VIRGINIA
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C.M.G. Buttery, M.D., M.P.H., Commissioner
Grayson B. Miller, Jr., M.D., Epidemiologist
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Lyme Disease in Virginia—1988

The Disease

Lyme disease is a multisystem disease with distinct acute and chronic clinical syndromes. It is caused by injection of a spirochete, *Borrelia burgdorferi,* into a human or animal host by an arthropod vector. The disease manifests early with flu-like symptoms (headache, myalgia, nausea, lymphadenopathy) and erythema chronicum migrans (ECM), a distinctive circular red rash with central clearing. These symptoms will appear in about two-thirds of patients, from several days to several weeks after receiving a bite from an infected tick.

Later symptoms of Lyme disease involve the joints, central and peripheral nervous systems, and the heart. Joint involvement may consist of arthritis or arthralgia, with the knee joint being most commonly affected. Neurological changes include meningitis, encephalitis, Bell's palsy and peripheral neuropathis. Cardiac involvement manifests as atrioventricular conduction defects, myocarditis or left ventricular dysfunction (seen clinically as palpitations and dyspnea). These later syndromes usually appear from two weeks to six months after infection. Arthritic changes occur commonly, heart and brain changes less frequently.

The "deer tick," *Ixodes dammini,* is the most common arthropod vector of *Borrelia burgdorferi,* the etiologic agent of Lyme disease. *I. dammini* consists of three morphological types: the larva, the nymph and the adult. All forms are very small, ranging in size from a typewritten comma to the size of a pinhead, respectively. The tick takes a blood meal three times during its life. During these feedings, the tick may pick up *B. burgdorferi* from an infected host, and then harbor the spirochete until its next feeding, at which time the new host may become infected. Feeding takes place primarily in the late spring or summer, but may occur at any time, especially in warmer climates. The larva is not infective because it has had no exposure to the infective agent (no transovarial transmission takes place). The disease is transmitted by the nymph and adult forms. Despite its name, the "deer tick" will feed on a variety of hosts, including small rodents, dogs, cats and humans. Although other tick species, including *Amblyomma americanum, I. scapularis* and *Dermacentor variabilis,* as well as mosquitos and biting flies, have been found to occasionally carry the infective spirochete, they have not

Continued to page 2

Inside

Continued from page 1
been proven to be capable of transmitting the spirochete between mammalian hosts.

Lyme disease has been reported in 43 states, including Virginia, but the majority of cases originate in the northeastern states, Minnesota, Wisconsin, and California. Only twenty-one of 4,572 cases reported in the U.S. with onset in 1988 involved Virginia residents. One of the Virginia cases probably contracted the disease in an endemic area of New York state. The low incidence of the disease in Virginia may be attributed to a sparse population of *Ixodes dammini*. Eighteen counties and two independent cities in Virginia have been classified as endemic, with most of these localities situated in coastal and central Virginia (Figure 1). Several isolated endemic pockets also exist throughout the state. Nine localities reported Lyme disease in 1988. Isle of Wight County and the City of Virginia Beach became newly endemic in 1988. A locality is defined as endemic if a definite case has been documented or if *I. dammini* ticks are found in the area.

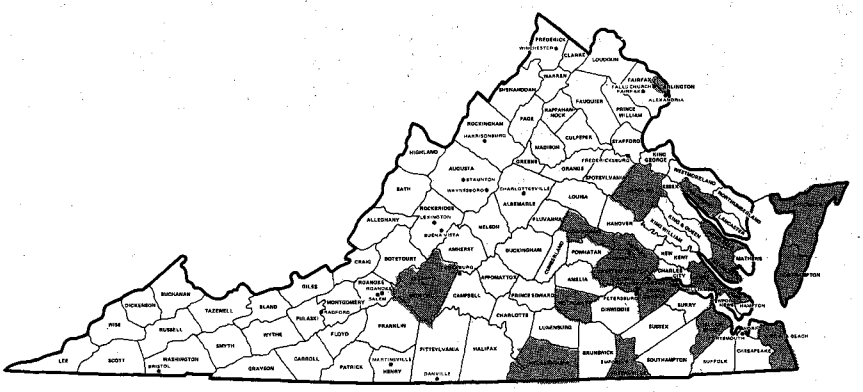
Case Definition

For surveillance purposes in Virginia, the CDC case definition was used. A definite case was defined as ECM with potential tick exposure in an endemic county or city, or, in the absence of ECM, involvement of at least one of the three organ systems and either a positive serologic test for Lyme disease (>256 by IFA method or optical density ratio ≥0.2 by ELISA) or isolation of *Borrelia burgdorferi* from a clinical specimen. In a non-endemic area, definition of a definite case required ECM with involvement of at least two of the three organ systems, or ECM and a positive serologic test for Lyme disease, or isolation of *B. burgdorferi* from a clinical specimen. A positive serologic test for Lyme disease was not considered diagnostic without the appropriate accompanying clinical presentation.

Virginia Cases

From physician, laboratory, and hospital reports, 21 definite Lyme disease cases were identified in Virginia with onset in 1988. This group consisted of 11 females (52%) and 10 males (48%). Ages ranged from less than 1 year to 69 years.

FIGURE 1
Lyme Disease Endemic Counties
As of July 15, 1989



The mean age was 29 years, with a standard deviation of 18 years.

The greatest number of cases were reported in Accomack County (6), followed by Chesterfield and York Counties and Newport News (3 each), and finally Northampton, Isle of Wight and James City Counties and Virginia Beach (1 each). One case was contracted in Long Island, NY. Onset dates were reported in every month from April through November, with 16 (76%) of 21 cases having an onset date between May and August.
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ECM was reported in 20 (95%) of the cases. Arthritis and/or arthralgia occurred in 4 cases (19%), neurologic symptoms in 3 cases (14%) and cardiac symptoms in 2 cases (10%). There were 5 cases with delayed symptoms in the three organ systems. Three of those cases had both arthritic and neurologic changes. Other flu-like symptoms were reported in 7 cases (33%).

The history of a definite tick bite existed in 14 (67%) out of 21 cases, although the ticks were not speciated. Tick exposure was possible in all of the remaining cases; i.e. the patient had recently been in an area which could support ticks. Nineteen cases were reported from previously endemic areas, and two cases from non-endemic areas, thus establishing two new endemic localities (Isle of Wight county and Virginia Beach).

Blood was submitted for serology in 16 cases, of which nine (56%) were positive for antibody to *B. burgdorferi*. Convalescent serology was obtained on two patients and showed a decreasing titer. Antibiotics were administered to 71% of the cases.

The onset months of April through November probably reflect the life cycle of the tick. The nymphs feed in the early spring to summer and the adults in the fall. Early constitutional signs and ECM typically follow shortly afterwards. Occasionally, the early manifestations will go unnoticed, and the disease will present months later, without apparent connections to the tick's life cycle. This did not occur in any of the 1988 cases. Most cases (66%) occurred in May, June and July, which may indicate that more cases are contracted from nymphs than adult ticks. This theory is supported by evidence that the adult tick strongly prefers the white-tailed deer as its final host, while the nymph will feed on any number of mammalian species.

The percentage of cases reporting classic ECM lesions in Virginia (95%) is higher than the national average (70%). This is biased by our case definition, which uses ECM as a major criterion for classifying Lyme disease cases. Without ECM, substantial additional evidence of the disease is necessary for inclusion as a definite case for epidemiologic purposes.

Diagnosis

Serologic results are not always reliable for diagnosing recent infection. Antibodies to *B. burgdorferi*

develop between six weeks and six months after infection, therefore many samples obtained at the onset of ECM are negative. Early treatment with antibiotics also reduces the likelihood of measurable antibody production. In areas of high endemnicity for Lyme disease many people may have titers that reflect past infection unrelated to any current symptoms. Laboratories also vary in their definition of a "positive" titer. The Division of Consolidated Laboratory Services uses the following criteria to interpret serology: IFA titers of 1:64 to 1:128 are considered borderline, titers of 1:256 or greater are considered indicative of infection. The CDC uses the ELISA test and considers an optical density ratio of ≥ 0.2 to be positive. Because serologic results can be unreliable, clinical signs should be used to determine whether or not to administer therapy.

The history of tick exposure may be very important to the diagnosis of Lyme disease. Several cases reported no tick bite, but all had exposure to a wooded or potentially tick-infested area. On the other hand, not all tick bites lead to Lyme disease. The *Ixodes dammini* tick appears to be scarce in most of Virginia, although it has been identified in Accomack, Northampton, Caroline, and Nottoway counties. The vector in its nymph form is also so small that it is difficult to see, and its bite is often painless. Many people reporting a tick bite will have been bitten by a tick which is more easily detected than that containing the infective spirochete. Furthermore, identification of the specific tick is not possible for the layman, but requires the services of a trained entomologist.

Treatment

Current recommendations for the treatment of Lyme disease were recently reviewed and are summarized here.[1] These recommendations are based on limited data and should be considered tentative. Doxycycline 100 mg bid × 10–21 days or amoxicillin 250–500 mg tid (pediatric dose is 20–40 mg/kg/day) × 10–21 days is recommended for early disease. Tetracyclines (including doxycycline) should not be prescribed for children less than eight years of age or for pregnant or lactating women.

Mild neurologic disease, cardiac disease, or arthritis is treated with doxycycline 100 mg bid × 1 month (21 days for cardiac disease) or amoxicillin 250–500 mg (500 mg for arthritis—some clinicians also add probenecid) tid × 1 month (21 days for cardiac disease).

More serious manifestations are generally treated with parenteral antibiotics including ceftriaxone 2 gm/day IV × 10–21 days or penicilin G 20–24 million units/day IV × 10–21 days.

For pediatric dosages and a complete listing of alternative regimens, please consult the *Medical Letter* review.[1]

Prevention

The best defense against Lyme disease is to wear protective clothing when entering tick-infested areas, and to treat skin with a DEET (N,N-diethyl-m-toluamide)-containing product and clothing with a permethrin-based spray (e.g. **Permanone***). Frequent inspection and tick removal will reduce risk of ticks transmitting organisms. Removal of ticks from the family pet is recommended to prevent the disease in the animal (however, animals are not likely to carry the disease to their owners because the tick has already taken its yearly meal by the time the animal brings it home).

While it is important to exercise these precautions, the likelihood of contracting the disease is low enough that people may continue to enjoy the outdoors without fear. Awareness of the disease and practice of the above precautions should be sufficient for protection against this infection.

Submitted by Laura Bogert, Senior Veterinary Student on elective with the Virginia Department of Health.

**Trade names are used for identification only and do not imply endorsement by the Virginia Department of Health.*

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Hand-Foot-and-Mouth Disease

Hand-foot-and-mouth disease is a selflimited viral syndrome most commonly caused by Coxsackie A16. Clinical expression of the syndrome decreases with age. Virtually 100% of small children will be symptomatic, while only about 11% of adults develop the characteristic clinical syndrome. Enterovirus 71 has been identified as the responsible agent for occasional outbreaks of hand-foot-and-mouth disease. This virus can produce a more severe clinical syndrome including aspectic meningitis, encephalitis and paralytic disease.

Symptoms of hand-foot-and-mouth disease include diffuse oral lesions which may involve the buccal mucosa, tongue and gums. The intraoral lesions are usually ulcerative, and may be painful, interfering with eating. Lesions range in size from 4–8 millimeters. Papulovesicular lesions also occur as an exanthem, most commonly on the palms, soles and occasionally the buttocks. Lesions clear by absorption of fluid in a week to 10 days.

Occurrence is worldwide. The greatest incidence for this disease occurs in summer and early fall. The disease occurs frequently as outbreaks among groups of children.

This illness is transmitted via the fecal-oral route and by direct contact with the oral secretions of an infected individual. The incubation period is 3–5 days. The period of communicability is during the acute phase and beyond, since the virus can be shed in the stool for several weeks after infection has occurred.

The practice of good hygiene is the best precaution for preventing transmission. Other precautions that may reduce the spread of this virus in group care settings, such as day care, is to use a dilute (1:10) bleach solution to wipe down surfaces in toilets and diaper changing areas where fecal contamination might be present. Cohorting of symptomatic individuals in certain situations, where practical, might be considered. There is not a public health reason for excluding symptomatic children from school, day care or other group settings. Occasionally, severity of symptoms may require that a child be kept at home. The child's physician is best qualified to make that decision.

Recommendations of the Immunization Practices Advisory Committee of the U.S. Public Health Service

Pneumococcal Polysaccharide Vaccine

These recommendations update the last statement by the Immunization Practices Advisory Committee (ACIP) on pneumococcal polysaccharide vaccine and include new information regarding 1) vaccine efficacy, 2) use in persons with human immunodeficiency virus (HIV) infection and in other groups at increased risk of pneumococcal disease, and 3) guidelines for revaccination.

Introduction

Disease caused by *Streptococcus pneumoniae* (pneumococcus) remains an important cause of morbidity and mortality in the United States, particularly in the very young, the elderly, and persons with certain high-risk conditions. Pneumococcal pneumonia accounts for 10%–25% of all pneumonias and an estimated 40,000 deaths annually (*1*). Although no recent data from the United States exist, in the United Kingdom pneumococcal infections may account for 34% of pneumonias in adults who require hospitalization (*2*). The best estimates of the incidence of serious pneumococcal disease in the United States are based on surveys and community-based studies of pneumococcal bacteremia. Recent studies suggest annual rates of bacteremia of 15-19/100,000 for all persons, 50/100,000 for persons ≥65 years old, and 160/100,000 for children ≤2 years old (*3,4*). These rates are 2–3 times those previously documented in the United States. The overall rate for pneumococcal bacteremia in some Native American populations can be six times the rate of the general population (*5*). The incidence of pneumococcal pneumonia can be 3–5 times that of the detected rates of bacteremia. The estimated incidence of pneumococcal meningitis is 1-2/100,000 persons.

Mortality from pneumococcal disease is highest in patients with bacteremia or meningitis, patients with underlying medical conditions, and older persons. In some high-risk patients, mortality has been reported to be >40% for bacteremic disease and 55% for meningitis, despite appropriate antimicrobial therapy. Over 90% of pneumococci remain very sensitive to penicillin.

In addition to the very young and persons ≥65 years old, patients with certain chronic conditions are at increased risk of developing pneumococcal infection and severe pneumococcal illness. Patients with chronic cardiovascular diseases, chronic pulmonary disease, diabetes mellitus, alcoholism, and cirrhosis are generally immunocompetent but have increased risk. Other patients at greater risk because of decreased responsiveness to polysaccharide antigens or more rapid decline in serum antibody include those with functional or anatomic asplenia (e.g., sickle cell disease or splenectomy), Hodgkin's disease, lymphoma, multiple myeloma, chronic renal failure, nephrotic syndrome, and organ transplantation. In a recent population-based study, all persons 55–64 years old with pneumococcal bacteremia had at least one of these chronic conditions (*4*). Studies indicate that patients with acquired immunodeficiency syndrome (AIDS) are also at increased risk of pneumococcal disease, with an annual attack rate of pneumococcal pneumonia as high as 17.9/1000 (*6/8*). This observation is consistent with the B-cell dysfunction noted in patients with AIDS (*9,10*). Recurrent pneumococcal meningitis may occur in patients with cerebrospinal fluid leakage complicating skull fractures or neurologic procedures.

Pneumococcal Polysaccharide Vaccine

The current pneumococcal vaccine (Pneumovax® 23, Merck Sharp & Dohme, and Pnu-Imune® 23, Lederle Laboratories) is composed of purified capsular polysaccharide antigens of 23 types of *S. pneumoniae* (Danish types 1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19F, 19A, 20, 22F, 23F, 33F). It was licensed in the United States in 1983, replacing a 14-valent vaccine licensed in 1977. Each vaccine dose (0.5 mL) contains 25 μg of each polysaccharide antigen. The 23 capsular types in the vaccine cause 88% of the bacteremic pneumococcal disease in the United States. In addition, studies of the human antibody response indicate that cross-reactivity occurs for several types (e.g., 6A and 6B) that cause an additional 8% of bacteremic disease (*11*).

Most healthy adults, including the elderly, show a twofold or greater rise in type-specific antibody, as measured by radioimmunoassay, with 2–3 weeks of vaccination. Similar antibody responses have been reported in patients with alcoholic cirrhosis and diabetes mellitus requiring insulin. In immunocompromised patients, the response to vaccination may be less. In children <2 years old, antibody response to most capsular types is generally poor. In addition, response to some important pediatric pneumococcal types (e.g., 6A and 14) is decreased in children <5 years old (*12,13*).

Following vaccination of healthy adults with polyvalent pneumococcal vaccine, antibody levels for most pneumococcal vaccine types remain elevated at least 5 years; in some

persons, they fall to prevaccination levels within 10 years (*14,15*). A more rapid decline in antibody levels may occur in children. In children who have undergone splenectomy following trauma and in those with sickle cell disease, antibody titers for some types can fall to prevaccination levels 3–5 years after vaccination (*16,17*). Similar rates of decline can occur in children with nephrotic syndrome (*18*).

Patients with AIDS have been shown to have an impaired antibody response to pneumococcal vaccine (*10,19*). However, asymptomatic HIV-infected men or those with persistent generalized lymphadenopathy respond to the 23-valent pneumococcal vaccine (*20*).

Special Request

Help us isolate the etiologic agent for human *Ehrlichia canis*.

If you suspect *E. canis* in a human patient do the following *before* administering antibiotic therapy:

1. Harvest 2 individual buffy coat samples (up to 1cc per vial if possible);
2. Freeze as rapidly as possible to −70°C or −90°C and after 2 days transfer to liquid nitrogen storage,
3. If patient shows a fourfold or greater rise in titer to *E. canis*, make stored buffy coats available to the Centers for Disease Control for isolation studies by calling Dr. Suzanne Jenkins (804) 786-6261 for further instructions.

Vaccine Efficacy

In the 1970s, pneumococcal vaccine was shown to reduce significantly the occurrence of pneumonia in young, healthy populations in South Africa and Papua New Guinea, where incidence of pneumonia is high (*21,22*). It was also demonstrated to protect against systemic pneumococcal infection in hyposplenic patients in the United States (*23*). Since then, studies have attempted to assess vaccine efficacy in other U.S. populations (*24–30;* CDC, unpublished data). A prospective, ongoing case-control study in Connecticut has shown an overall protective efficacy of 61% against pneumococcal bacteremia caused by vaccine- and vaccine-related serotypes. The protective efficacy was 60% for patients with alcoholism or chronic pulmonary, cardiac, or renal disease and 64% for patients ≥55 years old without other high-risk chronic conditions (*25,26*). In another multicenter case-control study, vaccine efficacy in immunocompetent persons ≥55 years old was 70% (*27*). A smaller case-control study of veterans failed to show efficacy in preventing pneumococcal bacteremia (*28*), but determination of the vaccination status was judged to be inadequate and the selection of controls was considered to be potentially biased.

Studies based on CDC's pneumococcal surveillance system suggest an efficacy of 60%–64% for vaccine-type strains in patients with bacteremic disease. For all persons ≥65 years of age (including persons with chronic heart disease, pulmonary disease, or diabetes mellitus), vaccine efficacy was 44%–61% (*29;* CDC, unpublished data). In addition, estimates of vaccine efficacy for serologically related types were 29%–66% (*29*). Limited data suggest that clinical efficacy may decline ≥6 years after vaccination (CDC, unpublished data).

A randomized, double-blind, placebo-controlled trial among high-risk veterans showed no vaccine efficacy against pneumococcal pneumonia or bronchitis (*30*); however, case definitions used were judged to have uncertain specificity. In addition, this study had only a 6% ability to detect a vaccine efficacy of 65% for pneumococcal bacteremia (*31*). In contrast, a French clinical trial found pneumococcal vaccine to be 77% effective in reducing the incidence of pneumonia in nursing home residents (*32*).

Despite conflicting findings, the data continue to support the use of the pneumococcal vaccine for certain well-defined groups at risk.

Recommendations For Vaccine Use

Adults

1. Immunocompetent adults who are at increased risk of pneumococcal disease or its complications because of chronic illnesses (e.g., cardiovascular disease, pulmonary disease, diabetes mellitus, alcoholism, cirrhosis, or cerebrospinal fluid leaks) or who are ≥65 years old.
2. Immunocompromised adults at increased risk of pneumococcal disease or its complications (e.g., persons with splenic dysfunction or anatomic asplenia, Hodgkin's disease, lymphoma, multiple myeloma, chronic renal failure, nephrotic syndrome, or conditions such as organ transplantation associated with immunosuppression).
3. Adults with asymptomatic or symptomatic HIV infection.

Children

1. Children ≥2 years old with chronic illnesses specifically associated with increased risk of pneumococcal disease or its complications (e.g., anatomic or functional asplenia [including sickle cell disease], nephrotic syndrome, cerebrospinal fluid leaks, and conditions associated with immunosuppression).
2. Children ≥2 years old with asymptomatic or symptomatic HIV infection.
3. The currently available 23-valent vaccine is not indicated for patients having only recurrent upper respiratory tract disease, including otitis media and sinusitis.

Special Groups

Persons living in special environments or social settings with an identified increased risk of pneumococcal disease or its complications (e.g., certain Native American populations).

Adverse Reactions

Approximately 50% of persons given pneumococcal vaccine develop mild side effects, such as erythema and pain at the injection site. Fever, myalgia, and severe local reactions have been reported in <1% of those vaccinated. Severe systemic reactions, such as anaphylaxis, rarely have been reported.

Precautions

The safety of pneumococcal vaccine for pregnant women has not been evaluated. Ideally, women at high risk of pneumococcal disease should be vaccinated before pregnancy.

Timing of Vaccination

When elective splenectomy is be-

Continued to page 6

Continued from page 5
ing considered, pneumococcal vaccine should be given at least 2 weeks before the operation, if possible. Similarly, for planning cancer chemotherapy or immunosuppressive therapy, as in patients who undergo organ transplantation, the interval between vaccination and initiation of chemotherapy or immunosuppression should also be at least 2 weeks.

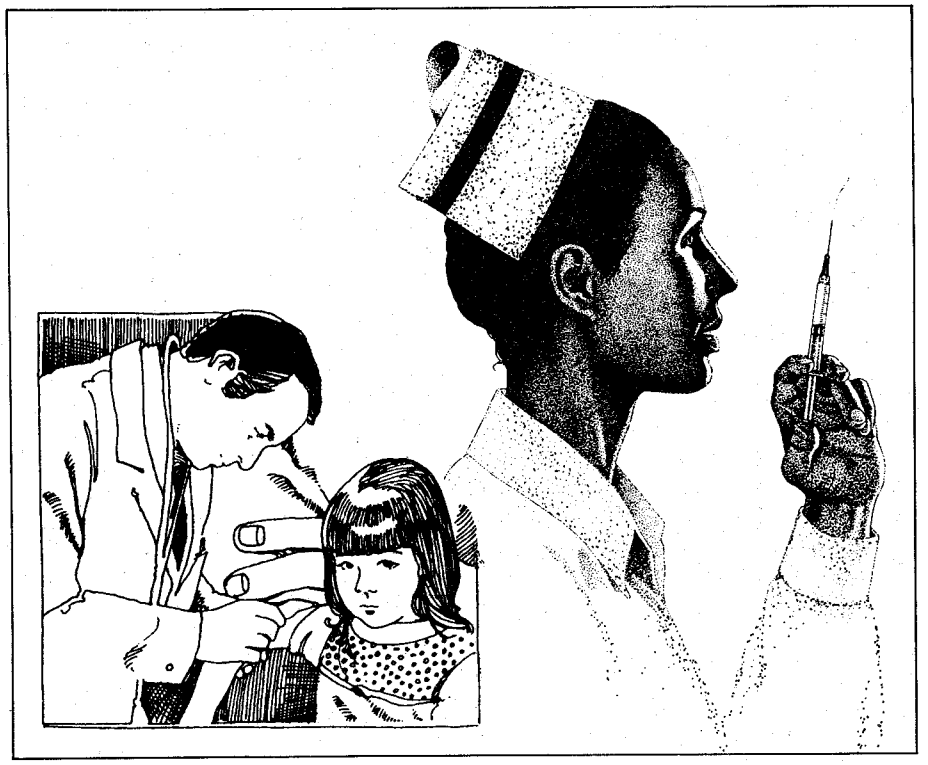
Revaccinations

In one study, local reaction after revaccination in adults were more severe than after initial vaccination when the interval between vaccinations was 13 months (*33*). Reports of revaccination after longer intervals in children and adults, including a large group of elderly persons revaccinated at least 4 years after primary vaccination, suggest a similar incidence of such reactions after primary vaccination and revaccination (unpublished data; *17,34–38*).

Without more information, persons who received the 14-valent pneumococcal vaccine should not be routinely revaccinated with the 23-valent vaccine, as increased coverage is modest and duration of protection is not well defined. However, revaccination with the 23-valent vaccine should be strongly considered for persons who received the 14-valent vaccine if they are at highest risk of fatal pneumococcal infection (e.g., asplenic patients). Revaccination should also be considered for adults at highest risk who received the 23-valent vaccine ≥6 years before and for those shown to have rapid decline in pneumococcal antibody level (e.g., patients with nephrotic syndrome, renal failure, or transplant recipients), Revaccination after 3–5 years should be considered for children with nephrotic syndrome, asplenia, or sickle cell anemia who would be ≤10 years old at revaccination.

Strategies for Vaccine Delivery

Recommendations for pneumococcal vaccination have been made by the ACIP, the American Academy of Pediatrics, the American College of Physicians, and the American Academy of Family Physicians. Recent analysis indicates that pneumococcal vaccination of elderly persons is cost-effective (*39*). The vaccine is targeted for approximately 27 million persons aged ≥65 years and 21 million persons aged <65 years with high-risk conditions (*1*). Despite Medicare reimbursement for costs of the vaccine and its administration, which began in 1981, annual use of pneumococcal vaccine has not increased above levels observed in earlier years (*40*). In 1985, <10% of the 48 million persons considered to be at increased risk of serious pneumococcal infection were estimated to have ever received pneumococcal vaccine (*1*).



Opportunities to vaccinate high-risk persons are missed both at time of hospital discharge and during visits to clinicians' offices. Two thirds or more of patients with serious pneumococcal disease had been hospitalized at least once within 5 years before their pneumococcal illness, yet few had received pneumococcal vaccine (*40*). More effective programs for vaccine delivery are needed, including offering pneumococcal vaccine in hospitals (at the time of discharge), clinicians' offices, nursing homes, and other chronic-care facilities. Many patients who receive pneumococcal vaccine should also be immunized with influenza vaccine (*41*), which can be given simultaneously at a different site. In contrast to pneumococcal vaccine, influenza vaccine is given annually.

Vaccine Development

A more immunogenic pneumococcal vaccine preparation is needed, particularly for children <2 years old. The development of a protein-polysaccharide conjugate vaccine for selected capsular types holds promise.

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Adapted from MMWR 1989; 38:64-68, 73-76.

Erratum

Please note the following change in your copy of the June 1989 Virginia Epidemiology Bulletin (Volume 89, Number 6): page 3, first paragraph, line 3 should read "**retested** for syphilis," not "retreated for syphilis."

Cases of selected notifiable diseases, Virginia, for the period July 1 through July 31, 1989.

Disease	Total Cases Reported This Month						Total Cases Reported To Date		
	State	Regions					This Year	Last Year (State Totals)	5 Year Average
		N.W.	N.	S.W.	C.	E.			
Acquired Immunodeficiency Syndrome	30	3	13	4	7	3	244	211	—
Campylobacter Infections	94	30	26	11	15	12	377	287	321
Gonorrhea	1211	—	—	—	—	—	8851	7657	9665
Hepatitis A	20	0	0	1	17	2	190	256	129
B	24	1	5	7	4	7	169	199	267
Non A–Non B	14	1	3	1	7	2	40	51	48
Influenza	14	0	0	0	0	14	1815	2420	1918
Kawasaki Syndrome	0	0	0	0	0	0	7	11	15
Legionellosis	3	2	0	1	0	0	5	6	9
Lyme Disease	4	0	0	0	0	4	18	17	7
Measles	1	0	0	0	0	1	21	143	45
Meningitis—Aseptic	21	6	9	1	5	0	93	67	96
Bacterial*	7	1	2	2	1	1	113	94	132
Minngococcal Infections	10	4	1	1	3	1	40	39	45
Mumps	4	0	2	0	2	0	59	104	48
Pertussis	3	1	0	0	1	1	9	16	19
Rabies in Animals	24	9	2	5	4	4	163	232	163
Reye Syndrome	0	0	0	0	0	0	1	0	2
Rocky Mountain Spotted Fever	2	1	0	1	0	0	5	12	16
Rubella	0	0	0	0	0	0	0	11	3
Salmonellosis	215	24	54	34	46	57	714	667	743
Shigellosis	50	3	12	2	11	22	304	242	109
Syphilis (Primary & Secondary)	52	0	9	13	17	13	319	246	209
Tuberculosis	36	6	7	0	9	14	206	226	230

Localities Reporting Animal Rabies: Amelia 1 raccoon; Arlington 1 bat; Augusta 1 cat, 1 fox, 2 raccoons; Culpeper 2 raccoons; Fauquier 1 fox; Gloucester 3 raccoons; Grayson 1 bat; Highland 1 raccoon; Loudoun 1 bat; Nottoway 1 cat, 1 fox; Prince Edward 1 raccoon; Scott 1 skunk; Spotsylvania 1 raccoon; Tazewell 1 skunk; Washington 2 skunks; York 1 raccoon.

Occupational Illnesses: Asbestosis 51; Carcinoma of the Lung 1; Carpal Tunnel Syndrome 20; Chemical Poisoning 1; Coal Workers' Pneumoconiosis 28; Dermatitis 1; Loss of Hearing 11; Mesothelioma 1; Repetitive Trauma Disorder 12.

*other than meningococcal

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