Nosocomial Sexually Transmitted Diseases

Sexually transmitted diseases (STD) are caused by the heterogeneous collection of organisms listed in the Table. They are grouped together, because in spite of their biological differences, sexual transmission is felt to play a significant role in the epidemiology of each. With the exception of spermatozoa (the etiologic agent of unwanted pregnancy), none is exclusively venereal; that is, none of these diseases is acquired only through coitus. The precise epidemiologic contribution of sexual contact varies tremendously among them. Vulvovaginal candidiasis, for example, is usually not sexually transmitted, while gonorrhea or chlamydial infection usually is.

In the community, sexual transmission is significant for several reasons. There is limited environmental survival of the pathogens. Thus fomites play a relatively small role. The failure of such agents to survive in the environment also reduces the contribution of environmental reservoirs to ongoing outbreaks. Indeed, an ongoing nosocomial STD problem has not been reported in the absence of direct person-to-person transmission. Terminal disinfection is unnecessary, and the spread of infection by contaminated food, equipment or materials obtained from central supply, or medications prepared in the pharmacy is highly unlikely.

Usually only certain anatomic sites are susceptible to infection in the normal host. For example, *N. gonorrhoeae* can directly infect only the urethra, endocervix, anorectum, oropharynx, or conjunctiva of the adult. Thus, gonorrhea is very unlikely to be contracted by shaking hands or sitting on the much-maligned toilet seat.¹

STD lesions containing large numbers of organisms are most commonly located around genital or mucous membranes, further restricting the possibility of transmission through casual contact. Contact between staff

and patients in a hospital setting, however, is often anything but casual. In almost no other circumstance will a patient's genitals or mucous membranes be manipulated by other than a sexual partner. In no other setting is someone other than a sexual partner likely to come into direct contact with the patient's genital discharge or lesions. Direct invasion of the patient's integumental integrity is common, and needlestick accidents invade the integrity of hospital personnel as well. For these reasons, risks of nosocomial acquisition by hospital personnel are with some exceptions greater than risk of acquisition by other patients.

Unlike many other nosocomial pathogens, the agents of STD are highly virulent and fully capable of infecting a normal host if only they can be delivered to the proper anatomic site.

STDs are often overlooked among hospital admissions. They are usually not the reason for admission and may represent a secondary diagnosis of far less immediate clinical significance than the condition bringing the patient to the hospital. Thus extensive contact with hospital staff may occur before the sexually transmitted infection is first noted.

Aside from any situation in which hospital staff might have direct contact with the patients' lesions or discharge, certain areas of the hospital are of particular concern. Sexually transmitted diseases frequently present in the newborn nursery, for neonates may contract infection in utero or during the birth process. Because neonates lack some host defenses effective against sexually transmitted agents, they may be at risk for more severe disease. Conditions in nurseries frequently make it difficult for staff to wash hands between infant contacts, fostering the spread of infection.² Because it is often hard to tell whether infections occurring in the nursery are acquired intrapartum or during hospitalization, the CDC National Nosocomial Infection Study has chosen to define all neonatal infections as nosocomial.³

Address reprint requests to Michael F. Rein, MD, Associate Professor of Medicine, Division of Infectious Diseases, University of Virginia School of Medicine, Charlottesville, VA 22908.

TABLE

SEXUALLY TRANSMITTED AGENTS

Viruses

Herpes simplex Cytomegalovirus

Molluscum contagiosum (Poxvirus) Wart virus (Papovavirus)

vvart virus (Papovaviri Enetoin-Barr

Epstein-Barr Hepatitis A and B ? Agent of AIDS

Bacteria

Neisseria gonorrhoeae Neisseria meningitidis Treponema pallidum Hemophilus ducreyi Shigellae Group B streptococci? Gardnerella vaginalis Listeria monocytogenes? Calymmatobacterium granulomatis

Spermatozoa

Campylobacter fetus

Mycoplasmas

Mycoplasma hominis Ureaplasma urealyticum

Chlamydia

Chlamydia trachomatis

Fungi

Candida species

Endoparasites

Entamoeba histolytica Trichomonas vaginalis Giardia lamblia

Ectoparasites

Phthirus pubis Sarcoptes scabiei

The clinical laboratory is another area of concern, because blood and discharges are processed here. Nosocomial transmission may also occur in the outpatient department, where the majority of STDs will be diagnosed.

Gonorrhea

Because *N. gonorrhoeae* can primarily infect only certain anatomic sites, the major risk to hospital personnel is conjunctivitis. Infected discharge can be inoculated into the eyes on fingers contaminated with a patient's genital discharge. The infection usually has an incubation period of three to five days, and manifests considerable conjunctival injection and purulent discharge. Infection with minimal inflammatory response, however, has also been reported. The disease is usually unilateral and should be treated with parenteral antibiotics and saline irrigation for at least five days. ⁵

Nosocomial gonorrhea in adults is preventable by careful hand washing and avoiding touching one's face during patient examination. Urethral, cervical, or rectal discharge is infectious, and patients with gonorrhea should be put on body discharge precautions until 24 hours following completion of appropriate treatment.⁶ The skin lesions of disseminated gonococcal infection pose no particular infectious risk, because the lesions rarely contain viable organisms.⁷ Initially, however, such patients should be placed on body discharge precautions because they frequently have asymptomatic genital infection as a primary focus.⁸

Pharyngeal infection in adults is usually asymptomatic and does not appear to pose a significant risk; unlike N.

meningitidis, N. gonorrhoeae is not apparently transmissible in respiratory secretions.

The newborn nursery is an area of particular concern for gonococcal infection. Even in the presence of silver nitrate prophylaxis, babies born to women with cervical gonococcal infection appear to have about a 2% chance of developing gonococcal ophthalmia neonatorum. The effectiveness of silver nitrate prophylaxis may be reduced if the agent is rapidly washed out of the eye with saline, which precipitates the silver ion as silver chloride. Infection is presumably acquired by intrapartum inoculation of the conjunctivae during passage through an infected birth canal and is consequently completely preventable if maternal, cervical gonorrhea is diagnosed during pregnancy and treated prior to delivery.

Silver nitrate prophylaxis may delay the appearance of gonococcal ophthalmia neonatorum. In one series, the mean incubation period was more than six days.9 Infection may therefore go on unsuspected for several days during which time the baby poses some risk to hospital personnel and to other infants in the nursery. Indeed, babies born to infected mothers may acquire other, asymptomatic gonococcal infections. Premature rupture of the fetal membranes can permit assent of gonococci into the amniotic fluid resulting in orogastric contamination of the baby. 11 Although initially asymptomatic, some of these infants develop disseminated gonococcal infection. Because of the possibility of asymptomatic neonatal infection, babies born to mothers subsequently shown to have cervical gonorrhea should be treated with penicillin.⁵ It is essential that postpartum information on maternal infection be transmitted promptly to personnel caring for newborns so that appropriate precautions can be taken.

In prepubescent girls, the entire vaginal epithelium is susceptible to gonococcal infection. Infected children develop not merely cervicitis or urethritis but gonococcal vulvovaginitis. This infection often manifests as a copius vaginal and accompanying rectal discharge which contains gonococci.

Infected children should be maintained on body discharge precautions until 24 hours after completion of therapy, although the risk of spread to other children appears very small.¹

CHLAMYDIA TRACHOMATIS INFECTION

Genital infection with *Chlamydia trachomatis* is probably more common than gonorrhea. Diagnosis of these infections is more difficult because the organism is an obligate intracellular parasite and can be recovered only using tissue culture techniques. ¹² Since this capability is not generally available, many chlamydial infections, particularly in women, go unsuspected. The clinical spectrum of disease caused by *C. trachomatis* is very similar to that caused by *N. gonorrhoeae* as are the modes of transmission. Applying discharge precautions to patients with genital discharges remains an appropriate isolation technique.

Intrapartum transmission to the neonate from maternal, cervical infection is very common. The organism has been isolated from 70% of infants born to infected moth-

ers.¹³ About half of the infected infants are colonized in the eye which gives rise to chlamydial ophthalmia neonatorum, clinically very similar to gonococcal disease.⁹ The infection has an incubation period of about eight days, and transmission in the nursery is a possibility. Unfortunately, silver nitrate prophylaxis, highly effective in reducing the incidence of gonococcal conjunctivitis, is ineffective in preventing similar chlamydial infection.¹⁴ Chlamydial conjunctivitis must therefore be considered even in infants who have received silver nitrate prophylaxis. Erythromycin ophthalmic ointment is effective,¹⁴ but its value in preventing gonococcal eye disease is uncertain. Perhaps future recommendations will involve some combination of these two prophylactic methods.

About half of the infected infants are colonized in the nasopharynx.¹³ Such colonization may result in pneumonia which, however, usually presents after the fourth week of life and may not readily be recognized as a nosocomial infection acquired during delivery. 15 About half of these infants will have an accompanying chlamydial conjunctivitis, and body discharge precautions seems an appropriate manner of isolation until therapy has been completed. There is no convincing evidence of person-to-person spread of chlamydial pneumonia by droplet nuclei. Cases in which siblings or hospital roommates of an infected child have developed disease are not documented. The long incubation period for chlamydial pneumonia and uncertainty regarding its diagnosis may have interfered with our ability to detect such spread, but there are no convincing indications for strict respiratory isolation of infants with such infections.

Women with cervical chlamydial infection who undergo vaginal delivery may develop a postpartum endometritis 48 hours to six weeks later. ¹⁶ This syndrome produces several problems for the nosocomial infection surveillance system. Its late onset may prevent its being recognized, and its etiology and significance are certainly different from other postpartum infections. Intrapartum fever was observed in 9% of women with chlamydial infection and might affect reported nosocomial infection rates in areas of high chlamydial prevalence. ¹⁶ Neither complication was associated with Cesarean section.

HERPES SIMPLEX VIRUS GENITAL INFECTIONS

Lesions of recurrent genital herpes are usually covered by several layers of clothing unlike orolabial herpes in which active lesions are exposed. There is no information strongly suggesting that genital herpes among hospital personnel represents an infectious risk for patients.¹⁷ It would perhaps be reasonable, however, to exclude staff members with active genital herpes, if they can be identified, from caring for certain high-risk groups of patients including immunocompromised hosts and neonates. Strict observance of good hand washing technique should be advised.

Patients with active genital herpes probably represent a greater risk because the patient's genitalia may be uncovered for examination or treatment and may be touched by medical staff. Herpetic whitlow with type II herpes simplex virus has been described in hospital personnel. Body discharge precautions should be applied

to patients with active genital herpes. Although transmission of genital herpes infection by toilet seats must be extremely uncommon (if it were common, then genital herpes would be a frequent occurrence in sexually inactive people), it is probably reasonable to provide infected patients with private bathroom facilities. Certainly such patients should not share bathroom facilities with immunocompromised, hosts.

Hospitalized patients given immunosuppressive therapy may suffer a reactivation of genital infection with herpes simplex virus. Such patients may develop disseminated infection as well. 19,20

The most important nosocomial problem with genital herpes, as with most other sexually transmitted diseases, results from the possibility of intrapartum transmission to the neonate from a maternal, genital focus. Crude estimates suggest that perhaps half of the babies born to mothers with active genital infection become infected and that without effective treatment, at least half of these infections are fatal.²¹ Unfortunately, 70% of women delivering babies with neonatal infection are without signs and symptoms of active disease at the time of delivery. 22 Pregnant women with a history of genital herpetic infection should be cultured near term. If virus is isolated, serious consideration should be given to delivery by Cesarean section. If, however, the fetal membranes have been ruptured for more than four hours, virus must be assumed to have ascended into the amniotic cavity, and Cesarean section will not prevent neonatal infection. 21,23

Neonatal herpes usually appears between two and 12 days of life and is first manifest by vesicles in 50% of patients. The disease often progresses to visceral and central nervous system involvement.²²

Kibrick has formulated detailed and logical recommendations for management of infected mothers and their newborns.²⁴ These recommendations consider the possibility of postpartum transmission from mother to baby and the significant risk of transmission from an infected baby to others in the nursery. In brief, it is recommended that infected women have private rooms, that body discharge precautions be employed, and that postpartum contact with the infant be supervised. Bed linens and hospital gowns should be assumed to be infected. Before handling her infant, the mother should get out of bed and thoroughly wash her hands. She should put on a clean gown and be seated in a chair. Unsupervised rooming-in should be avoided. It makes little sense to subject a mother with active disease to Cesarean section, only to permit her to infect her infant after delivery.

Exposed neonates should be housed in a special care or isolation unit. Strict isolation is probably advisable, for although there is no direct evidence for droplet transmission of herpes simplex infection, such isolation serves to remind nursery personnel that special precautions must be taken. There is no need to delay the discharge of aymptomatic infants from the nursery, and indeed, early discharge may actually prevent nosocomial spread of the infection in the nursery. Readmission of symptomatic infants usually results in prompt isolation and may actually provide less risk to other infants than does the cryp-

tically infected neonate who first develops overt disease while in the nursery.

SYPHILIS

The syphilitic chancre is a painless, indurated ulcer which appears at the point of initial inoculation of spirochetes. Such lesions usually, therefore, occur about the genitalia, but they are increasingly recognized around the mouth, anus, or other areas. Because they are painless, chancres may go unnoticed by the patient. Patients with chancres should be treated with body discharge precautions, because the lesions contain large numbers of spirochetes and are contagious through direct contact.

In secondary syphilis, the spirochetes have widely disseminated, and the disease manifests as a generalized rash often involving the palms and soles. The dry skin lesions, usually maculopapular, are noncontagious. Darkfield examination following vigorous abrasion of these lesions only rarely reveals motile spirochetes. This is fortunate, since were they to contain large numbers of organisms, such lesions might be highly contagious to casual contacts of infected patients, and nonvenereal syphilis would be more the rule than the very rare exception.

In moist body sites potentially contagious lesions do develop. The oral, vaginal, and anal mucosae may show mucous patches. These are painless, shallow ulcerations which contain large numbers of spirochetes. In earlier times, when syphilis was a more common disease, barehanded dentists might develop chancres of the finger after coming in contact with patients' oral lesions. Similar nosocomial transmission could occur today during uncautious physical examination. In areas where skin is oppressed to skin (eg. perivaginally, perianally, even between the toes), patients with secondary syphilis may develop condylomata lata. These are flat, wart-like, hypertrophic lesions which also contain large numbers of spirochetes and may therefore transmit infection.

Risks to hospital staff exist because the spirochete can penetrate not only intact mucous membranes but through microscopic breaks in skin as well. Twenty-four hours after initiating treatment with penicillin, however, spirochetes have disappeared from moist lesions, and the risk of infection is over. Patients with latent or late syphilis do not have surface lesions containing spirochetes and present no infectious risk.

At all stages of syphilis, spirochetes may be found in some lymph nodes and sometimes in other tissues. Therefore syphilis could theoretically be contracted by a surgeon who knicks himself during a procedure. Such cases must be extraordinarily rare, but the risk can be avoided if elective surgery is postponed until syphilitic infection has been treated. In experimental animals, syphilis can be shown to suppress certain elements of the immune response, ²⁵ which could conceivably increase the risk of postoperative or other nosocomial infections, but such an association has not been described. Older observations suggest that patients with syphilis have delayed wound healing, ²⁶ perhaps an additional argument for treatment of syphilis prior to elective surgery.

Syphilotherapy may induce a transient febrile response known as the Jarisch-Herxheimer reaction, and treatment following surgery might result in a fever spike that could be confused with postoperative infection.

Spirochetemia may precede the development of the primary chancre or serologic reactivity and frequently occurs during primary, secondary, and early latent syphilis. Such spirochetemias are transient, and most syphilitics have spirochetes in the blood for only short periods of time. Spirochetemia is extremely rare in syphilis of greater than two years duration. Blood containing spirochetes can, however, transmit the infection, and transfusion syphilis was at one time a potential complication for blood recipients. 26 Nontreponemal serologic tests for syphilis are performed on blood donated to the American Red Cross, but such testing is no longer required by the American Association of Blood Banks. Indeed, given modern blood storage technology, the risk of transfusion syphilis is vanishingly small. Virulent spirochetes have not been recovered from blood stored at 5° C for 72 hours,²⁷ and thus routinely stored blood should be risk-free. In some pediatric surgical cases involving extracorporeal circulation, the pump is primed with blood less than 48 hours old. In this situation, transmission of syphilis could conceivably occur, but the blood would have to have been donated by a patient with very recently acquired syphilis who had a spirochetemia in the absence of a positive serologic test for syphilis.

Patients acquiring transfusion syphilis have spirochetes inoculated directly into the blood stream. They therefore do not develop a chancre, which results from local multiplication of spirochetes at the points of inoculation. Rather, these patients may first present with disease resembling secondary syphilis, or their infections may remain asymptomatic for long periods of time.

Needlestick injuries by personnel caring for patients with positive serologic tests for syphilis are a source of concern. The risk of infection in this setting is extremely small. Most patients with positive serologic tests for syphilis are not experiencing spirochetemia. Investigation of such injury cases should include obtaining a history of past or present syphilitic infection from the patient. Serologic tests for syphilis remain positive for various intervals following successful treatment of the disease. A patient who had been successfully treated for syphilis presents no significant risk. Further, many patients acutely ill with a variety of inflammatory or infectious diseases may have a false positive nontreponemal test for syphilis (eg, VDRL, RPR, ART). Syphilis is not a serious consideration if the confirmatory treponemal tests (eg, FTA-ABS, MHA-TP) are not reactive. In the absence of clinically active syphilis in the patient, one might best merely follow staff receiving needlestick injuries. A serologic test for syphilis might be performed immediately and repeated in three months. In the interval, the staff member should be cautioned to report immediately the development of a sore at the point of inoculation or enlargement of regional lymph nodes.

Sometimes a staff member suffering a needlestick injury is concerned to the point of obsession about the consequences. In this situation, it may be best to administer 2.4 million units of benzathine penicillin G intramuscularly and thereby assure the victim that neither clinical nor serologic manifestations of syphilis will develop. Such prophylactic treatment is relatively low risk and very inexpensive (compared, for example, to the cost of Hepatitis B Immune Globulin which may also have to be administered). Such treatment obviates the need for serologic followup, insures persistent seronegativity, prevents subsequent transmission of disease, and serves to reduce emotional stress.

Babies may acquire syphilis in utero by transplacental transmission of T. pallidum. Infected infants are asymptomatic at birth or may display a variety of lesions. Several of these lesions strongly suggest a diagnosis of congenital syphilis and also put nursery personnel at risk because they contain large numbers of spirochetes. Vesicular skin lesions rupture to form shallow ulcers which contain organisms. Shallow ulcers of the mucous membranes, called mucous patches, are painless and also contain large numbers of organisms. Likewise, snuffles, chronic mucopurulent nasal discharge, must be considered highly contagious. Condylomata lata develop in congenital syphilis as they do in acquired secondary syphilis, in moist areas. These wart-like lesions also contain large numbers of organisms. Babies with congenital syphilis should be placed on body discharge precautions until 24 to 48 hours after initiation of therapy. There is no need to isolate babies from mothers who are also on antisyphilitic therapy because they are thereby protected from cross infection.

TRICHOMONIASIS

Unlike the pathogens already discussed, Trichomonas vaginalis can infect only the urogenital tract. Thus accidental infection of hospital personnel is extremely unlikely. T. vaginalis can, however, survive for short periods in a moist environment.^{28,29} Thus viable organisms have been recovered after 90 minutes on a wet sponge or up to three hours in urine. Trichomonads have also been recovered from 1% of bath tubs and 13% of toilet seats after use by infected women. That such survival ever results in transmission is not documented. Early studies suggest that an inoculum of less than 104 trichomonads will initiate experimental infection in some women,³⁰ but it is not clear that an inoculum sufficiently large to cause infection will survive exposure in hospital bathrooms. It must be remembered that sitting on a trichomonad will not produce infection; the organisms must be inoculated directly into the urogenital tract. Under no circumstances should patients share wash cloths or towels, because trichomonads can survive up to 24 hours on moist towels, and such transmission may account for outbreaks of the infection in institutionalized populations.³¹ Although the risk of transmission from patient to patient is extremely small, it seems reasonable to place patients with active trichomoniasis on body discharge precautions until 24 hours after initiation of treatment.

Up to 5% of babies born to infected mothers develop neonatal trichomoniasis.³² The infection is asymptomatic and self limited in many babies, but some will develop persistent infection or symptomatic vaginitis which requires treatment. Until treated, such babies should be isolated in the nursery, and strict handwashing technique should be observed.

GENITAL WARTS

There is no strong epidemiological association between genital warts and warts on other body surfaces in the same patient. There are, however, some cases in which genital warts and warts elsewhere have coexisted.³³ The risk to hospital staff of caring for patients with genital warts must be extremely small. Babies born to mothers with genital warts have, however, been noted to develop laryngeal papillomata, and some workers, believing that the papilloma virus may be acquired during the birth process, have suggested that extensive vaginal venereal warts may be an indication for delivery by Cesarean section.³⁴ Babies born to mothers with venereal warts need not be subject to special isolation in the nursery, nor need one take special precautions concerning rooming-in.

ECTOPARASITES

Several nosocomial outbreaks of scabies have been reported. The disease is spread by close contact, and infection has been reported in nurses and attendants caring for infected patients. 35,36 So called Norwegian scabies, a variant characterized by large numbers of mites, is particularly contagious, and infection was documented in 30 of 86 hospital employees having direct contact with an unsuspected case. Skin-to-skin contact, particularly that likely to be associated with lifting or turning the patient, may cause infection. Incubation periods in hospital personnel ranged from 7 to 27 days but was usually about two weeks. Transmission by inanimate objects seems very unlikely. Patients with scabies should be put on wound and skin precautions until 24 hours after the initiation of treatment with lindane.

Far less information is available concerning nosocomial transmission of the crab louse, *Phthirus pubis*. Unlike the body louse, the pubic louse can survive for no more than 48 hours away from a human host. It is conceivable that transmission by toilet seats or by shared towels might occur in a hospital setting, but reports are lacking.⁸⁷ Pubic lice are occasionally found in the eyebrows and in the axillary hair. Effective treatment consists of brief shampooing of the affected areas with lindane, after which the patient is no longer contagious. Thus, recognition of the infection might better be dealt with by rapid treatment than by instituting isolation procedures.

OTHER CONSIDERATIONS

Patients in the hospital may have sexually transmitted infection spread from one anatomic site to another. One should not perform a rectal exam using the same glove that has been inserted in the vagina. The practice is based on the hypothesis that the microbial flora of the vagina and the rectum are essentially the same. This is not necessarily the case in patients with sexually transmitted infections. Gonococci, chlamydia, or herpes simplex virus might be transmitted from the cervix or vagina to the rectum during such an examination. If a rectal examina-

tion is to be performed after a digital vaginal examination, gloves should be changed.

Sexual transmission plays an important role in the epidemiology of several other infections such as hepatitis A, hepatitis B, and the acquired immunodeficiency syndrome (AIDS). Risks of nosocomial transmission for these infections are generally carefully considered.

REFERENCES

- Rein MF: Nonsexual acquisition of genital gonococcal infection. N Engl J Med 1979; 301:1347.
- Brachman PS: Inroduction, in Bennett JV, Brachman PS (eds): Hospital Infections. Boston, Little Brown & Co, 1979, p 5.
- Allen JR, Oliver TK Jr: The newborn nursery, in Bennett JV, Brachman PS (eds): Hospital Infections. Boston, Little Brown Co, 1979.
- 4. Podgore JK, Holmes KK: Ocular gonococcal infection with minimal or no inflammatory response. *JAMA* 1981; 246:242-243.
- Centers for Disease Control: Sexually transmitted diseases treatment guidelines, 1982. Morbidity and Mortality Weekly Report 1982; 31(2S):335-605.
- Wenzel RP, Osterman CA, Mandell GL: University of Virginia Medical Center—Isolation procedure and infection control manual, in Wenzel RP (ed): Handbook of Hospital Acquired Infections. Boca Raton, CRC Press Inc, 1981, pp 127-211.
 Tronca E, Handsfield HH, Wiesner PJ, et al: Demonstration of
- Tronca E, Handsfield HH, Wiesner PJ, et al: Demonstration of Neisseria gonorrhoeae with fluorescent antibody in patients with disseminated gonococcal infection. J Infect Dis 1974; 129:583-586.
- Holmes KK, Counts GW, Beaty HN: Disseminated gonococcal infection. Ann Intern Med 1971; 74:979-993.
- Armstrong JH, Zacarias F, Rein MF: Ophthalmia neonatorum: A chart review. *Pediatrics* 1976; 57:884-892.
- Barson PC: Specific prophylaxis of gonorrheal ophthalmia neonatorum: A review. N Engl J Med 1966; 274:731-734.
- Handsfield HH, Hodson WA, Holmes KK: Neonatal gonococcal infection I. Orogastric contamination with Neisseria gonorrhoeae. JAMA 1973; 225:697-701.
- Schachter J: Chlamydial infections. N Engl J Med 1978; 298:428-549.
- 13. Schachter J, Hold J, Goodner E, et al: Prospective study of chlamydial infection in neonates. *Lancet* 1979; 2:377-379.
- Hammerschlag MR, Chandler JW, Alexander ER, et al: Erythromycin ointment for ocular prophylaxis of neonatal chlamydial infection. JAMA 1980; 244:2291-2293.
- Harrison HR, English MG, Lee CK, et al: Chlamydia trachomatis infant pneumonitis: Comparison with matched controls and other infant pneumonias. N Engl J Med 1978; 298:702-708.
- Wager GP, Martin DH, Koutsky L, et al: Puerpural infectious morbidity: Relationship to route of delivery and to antepartum Chlamydia trachomatis infection. Am J Obstet Gynecol 1980; 138:1028-1033.
- 17. Kaslow RA, Garner JS: Hospital personnel, in Bennett JV, Brachman PS (eds): *Hospital Infections*. Boston, Little Brown & Co, 1979, p 37.

- 18. Nahmias AJ, Roizman B: Infection with herpes-simplex viruses 1 and 2. N Engl J Med 1973; 289:667-789.
- Stone WJ, Scowden EB, Spannuth CL, et al: Atypical herpesvirus hominis type 2 infection in uremic patients receiving immunosuppressive therapy. Am J Med 1977; 63:511-516.
- Linnemann CC, First MR, Avira MM, et al: Herpesvirus hominis type 2 meningoencephalitis following renal transplantation. Am J Med 1976; 61:703-708.
- 21. Nahmias AJ, Josey WE, Naib ZM, et al: Perinatal risk associated with maternal genital herpes simplex virus infection. *Am J Obstet Gynecol* 1971; 110:824-834.
- 22. Whitely RJ, Nahmias AJ, Visintine AM, et al: The natural history of herpes simplex virus infection of mother and newborn. *Pediatrics* 1980; 66:489-494.
- Light IJ, Linnemann CC: Neonatal herpes simplex infection following delivery by cesarean section. Obstet Gynecol 1974; 44:496-499.
- 24. Kibrick S: Herpes simplex infection at term: What to do with mother, newborn, and nursery personnel. *JAMA* 1980; 243:157-160.
- Bey RF, Johnson RC, Fitzgerald TJ: Suppression of lymphocyte response to concanavallin A by mucopolysaccharide material from Treponema pallidum—Infected rabbits. Infect Immun 1979; 26:64-69.
- Stokes JH, Beerman H, Ingraham NR: Modern Clinical Syphilology, ed 3. Philadelphia, WB Saunders Co, 1944, pp 470-474.
- Willcox RR, Guthe T: Treponema pallidum: A bibliographical review of the morphology, culture and survival of T. pallidum and associated organisms. Bull WHO 1966; 35(suppl):86-87.
- 28. Jirovec O, Petru M: Trichomonas vaginalis and trichomoniasis. Adv Parasitol 1968; 6:117-188.
- Gallai Z, Sylvester L: The present status of urogenital trichomoniasis: A general review of the literature. Applied Therapeutics 1966; 8:773-778.
- 30. Hesseltine HC, Wolters SC, Campbell A: Experimental human vaginal trichomoniasis. *J Infect Dis* 1942; 71:127-130.
- 31. Burch TA, Rees CW, Reardon LV: Epidemiologic studies on human trichomoniasis. *Am J Trop Med Hyg* 1959; 8:312-318.
- 32. Al-Salihi FL, Curran JP, Wang J-S: Neonatal *Trichomonas vaginalis:* Report of 3 cases and review of the literature. *Pediatrics* 1974; 53:196-200.
- 33. Oriel JD: Genital warts. Sex Transm Dis 1981; 8:326-329.
- Quick CA, Watts SC, Krzyzek RA, et al: Relationship between condylomata and laryngeal papillomata (clinical and molecular virological evidence). *Annals of Otology* 1980; 89:467-871.
- 35. Belle EA, D'Souza TJ, Zarzour JY, et al: Hospital epidemic of scabies: Diagnosis and control. Can J Public Health 1979; 70:133-135.
- Gooch JJ, Strasius SR, Beamer B, et al: Nosocomial outbreak of scabies. Arch Dermatol 1978; 114:897-898.
- 37. Minster J: Nursing management of patients with scabies and lice. Nurs Clin North Am 1980; 15:741-756.

Michael F. Rein, MD

Associate Professor of Medicine Division of Infectious Diseases University of Virginia School of Medicine Charlottesville, Virginia