EPIDEMIOLOGY BULLETIN

James B. Kenley, M.D., Commissioner
Grayson B. Miller, Jr., M.D., Epidemiologist

Editor: Carl W. Armstrong, M.D.

June, 1985

Volume 85, Number 6

Vibrio vulnificus and Patients With Liver Disease

Patients with chronic illness such as liver disease and other diseases with possible or definite hepatic involvement, such as hemochromatosis, thalassemia major, alcoholism or malignancy, should be advised not to eat raw or uncooked seafood. Increasing evidence indicates that patients with such chronic diseases are at higher risk of septicemia and death from eating raw seafood, especially raw oysters, due to contamination with *Vibrio vulnificus*.[1,2]

The reason for the higher susceptibility to *V. vulnificus* infection among patients with liver disease is not entirely clear. However, it is suspected that these patients are more susceptible to bacterial infections because of poor nutrition and defective blood cell functions, including decreased bactericidal activity. Impaired iron metabolism in patients with hemochromatosis and alcohol-related and other liver disease is likely to play a role in susceptibility to infection.

V. vulnificus is endemic in the marine environment and is commonly recovered from nonpolluted seawater, estuarine sediments and seafood. There is no known practical measure to identify seafood that may be contaminated with this organism. Current data suggest that *V. vulnificus* infections are endemic along the coast of the Gulf of Mexico and to a lesser extent along the Pacific and Atlantic coasts, including the Chesapeake Bay.[3] The infections tend to occur in fishermen and shellfish consumers during the relatively warm months of May to October.[4-7]

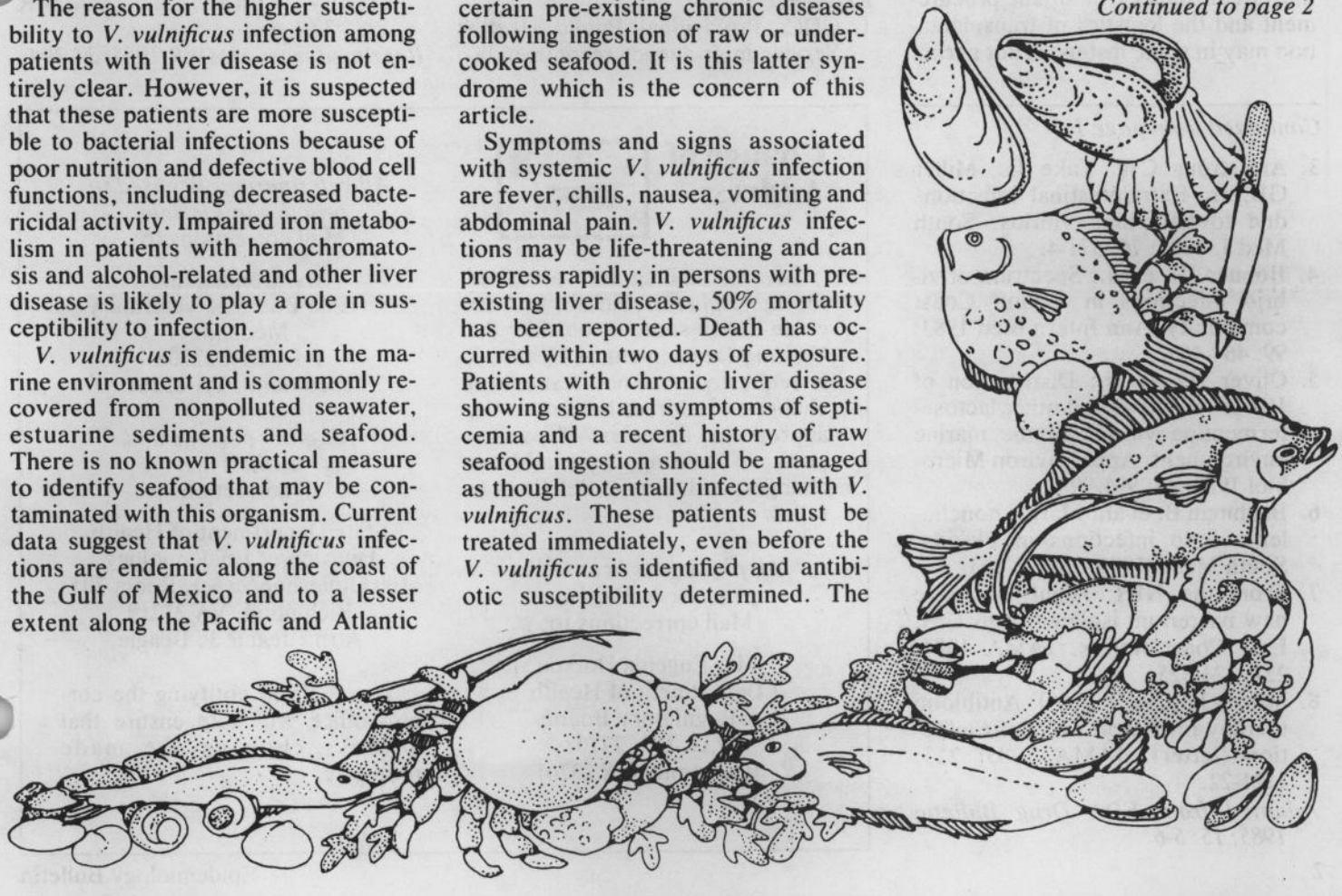
V. vulnificus causes two distinct clinical syndromes with different portals of entry. The first is wound infection, which results from injury sustained in, or secondarily contaminated by, seawater or shellfish handling. The second syndrome is that of septicemia among persons with certain pre-existing chronic diseases following ingestion of raw or undercooked seafood. It is this latter syndrome which is the concern of this article.

Symptoms and signs associated with systemic *V. vulnificus* infection are fever, chills, nausea, vomiting and abdominal pain. *V. vulnificus* infections may be life-threatening and can progress rapidly; in persons with preexisting liver disease, 50% mortality has been reported. Death has occurred within two days of exposure. Patients with chronic liver disease showing signs and symptoms of septicemia and a recent history of raw seafood ingestion should be managed as though potentially infected with *V. vulnificus*. These patients must be treated immediately, even before the *V. vulnificus* is identified and antibiotic susceptibility determined. The organism is sensitive to some antimicrobial agents used in sepsis or severe wound infections; e.g., tetracycline (drug of choice), furazolidone, and chloramphenicol.[8]

References

1. Tacket CO, et al.: Clinical features and an epidemiological study of *Vibrio vulnificus* infections. J. Infect Dis 1984; 149: 558-561.
2. Blake PA, et al.: Disease caused by marine vibrio. N Engl J Med 1979; 300: 1-5.

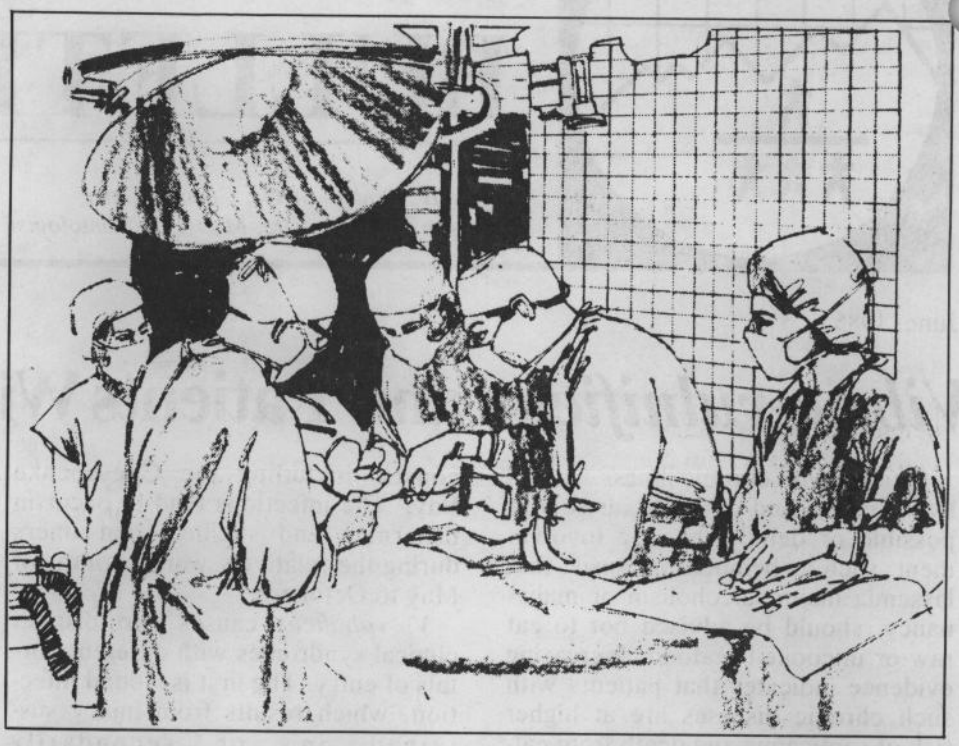
Continued to page 2



Antibody to HTLV-III/LAV

Testing Donors of Organs, Tissues, and Semen

The U.S. Public Health Service has recommended that all donated blood and plasma be tested for antibody to human T-lymphotropic virus type III/ lymphadenopathy-associated virus (HTLV-III/LAV), the virus that causes acquired immunodeficiency syndrome (AIDS) (*1*). It is additionally recommended that blood or serum from donors of organs, tissues, or semen intended for human use be similarly tested and that the test result be used to evaluate the appropriate use of such materials from these donors. Although AIDS has not been reported to have been associated with such use, semen and other body fluids, including blood, may harbor the virus. Thus, organs, tissues, and semen obtained from HTLV-III/LAV antibody-positive persons must be considered as potentially infectious. Persons in groups having an increased risk for AIDS should not donate organs, tissues, or semen, regardless of the result of the antibody test; this is the same policy currently followed for blood donations. It is recognized that the circumstances of organ procurement and the logistics of transplantation may in some instances not permit



the use of an HTLV-III/LAV test. However, when feasible such testing is prudent.

Reference

1. CDC. Provisional Public Health Service inter-agency recommendations for screening donated blood and plasma for antibody to the virus causing acquired immunodeficiency syndrome. MMWR 1985;34:1-5.

Reprinted from MMWR 1985; 34:294.

Continued from page 1

3. Armstrong CW, Lake JL, Miller GB, Jr. Extraintestinal infections due to halophilic vibrios. South Med J. 1983; 76: 571-4.
4. Bonner JR, et al.: Spectrum of vibrio infections in a Gulf Coast community. Ann Intern Med 1983; 99: 464-469.
5. Oliver JD, et al.: Distribution of *Vibrio vulnificus* and other lactose-fermenting vibrios in the marine environment. Appl Environ Microbiol 1983; 45: 985-998.
6. Bachman B, et al.: Marine noncholera vibrio infections in Florida. South Med J 1983; 76:296-303.
7. From the NIH: Highly invasive new bacterium isolated from U.S. East Coast waters. JAMA 1984; 251: 323-325.
8. Morris JG, and Tenny J: Antibiotic therapy for *Vibrio vulnificus* infection (letter). JAMA 1985; 253: 1121-22.

Adapted from FDA Drug Bulletin 1985; 15: 5-6.

Change of Address

Several mailing lists are used for the *Bulletin*. Since two lists come from sources outside the Virginia Department of Health, it is requested that notifications of changes of address be sent to the appropriate agencies. Please report, in writing, any address changes, additions or deletions as follows:

All physicians licensed to practice in Virginia.
Mail corrections to:

Ms. Eugenia Dorson
Department of Health
Regulatory Boards
P.O. Box 27708
Richmond, VA 23261

All veterinarians licensed to practice in Virginia.
Mail corrections to:

Ms. Connie Tate
Virginia Board of Veterinary Medicine
P.O. Box 27708
Richmond, VA 23261

All other persons and institutions.
Mail corrections to:

State Department of Health
Division of Epidemiology
109 Governor Street, Room 701
Richmond, VA 23219
Attn.: Jeanie P. Beagle

Your help in notifying the correct office will help ensure that address changes are made promptly.

Recommendation of the Immunization Practices Advisory Committee (ACIP) of the U.S. Public Health Service

Meningococcal Vaccines

Introduction

A polysaccharide vaccine against disease caused by *Neisseria meningitidis* serogroups A, C, Y, and W-135 is currently licensed in the United States. This statement updates the previous statement (*MMWR* 1978;27:327-9), summarizes available information on the vaccine, and offers guidelines for its use in the civilian population of the United States.

Meningococcal Disease

N. meningitidis causes both endemic and epidemic disease, principally meningitis and meningococcemia. It is the second most common cause of bacterial meningitis in the United States (approximately 20% of all cases), affecting an estimated 3,000-4,000 people each year. The case-fatality rate is approximately 10% for meningococcal meningitis and 20% for meningococcemia, despite therapy with antimicrobial agents, such as penicillin, to which all strains remain highly sensitive.

No major epidemic of meningococcal disease has occurred in the United States since 1946, although localized community outbreaks have been reported. The incidence of endemic meningococcal disease peaks in the late winter to early spring. Attack rates are highest among children aged 6-12 months and then steadily decline; by age 5 years, the incidence approximates that for adults. Serogroup B, for which a vaccine is not yet available, accounts for 50%-55% of all cases; serogroup C, for 20%-25%; and serogroup W-135, for 15%. Serogroups Y (10%) and A (1%-2%) account for nearly all remaining cases. Serogroup W-135 has emerged as a major cause of disease only since 1975 (*1*). While serogroup A causes only a small proportion of endemic disease in the United States, it is the most common cause of epidemics elsewhere. Less commonly, serogroups C and B can also cause epidemic disease.

People with certain chronic conditions appear to be at increased risk of developing meningococcal infection. Meningococcal disease is particularly common among individuals with component deficiencies in the final common complement pathway (C3, C5-C9), many of whom experience multiple episodes of infection (*2*). Asplenic persons seem also to be at increased risk of developing meningococcal disease and experience particularly severe infections (*3*). It is uncertain whether individuals with other diseases associated with immunosuppression are at higher risk of acquiring meningococcal disease, as they are for disease caused by other encapsulated bacteria. In the past, new military recruits were at especially high risk, particularly for serogroup C disease; however, since routine vaccination of recruits with the bivalent A/C vaccine began in 1971, disease caused by those serogroups has been uncommon. Military recruits currently receive the A,C,Y,W-135 vaccine.

Meningococcal Polysaccharide Vaccines

The recently licensed quadrivalent A,C,Y,W-135 vaccine (Menomune®—A/C/Y/W-135, manufactured by Squibb-Connaught) is the formulation currently available in the United States. The vaccine consists of 50 μg each of the respective purified bacterial capsular polysaccharides.

Vaccine efficacy. Numerous studies have demonstrated the immunogenicity and clinical efficacy of the A and C vaccines. The serogroup A polysaccharide induces antibody in some children as young as 3 months of age, although a response comparable to that seen in adults is not achieved until 4 or 5 years of age; the serogroup C component does not induce a good antibody response before age 18-24 months (*4,5*). The serogroup A vaccine has been shown to have a clinical efficacy of 85%-95% and to be of use in controlling epidemics. A similar level of clinical efficacy has been demonstrated for the serogroup C vaccine, both in American military recruits and in an epidemic. The group Y and W-135 polysaccharides have been shown to be safe and immunogenic in adults (*6-9*) and in children over 2 years of age; clinical protection has not been demonstrated directly, but is assumed, based on the production of bactericidal antibody, which for group C has been correlated with clinical protection. The antibody responses to each of the four polysaccharides in the quadrivalent vaccine are serogroup-specific and independent.

Duration of efficacy. Antibodies against the group A and C polysaccharides decline markedly over the first 3 years following a single dose of vaccine (*5,10-13*). This antibody decline is more rapid in infants and young children than in adults. Similarly, while vaccine-induced clinical protection probably persists in schoolchildren and adults for at least 3 years, a recent study in Africa has demonstrated a marked decline in the efficacy of the group A vaccine in young children over time. In this study, efficacy declined from greater than 90% to less than 10% over 3 years in those under 4 years of age at the time of vaccination; in older children, efficacy was still 67% 3 years after vaccination (*14*).

Recommendations for Vaccine Use

Routine vaccination of civilians with meningococcal polysaccharide vaccine is not recommended for the following reasons: (1) the risk of infection in the United States is low; (2) a vaccine against serogroup B, the major cause of meningococcal disease in the United States, is not yet available; and (3) much of the meningococcal disease in the United States occurs among children too young to benefit from the vaccine. However, the vaccine has been shown to be of use in aborting outbreaks due to serogroups represented in the vaccine and should be used in their control. In an out-

Continued to page 4

Continued from page 3

break, the serogroup should be determined and the population at risk delineated by neighborhood, school, dormitory, or other reasonable boundary. Although endemic disease is very uncommon above age 5 years, older children, adolescents, and young adults constitute a higher proportion of cases during epidemics and may warrant vaccination during an outbreak (*15*).

Routine immunization with the quadrivalent vaccine is recommended for particular high-risk groups, including individuals with terminal complement component deficiencies and those with anatomic or functional asplenia. Persons splenectomized because of trauma or nonlymphoid tumors and those with inherited complement deficiencies have acceptable antibody responses to meningococcal vaccine, although clinical efficacy has not been documented (*2,16*). It should be recognized that such individuals frequently have preexisting antibody against *N. meningitidis* and may not be protected by vaccination.

Vaccination with the A-C vaccine may benefit some travelers to countries recognized as having hyperendemic or epidemic disease and Americans living in these areas, particularly those who will have prolonged contact with the local populace. One area of the world recognized as having recurrent epidemics of meningococcal disease is the part of sub-Saharan Africa known as the "meningitis belt," which extends from Mauritania in the west to Ethiopia in the east. Epidemics have been recognized in other parts of the world, and updated information can be obtained from travelers' clinics, state health departments, and CDC.

Primary Immunization. For both adults and children, vaccine is administered subcutaneously as a single 0.5-ml dose. The vaccine can be given at the same time as other immunizations, if needed. Good antibody levels are achieved within 10-14 days after vaccination.

Precautions and Contraindications

Reactions. Adverse reactions to meningococcal vaccine are mild and infrequent, consisting principally of localized erythema lasting 1-2 days. Up to 2% of young children develop fever transiently after vaccination (*13*).

Pregnancy. On theoretical grounds, it is prudent not to immunize pregnant women unless there is a substantial risk of infection. However, evaluation of the vaccine in pregnant women during an epidemic in Brazil demonstrated no adverse effects. Further, antibody studies in these women showed good antibody levels in maternal and cord blood following vaccination during any trimester; antibody levels in the infants declined over the first few months and did not affect their subsequent response to immunization (*17*).

Revaccination

Revaccination may be indicated for individuals at high risk of infection, particularly children who were first immunized under 4 years of age; such children should be considered for revaccination after 2 or 3 years if they remain at high risk. The need for revaccination in older children and adults remains unknown.

Prospects for Future Meningococcal Vaccines

Work is continuing on a serogroup B meningococcal vaccine, as well as on improved A and C vaccines. Candidate vaccines include capsular polysaccharides complexed with meningococcal outer-membrane proteins or covalently linked to carrier proteins. Clinical efficacy data for these vaccines are not available.

Antimicrobial Chemoprophylaxis

Antimicrobial chemoprophylaxis of intimate contacts remains the chief preventive measure in sporadic cases of *N. meningitidis* disease in the United States. Intimate contacts include (1) household members, (2) day-care-center contacts, and (3) anyone directly exposed to the patient's oral secretions, such as through mouth-to-mouth resuscitation or kissing. The attack rate for household contacts is 0.3%-1%, 300-1,000 times the rate in the general population.

Unless the causative organism is known to be sensitive to sulfadiazine, the drug of choice is rifampin, given twice daily for 2 days (600 mg every 12 hours to adults; 10 mg/kg every 12 hours to children 1 month of age or older; 5 mg/kg every 12 hours to children under 1 month of age). Rifampin has been shown to be 90% effective in eradicating nasopharyngeal carriage. No serious adverse effects have been noted. However, rifampin prophylaxis is not recommended for pregnant women, as the drug is teratogenic in laboratory animals. Also, as well as turning urine orange, rifampin is excreted in tears, resulting in staining of contact lenses; thus, they should not be used during the course of therapy.

Because systemic antimicrobial therapy of meningococcal disease does not reliably eradicate nasopharyngeal carriage of *N. meningiditis*, it is also important to give chemoprophylaxis to the index patient before discharge from the hospital (*18*).

Nasopharyngeal cultures are not helpful in determining who warrants chemoprophylaxis and unnecessarily delay institution of this preventive measure.

References

1. Band JD, Chamberland ME, Platt T, Weaver RE, Thornsberry C, Fraser DW. Trends in meningococcal disease in the United

States, 1975-1980. J Infect Dis 1983;148:754-8.
2. Ross SC, Densen P. Complement deficiency states and infection: epidemiology, pathogenesis and consequences of neisserial and other infections in an immune deficiency. Medicine 1984;63:243-73.
3. Francke EL, Neu HC. Postsplenectomy infection. Surg Clin North Am 1981;61:135-55.
4. Peltola H, Käyhty H, Kuronen T, Haque N, Sarna S, Mäkelä PH. Meningococcus group A vaccine in children three months to five years of age. Adverse reactions and immunogenicity related to endotoxin content and molecular weight of the polysaccharide. J Pediatr 1978;92:818-22.
5. Gold R, Lepow ML, Goldschneider I, Draper TF, Gotschlich EC. Kinetics of antibody production to group A and group C meningococcal polysaccharide vaccines administered during the first six years of life: prospects for routine immunization of infants and children. J Infect Dis 1979;140:690-7.
6. Griffiss JM, Brandt BL, Altieri PL, Pier GB, Berman SL. Safety and immunogenicity of group Y and group W135 meningococcal capsular polysaccharide vaccines in adults. Infect Immun 1981;34:725-32.
7. Armand J, Arminjon F, Mynard MC, Lafaix C. Tetravalent meningococcal polysaccharide vaccine groups A,C,Y,W135: clinical and serological evaluation. J Biol Stand 1982;10:335-9.
8. Ambrosch F, Wiedermann G, Crooy P, George AM. Immunogenicity and side-effects of a new tetravalent meningococcal polysaccharide vaccine. Bull WHO 1983;61:317-23.
9. Vodopija I, Baklaic Z, Hauser P, Roelants P, Andre FE, Safary A. Reactivity and immunogenicity of bivalent (AC) and tetravalent (ACW135Y) meningococcal vaccines containing O-acetyl-negative or O-acetyl-positive group C polysaccharide. Infect Immun 1983;42:599-604.
10. Artenstein MS. Meningococcal infections: 5. Duration of polysaccharide-vaccine-induced antibody. Bull WHO 1971;45:291-3.
11. Lepow ML, Goldschneider I, Gold R, Randolph M, Gotschlich EC. Persistence of antibody following immunization of children with groups A and C meningococcal polysaccharide vaccines. Pediatrics 1977;60:673-80.
12. Greenwood BM, Whittle HC, Bradley AK, Fayet MT, Gilles HM. The duration of the antibody response to meningococcal vaccination in an African village. Trans R Soc Trop Med Hyg 1980;74:756-60.
13. Käyhty H, Karanko V, Peltola H, Sarna S, Mäkelä PH. Serum antibodies to capsular polysaccharide vaccine of group A *Neisseria meningitidis* followed for three years in infants and children. J Infect Dis 1980;142:861-8.
14. CDC. Unpublished data.
15. Peltola H. Meningococcal disease: still with us. Rev Infect Dis 1983;5:71-91.
16. Ruben FL, Hankins WA, Zeigler Z, et al. Antibody responses to meningococcal polysaccharide vaccine in adults without a spleen. Am J Med 1984;76:115-21.
17. McCormick JB, Gusmao HH, Nakamura S, et al. Antibody response to serogroup A and C meningococcal polysaccharide vaccines in infants born of mothers vaccinated during pregnancy. J Clin Invest 1980;65:1141-4.
18. Abramson JS, Spika JS. Persistence of *Neisseria meningitidis* in the upper respiratory tract after intravenous antibiotic therapy for systemic meningococcal disease. J Infect Dis 1985;151:370-1.

Reprinted from MMWR 1985;34: 255-9.

Month: June, 1985

Disease	State				Mean 5 Year To Date	Regions				
	This Month	Last Month	Total to Date 1985	Total to Date 1984		This Month N.W.	N.	S.W.	C.	E.
Measles	2	4	18	2	68	0	2	0	0	0
Mumps	7	5	28	12	39	2	1	2	1	1
Pertussis	2	0	5	11	12	0	0	1	1	0
Rubella	0	1	1	0	11	0	0	0	0	0
Meningitis—Aseptic	24	5	89	62	48	3	5	2	10	4
*Bacterial	16	9	135	138	118	1	6	1	6	2
Hepatitis A (Infectious)	14	7	101	55	93	2	10	0	0	2
B (Serum)	63	49	292	247	251	2	28	5	14	14
Non-A, Non-B	11	4	50	55	33	0	3	2	0	6
Salmonellosis	230	152	752	482	531	43	37	34	54	62
Shigellosis	7	7	37	119	245	1	2	0	0	4
Campylobacter Infections	102	49	299	245	123	15	33	3	23	28
Tuberculosis	42	37	187	220	—	—	—	—	—	—
Syphilis (Primary & Secondary)	23	21	155	218	290	1	3	4	5	10
Gonorrhea	1924	1264	9056	9615	9932	—	—	—	—	—
Rocky Mountain Spotted Fever	7	3	10	16	21	0	3	1	2	1
Rabies in Animals	14	9	88	129	165	4	7	1	2	0
Meningococcal Infections	4	1	36	41	44	1	0	0	1	2
Influenza	9	17	922	1094	1571	2	1	6	0	0
Toxic Shock Syndrome	0	0	0	5	4	0	0	0	0	0
Reyes Syndrome	1	0	2	5	8	0	0	1	0	0
Legionellosis	0	2	7	12	9	0	0	0	0	0
Kawasaki's Disease	1	6	21	8	12	0	0	1	0	0
Other: Acquired Immunodeficiency Syndrome	7	7	32	13	—	0	2	0	4	1

Counties Reporting Animal Rabies: Madison 1 raccoon; Rockingham 1 lamb; Shenandoah 2 raccoons; Arlington 2 raccoons; Fairfax 1 bat, 1 raccoon; Loudoun 2 raccoons; Prince William 1 raccoon; Patrick 1 bat; Hanover 2 raccoons.

Occupational Illnesses: Pneumoconiosis 26; Carpal tunnel syndrome 24; Hearing loss 7; Asbestosis 4; Brucellosis 1; Cancer, prostate 1; Mesothelioma 1.

*other than meningococcal

Published Monthly by the
VIRGINIA HEALTH DEPARTMENT
Division of Epidemiology
109 Governor Street
Richmond, Virginia 23219