma membrane. Within the cytoplasm, uncoating occurs, and viral DNA enters the host nucleus (C). Thereafter transcription of messenger RNA (D) and synthesis in the cytoplasm of viral proteins (E), which enter the nucleus (F) to take part in viral DNA replication (G) and capsid assembly (H). Maturation occurs by budding of the nucleocapsids through the inner nuclear membrane (I). Enveloped virus particles are then released from the cell (J).

This replication cycle lasts about 8 hours in cell culture (and probably about the same amount of time in vivo).
Of the two types of herpes simplex virus, type 1 (HSV-1) produces 85% of infections above the waist (eg, mouth, face, eye, and brain), while type 2 (HSV-2) is responsible for most infections below the waist (eg, vulva, cervix, penis, anus, sacrum, and buttocks). However, a new study by Lipson, Szabo, and Lin (soon to be published) showed a population of Long Island (New York City suburban) teenagers with genital/perianal herpes to have a higher incidence of HSV-1 infection—believed due to increased popularity of orogenital sex in this age group.

Both types produce primary and recurrent infections; they are indistinguishable clinically. Virologic differentiation requires typing by immunofluorescence or plaque size in culture. Type 2 infection is the most common sexually spread virus of the female genital tract and has been incriminated as a causative factor in cancer of the cervix. By contrast, no relationship exists between type 2 herpes and cancer of the prostate.

Herpes progenitalis is the most common cause of recurring genital ulcerations (Figure 3), but clinical examination of such ulcers cannot make the diagnosis. This requires culture and serial serology studies beginning at the vesicular stage of a recurrence. Eruptions are often heralded by a prodrome of tingling or itching of the skin (locally or elsewhere), followed within 24–48 hours by the appearance of grouped vesicles on a red base (Figures 1E/F). Within a few days these vesicles rupture, leaving erosions, ulcers, and often crusts—and sometimes secondary bacterial infection with severe pain as well.

Herpetic ulcerations on the cervix are often misdiagnosed as nonspecific “erosions.” Orogenital contact may result in herpangina. Urinary retention and anorectal pain may also occur due to herpes simplex. Neonatal infection during the third trimester of pregnancy often causes infant death. Disseminated forms of infection, such as aseptic meningitis, are more common in immunologically compromised patients.

Management of cutaneous herpes simplex infections is still chiefly symptomatic. The tendency of herpes simplex episodes to recur in a given individual because of viral latency makes specific treatment extremely difficult. Because involvement of the skin by the virus appears secondary to intraneuronal replication, topical therapy proves no more than palliative and cannot be expected to significantly influence recurrence rates. The large number of suggested (usually unscientific) treatments for this disease underscores this problem. Among these, smallpox vaccinations are not only ineffective but also dangerous, as they can lead to encephalitis.

Topical idoxuridine preparations have successfully cured ocular lesions but not skin eruptions. Use of ribavirin, a synthetic nucleoside analog, and low-concentration zinc sulfate solution has most recently been reported effective, but controlled double-blind studies have not yet confirmed this.

Acyclovir has been found effective for systemic infections in immunosuppressed hosts. Oral acyclovir can shorten the duration of
HERPES SIMPLEX: FUNDAMENTAL PATHOGENETIC FEATURES

Primary infection

HSV enters the host organism (A) through mucocutaneous surfaces; the extent of virus replication at the inoculation site determines the clinical severity of the primary infection. After implantation in the host surface tissue (B), the virus begins to migrate toward corresponding sensory ganglia (C). During the acute phase of the disease, infectious virus can be demonstrated in cell-free sensory ganglia (D) homogenates of experimentally infected animals. The amount of infectious virus increases during the first 4-5 days, then declines, and cannot be detected in homogenates 2 weeks after primary infection.

Latent infection

After healing of the primary site (E) lesions, humoral and cell-mediated immune responses develop. Viruses in the sensory ganglion neurons (D) now assume a latent form.

Reactivation of latent virus

The hallmark of reactivation is the development of recurrent lesions (fever blisters, cold sores, genital herpes, keratoconjunctivitis) at sites innervated by latently infected neurons (F). The reactivated virus reaches these mucocutaneous sites by centrifugal axonal migration (G). The characteristic clinical lesion may then evolve at the mucocutaneous site in the sequence of inflammation, necrosis, and ulceration.

Reactivation can be elicited by a series of inducing factors that may act directly or indirectly on latent virus. A specific inducing action has not been established. The mechanism by which commonly associated factors, such as fever, emotional disturbance, and menstruation, are related to reactivation of latent virus is not known.

Frequency and intensity of recurrent HSV episodes vary greatly among patients—and even in a single patient. Their variability may be related to the antigenic type of latent virus and the specific ganglia in which latency is established.

Figure 3
both viral shedding and lesion presence in patients with recurrent genital herpes when therapy is self-initiated early in the course of a recurrent episode. This unique drug is incorporated by the virus to cause its self-destruction, but resistance to acyclovir has also been reported, suggesting development of resistant strains. Nevertheless, an oral form of the drug has recently been approved by the FDA.

Other aids for the herpes sufferer include warm compresses, mouthwashes, topical ether (to dry lesions), oral antibiotics (for secondary bacterial infection), and counseling support. The physician should remember that persons afflicted with this disease may also be at higher risk of having another sex-related skin disorder.

**Hepatitis**

While people usually contract hepatitis A by eating or drinking contaminated food or water, hepatitis B can be transmitted sexually. There are nearly a million chronic carriers of hepatitis B surface antigen (HBsAg) in the United States, and this pool of carriers is growing by 2–3% annually. Contact with body fluids (urine, saliva, semen, and vaginal secretions) is now known to be a major source of infection. Hepatitis B also occurs almost nine times more often among homosexual than heterosexual men. Unfortunately, there is still no specific cure, and no treatment will modify or terminate the hepatitis B carrier state.

The incubation period for type B virus is four weeks to six months.
VIRAL HEPATITIS: CLINICAL FEATURES

The clinical picture may be identical in HAV, HBV, and non-A, non-B disease, but more severe illness is usually seen with hepatitis B.

Incubation periods

Type A: two to six weeks
Type B: four weeks to six months
Non-A, non-B: probably four to 12 weeks

Symptoms and signs

Fatigue, malaise, and weakness are seen early in all three variants.

During the prodromal period, poor appetite, nausea, and intolerance to fatty foods and cigarettes may be associated with low-grade fever, headache, arthralgias, and abdominal pain.

Such complaints may increase with onset of the icteric phase. Jaundice usually peaks and begins to decrease within several days, but it may be more protracted in hepatitis B.

Hepatomegaly may be noted in 50–60% of cases.

Other findings may include:
- Splenomegaly (about 10%)
- Lymphadenitis (occasional)
- Urticaria and arthritis
- Glomerulonephritis (rare)

Note: The urticarial rash, usually most prominent on the distal extremities, lasts from three days to a week—fading as jaundice appears.

The convalescent phase of hepatitis begins with disappearance of jaundice and improved patient strength; this phase may last a few weeks (or be more prolonged with "posthepatitis syndrome").

Figure 4
(Figure 4). About 5% of patients in the preicteric phase of hepatitis develop a rash, which may be urticarial or, less commonly, maculopapular or erythematous and may be accompanied by migratory polyarthralgias. The rash, most prominent on the distal extremities, is immunologically mediated by antigen-antibody complexes that bind complement. The rash lasts from three days to a week, fading as jaundice occurs.

Jaundice is abnormal yellowish pigmentation of the skin, mucous membranes, and sclera resulting from an elevation of bile pigments. This, of course, indicates hepatic dysfunction and is usually easiest to detect in the sclera and on the trunk while using natural lighting.

The relative risk of developing primary liver cancer for hepatitis B carriers is almost 300 times greater than that for noncarriers, while 3–5% of patients with acute disease progress to chronic active hepatitis as a late complication.

A relatively new hepatitis B vaccine induces antibodies (anti-HB_s) to HB_sAg in over 90% of vaccine recipients and has proven virtually 100% effective preventing hepatitis B in those who develop anti-HB_s. Immunity probably lasts for five years for patients who have received all three doses (boosters at one and six months). Certain particularly high-risk groups, including healthcare personnel, should be considered prime candidates for this new vaccine.

**Cytomegalovirus Disease**

Human cytomegalic inclusion disease (CID) is caused by cytomegalovirus (CMV), a member of the herpesvirus group. CMV was first isolated from the salivary gland in 1956. It usually causes a subclinical infection in healthy, immunocompetent hosts. Transmission involves close or intimate contact with an infectious individual. CMV has also been isolated from urine, feces, saliva, and cervical secretions—the last suggesting that venereal transmission may be common. Petechial and purpuric exanths are the most common clinical findings, especially in congenitally acquired disease.

**Infectious Mononucleosis**

Infectious mononucleosis is caused by the Epstein-Barr virus (EBV), another member of the herpes group. Exanths occur in only about 3–5% of patients with the disorder; this eruption may be maculopapular, mimicking rubella, or manifest only a faint erythema. Much more commonly seen is a petechial palatal exanthem, notable in about 25–60% of patients (Figure 5). Virtually all patients with mononucleosis who take ampicillin develop a pruritic, maculopapular eruption within one week of starting this antibiotic.

**Scabies**

Scabies is caused by the tiny mite Sarcoptes scabiei. The adult female arthropod burrows into the superficial skin of the human host, laying eggs in the tunnels it has made. The main clinical complaint of nocturnal itching probably represents an allergic reaction to the mite's intracutaneous feces. Red, edematous, and often excoriated papules on typical locations—finger webs, penis and scrotum (Figures 6A/B), groin, buttocks, and elbows—are characteristic. The diagnosis often gains support when the patient's close contacts give a similar history of itching. Yet scabies is a disease of protean and confusing clinical expression; generalized urticaria may be the only presenting symptom.

Treatment includes lindane lotion for the body—applied evenly from the neck down, left on the skin for 8–24 hours, washed off, and repeated in one week. Lindane is a safe and effective drug when used properly. Topical crotamiton is also scabicidal and relieves pruritus as well.

The mites die within three days once off the human host. Hot-water laundering of infested clothes, linens, and towels can kill the parasites faster. Mite-killing sprays (for other inanimate objects) are also available.

Long-standing disease can produce larger, firm, red papules, most common on the elbows (“scabetic nodules”), that may require intralesional dilute corticosteroid injection for complete resolution. Infestation can occur without symptoms, and thus absence of active mites cannot always be determined by clinical examinations. All sexual and other (human) household contacts should therefore be treated even if asymptomatic.

**Lice**

The pubic louse, Phthirius pubis,
most commonly lives in the pubic hair but may also infest the eyelashes, eyebrows, and hairy areas of the chest, belly, and thighs (Figure 6C). The insect most often nests near the base of the pubic hair. The physician should also search for maculæ ceruleæ, grayish-blue spots on the skin produced by the anticoagulant such lice inject into the skin with their bloodsucking bites.

Lindane shampoo and lotion constitute the treatment of choice, applied once a week for two weeks, along with combing of involved hair using a fine-toothed comb to remove all nits. Measures to kill lice on inanimate objects parallel those described for mites.

Yeast Infections

Candida albicans can be detected in 5% of all gynecologic patients, but only one third of these women will report symptoms—usually intense vaginal itching. Physical exam often reveals a cheesy white vaginal discharge. The likelihood of such infection may rise with use of oral contraceptives or oral antibiotics and during pregnancy. One study found that 49% of male consorts of women harboring genital yeast were positive on culture,\(^9\) while another study noted that 80% of female consorts of men carrying yeast on the penis had vaginal yeast.\(^1\) The significant association of Candida with STDs supports the idea that genital yeast infection is often sexually transmitted.\(^32\)

In the male, this problem often presents early as red penile papules with some satellite pustule formation—other times simply as red, tender skin on the penile shaft or patches of red, shiny skin on the glans (Figures 6D/E).

One can prescribe miconazole, clotrimazole, or haloprogin as a cream, solution, or vaginal suppository. Oral nystatin should be given to minimize recurrence from GI sources. Griseofulvin is ineffective in killing Candida. Ketoconazole is now indicated only in systemic infections or in widespread mucocutaneous candidiasis.\(^33,34\)

Gonorrhea

Gonorrhea is caused by the gram-negative gonococcus (GC), Neisseria gonorrhoeae. This organism has been subdivided into at least five types based on culture studies. Recent serologic evidence implies that gonococci of different strains produce particular types of disseminate clinical effects, including skin manifestations.

Statistics on gonorrhea are understandably inaccurate. In the USA over one million cases are documented annually; although this figure is believed to represent under-reporting of 62%,\(^35\) gonorrhea remains the most commonly reported infectious disease. The economic cost of this disease approaches $300 million yearly, and its incidence continues to increase due to new trends in sexual behavior, frequent changing of partners, population mobility, gonococcal resistance to antibiotics, and varying techniques of contraception (ie, condoms versus other methods).

The high (85%) clinical risk of contracting GC during coitus with an infected partner accounts for rampant spread of the organism among communities, especially male homosexuals with increased numbers and frequency of sexual contacts. The incubation period ranges from three to seven days. The most common symptoms in men are a yellowish discharge from a reddened urethral meatus (Figure 6F) and painful urination. This discomfort usually makes the patient seek medical care. (The fact that 30% or more of men and over 70% of women have asymptomatic infections poses a major epidemiologic problem, requiring great efforts to identify and treat these unsuspecting disease carriers.)

Gonococci can also cause infections of the throat (possibly more common in individuals with orogenital contact), vulvovaginitis, and a dermatitis-arthritis syndrome that may be accompanied by tenosynovitis. Gonococcal septicemia can be fulminant and result in fatal endocarditis; or it may take a more benign form producing fever, migratory arthritis, and typical skin lesions (red papules and pustules, located most often on the hands and feet, that evolve into purpuric areas and possibly necrosis).

Diagnosis of GC infection can be suggested by Gram staining a discharge specimen and finding gram-negative intracellular diplococci, or made definitively by culture using Thayer-Martin medium or “chocolate” agar. A blood test can also be useful for screening individuals in high-risk populations: A positive slide agglutination test result strongly suggests present or past in-
INFECTIOUS MONONUCLEOSIS: POSSIBLE FINDINGS IN ADULTS

The clinical manifestations of EBV infection largely reflect the immune response of the host. Primary infection early in childhood is often asymptomatic or mild, while adults may manifest more pronounced symptoms.

Fever for 10–14 days
(In older patients, fever may be the predominating sign and last 22–30 days.)

Lymphadenopathy (mildly tender, involving axillary, posterior auricular, inguinal, cervical, and/or submandibular nodes)

Headache, malaise

Jaundice (5%)

Palatal petechiae (more significant in the patient who also has sore throat)

Cough

Hepatomegaly (10%)

Laboratory abnormalities:
- Heterophil antibodies
- Cold agglutinins (common)
- Reduced platelets (50% of patients)
- Neutropenia (most patients)

Spienomegaly (50%) — maximal at start of the second week

Abdominal discomfort

Rash (macules, petechiae, erythema, urticaria, or erythema multiforme)

Most patients with infectious mononucleosis recover over a period of 2–3 weeks, and full recovery is the rule even in those who develop complications.

Figure 5
fection (with a false-positive rate of 5%); a negative result could still occur in a patient with very recent infection whose antibodies have not yet reached sufficient quantities to be detected by the test.

Pelvic inflammatory disease (PID) represents a serious progression of infection in women that can cause peritonitis and sterility.

Effective therapy of uncomplicated gonorrhea is based on a short course of high-dose antibiotics. Three valid treatment regimens are: aqueous procaine penicillin G, 4.8 million units given intramuscularly; 3.5 grams of ampicillin with one gram of probenecid, both given orally; and tetracycline HCl, 1.5 grams orally followed immediately by 500 mg qid for five days. There has been a recent and steady increase in the incidence of “resistant” penicillinase-producing N gonorrhoeae. Spectinomycin (2–4 g IM) or cefoxitin (2 g IM, given with a gram of oral probenecid) has comprised effective therapy for such infections. Oral erythromycin stearate or estolate, 500 mg qid for five days, has proven successful in curing disseminated gonococcal infection.

Syphilis

Syphilis is caused by Treponema pallidum, a spiral-shaped bacterium. The disease is systemic from its onset and may generate skin manifestations at all three stages of infection. The organism invades the body through cut or abraded skin or through the mucous membrane. Some postulate that the more commonly traumatic sexual practices of male homosexuals (eg, anal intercourse) contribute to this group’s higher incidence of the disease. In 1980 there were over 27,000 reported cases of primary and secondary syphilis, and statistics indicate that the disorder is waxing rather than waning.42

The incubation period ranges from one to 12 weeks. The initial chancre (Figure 7A) is usually solitary and painless, with a firm base and a moist surface. Untreated, it will generally heal within a month. About 75% of patients with secondary syphilis have some sort of skin eruption; 50% have swollen regional lymph nodes. Manifestations of secondary syphilis start four to eight weeks after the primary chancre appears (so that both may be present simultaneously in some patients).

A recent study showed that many physicians fail to recognize the symptoms and signs of secondary syphilis. Though the rash can mimic many other skin disorders, a key to diagnosis lies in its nonirritating and usually asymptomatic nature. Discrete red, peripherally scaly lesions commonly resemble pityriasis rosea (Figure 7B). “Mother-eaten” patches of hair loss and reddish papules (later becoming hyperpigmented spots) on the palms (Figure 7C) and soles may also occur.

After these cutaneous signs of secondary syphilis have spontaneously disappeared, the patient again becomes asymptomatic and has entered the “latent” stage of the disease. Due to the frequency of penicillin use for acute infectious disease, cases of late (tertiary) syphilis—including tabes dorsalis and necrotic gumma formation—are now fortunately rare.

Congenital syphilis may present as a popular rash in the diaper area, often accompanied by hepatomegaly and rhinitis.

Syphilis can be detected within a few minutes using dark-field microscopy and a freshly scraped preparation from a primary or secondary lesion: Spirochetes should be seen making their typical gyrations. Note that T pallidum cannot be cultured in vitro. Since syphilitic infections produce two types of antibodies, two basic types of blood tests allow specific diagnosis. (1) The classic complement fixation (Wassermann) test, the flocculation reaction (VDRL), and the rapid plasma reagin (RPR) test all detect antilipid antibodies. (2) Formation of specific antitreponemal antibodies allows detection by the fluorescent treponemal antibody absorption (FTA-Abs) test, which is extremely sensitive and can make the diagnosis very early in the infection’s course.

In contrast to GC, which has developed some penicillin-resistant strains and which can be killed by short, high-dose antibiotic therapy, T pallidum has few if any penicillin-resistant strains but needs constant serum antibiotic concentration over a longer period of time to be eradicated. As treatment for primary or secondary syphilis, 1.2 million units of benzathine penicillin G are given IM in each buttock weekly for two successive weeks. Three such doses at one-week intervals should be given for late syphilis. Oral tetracycline, erythromycin, or minocycline can be substituted in patients allergic to penicillin.
SKIN LESIONS SEEN WITH STDs: SCABIES, LICE, YEAST INFECTION, GONORRHEA

A. Scabies of penis and scrotum

B. Another case of genital scabies

C. Lice in pubic and thigh hair

D. Yeast dermatitis on penile glans

E. Another case of yeast infection

F. Urethral discharge of gonorrhea

Figure 6
Other “Classic” Venereal Diseases

**Lymphogranuloma venereum (LVG)** is caused by *Chlamydia trachomatis*. More common in tropical and subtropical climates, the disease usually manifests as localized involvement of the lymphatic system. The primary lesion of LVG is a small spot or ulcer, usually on the penis or vulva, so insignificant in appearance as to mimic a “pimple.” After one to four weeks, however, one or both sets of inguinal lymph nodes swell, mat together, and become very painful and tender. The classic “sign of the groove” results from clear demarcation between upper and lower groups of glands. This inguinal bubo (Figure 7D) forms an abscess and ruptures, but even more dangerous are the later complications of LVG: Chronic lymphedema may lead to irreversible elephantiasis with gross swelling of the vulva (“esthiomene”) or male genitals (“saxophone penis”). Anorectal symptoms occur more commonly in women and male homosexuals. Sulfonamides or tetracycline can be curative in earlier stages.

**Chancroid**, or soft chancre, is caused by a bacterium, *Haemophilus ducreyi*. It appears unique among sexually transmitted diseases in that it seems to respect social class, more commonly attacking economically unfortunate people living in warmer climates. Genital ulcers develop within one week of contracting the disease. Unlike syphilis, chancroid more often causes multiple ulcers than solitary lesions (Figure 7E). Infection spreads to regional lymph nodes, with subsequent breakdown and discharge of pus. Sulfonamides prove effective if local hygienic measures are also employed.

**Granuloma inguinale** (GI) is common in parts of South America, southern China, and other warm areas. It is caused by *Calymmatobacterium granulomatis*, a gram-negative bacillus with a distinctly large capsule and a bulge of chromatin (the Donovan body) at one end that give it the look of a closed safety pin. The initial lesion is a beefy red, painless papule or vesicle that ulcerates and forms a raised, velvety mass. These lesions can progress to eat into skin and underlying tissues, including bone, without causing significant lymphadenitis. Tetracycline and streptomycin are the drugs of choice.

### Disseminated Kaposis Sarcoma

A recently described and usually fatal STD epidemic involves a disseminated type of Kaposis’s sarcoma seen primarily in New York and California among homosexual men with AIDS. Most victims have been younger than 35 at the time of diagnosis, with skin lesions the most frequent presenting complaint. Most have multiple blue-purple to red-brown papules, from 4 mm to 2.4 cm in diameter, distributed over their bodies. (Early lesions in swarthy patients may be hyperpigmented.) Many of these men have associated generalized adenopathy. The disease more closely resembles the rapidly fatal form of Kaposis’s sarcoma occurring in equatorial Africa than the classic disease Kaposis described in 1872, which affected older men with localized tumors and had a chronic and relatively benign course (Figure 8).

Epidemiologic studies reveal a strong history in most of these younger men of prior sexually transmitted diseases, including gonorrhea, hepatitis B, syphilis, amebiasis, condyloma acuminata, herpes genitalis, giardiasis, and lymphogranuloma venereum. Many also use multiple “recreational” drugs, including amyl and butyl nitrite.

Severe immunosuppression primarily involves the cellular rather than the humoral aspect of the immune system. Some individuals have Pneumocystis carinii pneumonia, toxoplasmosis, severe herpes simplex, and/or elevated serum EBV antibody titers (known to induce immunodeficiency).

### Miscellaneous STDs Involving Skin and Mucous Membranes

Fellatio has been incriminated as a cause of soft palate trauma. A broad band of ecchymosis or abrasion can extend across the soft palate, or bilateral areas of petechiae may be detected by carefully inspecting the patient’s mouth and oropharynx. This may provide a lead in explaining other STD-related problems. Fellatio can also cause purpura of the penis (Figure 7F).

Contact dermatitis from feminine deodorant douches can involve both the vaginal area and the penis of the male sexual partner. Scented or flavored products most commonly
SKIN LESIONS SEEN WITH STDs: SYPHILIS, LYMPHOGRANULOMA VENEREUM, CHANCROID, TRAUMATIC PURPURA

A. Primary syphilitic chancre

B. Rash of secondary syphilis

C. Another patient with secondary syphilis

D. Lymphogranuloma venereum

E. Multiple lesions of chancroid

F. Penile purpura due to fellatio

Figure 7
cause such irritation. (A povidone-iodine vaginal disinfectant has even produced short-term elevation of serum iodine levels, which could result in suppression of neonatal thyroid hormonogenesis.) Men may become allergic to condoms, usually caused by the antioxidants used to maintain their elasticity, resulting in a red, itchy dermatitis of the penile shaft. Vaginal creams containing estrogens, in addition to
irritating the skin, have even been incriminated as a cause of gynecomastia in the male sex partner.50

Toxic shock syndrome (TSS) is associated with a strain of *Staphylococcus aureus* that produces a unique epidermal toxin.51 The syndrome afflicts young menstruating women who regularly use vaginal tampons. A diffuse blanching, macular, scarlatiniform rash typically leads to palmpoplantar desquamation, which completely fades within three days of initial appearance. This rash can provide a diagnostic clue to early detection of this often fatal disease.52

References


