VIRGINIA
EPIDEMIOLOGY BULLETIN

C.M.G. Buttery, M.D., M.P.H., Commissioner
Grayson B. Miller, Jr., M.D., Epidemiologist
Editor: Carl W. Armstrong, M.D.

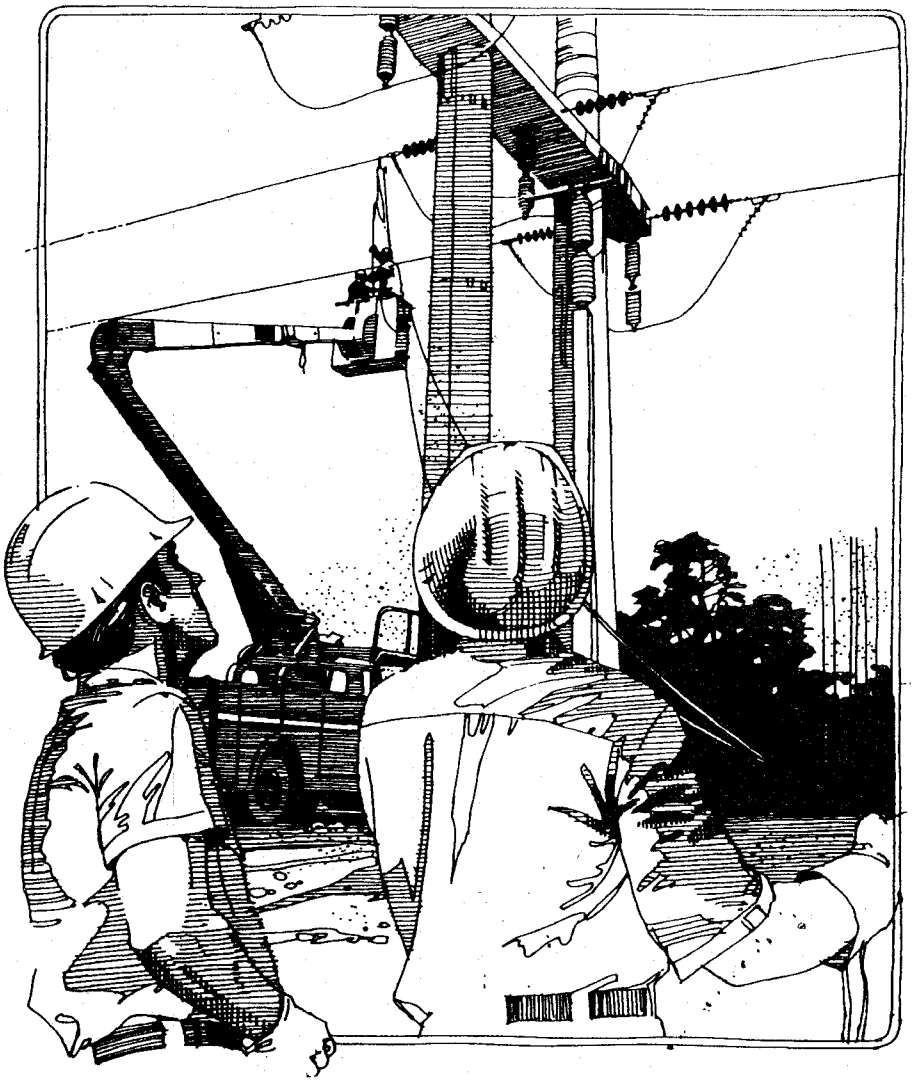
December, 1988
Volume 88, Number 12

Fatal Occupational Injuries Due to Electric Shock, Virginia

Fatal occupational injuries due to electric shock cause many years of potential working-life lost. To determine the characteristics of these injuries and deaths in Virginia, death certificates, worker's compensation files, and medical examiner's records were reviewed.[1]

For the years 1977–1985, 196 work-related electrical fatalities were identified for a mean rate of 0.9/100,000 workers per year. Death certificates identified 186, worker's compensation files identified 95, and 85 were included in both systems. Sixty-five percent of these deaths occurred between May and September each year. The median age at death was 29 years, and all but one of the decedents was male. Death rates were highest for male workers in utility companies (10.0/100,000), mining (6.8/100,000), and construction industries (4.3/100,000), but these high risk groups accounted for only 56% of the deaths. Most of these deaths resulted from power line contact (52%) and machine or tool usage or repair (18%). Only two of 101 workers who died within six hours of injury and had blood alcohol concentration tested were legally intoxicated.

Analysis of both electrical injuries and deaths was done with computerized worker's compensation files for the years 1980–1985. Of the 341 workers who sustained injury during these years and reported this injury, 72 were listed as deaths (crude fatality ratio = 21%). Utility workers had the highest fatality ratio (75%), four-



times greater than the fatality ratio for other workers (18%, relative risk = 4.1 [95% confidence interval 3.8, 5.2]). The circumstances of injury were known for 315 workers (92%), and the majority of injuries were related to machines or tools (53%).

Reported by Jacob E. Jones, M.D., former Assistant State Epidemiolo-

Continued to page 2

Continued from page 1

gist (currently Hanover Health District Director), with assistance from Jim Sutton with the Industrial Commission of Virginia, and Wayne Huffner and David Wiecking, M.D., Office of the Virginia Chief Medical Examiner.

Comment: Nationally, electric shock injuries are responsible for approximately 1000 deaths per year. In Virginia, about two thirds of fatal electric shock injuries occur at the workplace and occupational fatality reports to the Occupational Safety Division, Virginia Department of Labor and Industry, in 1983 and 1984 ranked electrical fatalities as the second leading cause of occupational death in Virginia. The National Institute for Occupational Safety and Health (NIOSH) has determined that there are five primary causes of fatal occupational electric shock injuries. These include direct physical contact between the worker and energized lines, contact of a vehicular boom with energized powerlines, contact of other equipment with energized powerlines, direct contact of the worker with energized equipment or conductors, and improperly installed or broken equipment.[2] Precautions to be used around powerlines are stipulated in the Virginia Occupational Safety and Health Standards, which are enforced by the Virginia Occupational Safety and Health Program in the Virginia Department of Labor and Industry. This study, however, emphasizes the need for safety education on the job for all workers, not just those at high risk. Furthermore, every electrical injury or death should be considered a preventable event.

References:

1. Centers for Disease Control. Abstracts of the 1987 Epidemic Intelligence Service Conference, Atlanta, Ga.

2. Centers for Disease Control. Occupational electrocution—Texas, 1981–1985. MMWR 1987;36:725–7.

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

Use of BCG Vaccines in the Control of Tuberculosis

Since 1979, when the last Immunization Practices Advisory Committee (ACIP) statement on vaccination with Bacillus of Calmette and Guérin (BCG) was published, additional data have been published on the epidemiology of tuberculosis (TB) in the United States and on the efficacy of childhood BCG vaccines. As a result, ACIP and the Advisory Committee for Elimination of Tuberculosis have issued the following educational update on BCG vaccines.†*

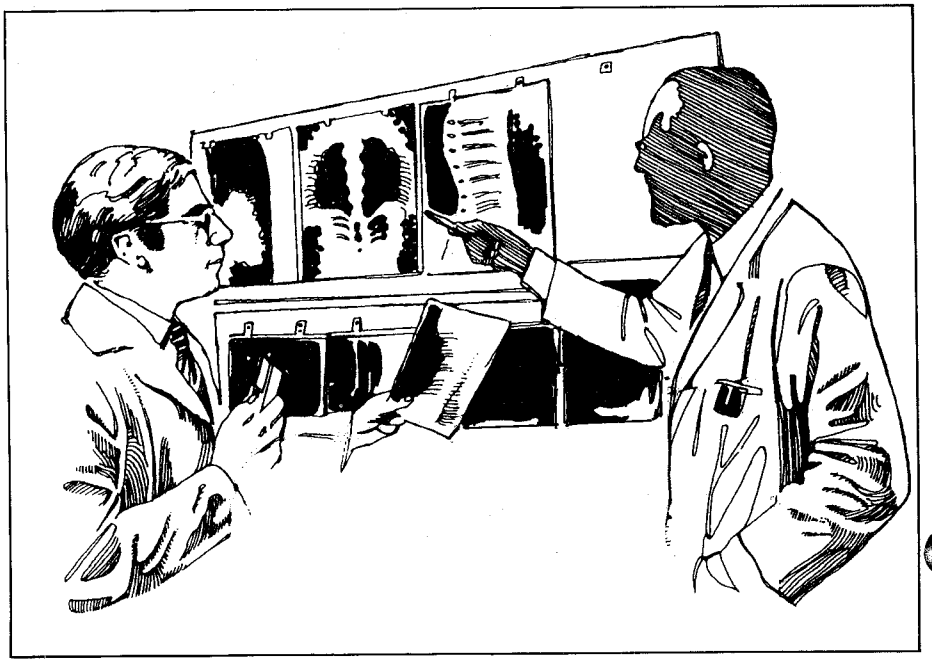
Immunization with BCG vaccine lowers the risk of serious complications of primary TB in children *(1–4)*. However, BCG vaccination should be considered only for children with negative tuberculin skin tests who fall into the following categories: 1) those who cannot be placed on isoniazid preventive therapy but who have continuous exposure to persons with active disease; 2) those with continuous exposure to patients with organisms resistant to isoniazid and rifampin; or 3) those belonging to groups with exceptionally high annual rates of new infection (i.e., >1% per year).

BCG vaccination is no longer recommended for health-care workers or other adults at high risk for acquiring TB infection. In addition, BCG should not be given to persons who are immunocompromised, including those with human immunodeficiency virus (HIV) infection.

Introduction

Transmission and Pathogenesis of TB

TB is a bacterial disease caused by organisms of the *Mycobacterium tuberculosis* complex (i.e., *M. tuberculosis, M. bovis, M. africanum*). It is transmitted primarily by airborne droplets; infection occurs when susceptible persons inhale infectious droplets producted by the exhalations of persons with respiratory tract TB. The risk for infection is directly related to duration and intensity of exposure to air contaminated with these droplets. TB infection usually begins in the lungs and spreads to the hilar lymph nodes, then to the blood stream. Thus, disease can occur in any organ of the body. Most infected persons react to the purified protein derivative (PPD) tuberculin skin test, and 5%–40% will develop clinically apparent TB. Infection is more likely to progress to clinical disease in the presence of certain risk factors, including



*Official name: BCG Vaccine.

†Replaces previous recommendation on BCG vaccines (*MMWR* 1979;28:241–4).

younger and older ages, male sex, infection within the past 2 years, leanness, and suppression of cell-mediated immunity.

TB can be presumptively diagnosed if acid-fast bacilli are found in sputum, body fluids, or tissue or if at least two of three other conditions are met: 1) symptoms are compatible with TB; 2) chest radiograph is abnormal or abnormalities are found on physical examination; or 3) reaction to the tuberculin skin test is positive. Definitive diagnosis requires isolation and identification of organisms of the *M. tuberculosis* complex from a clinical specimen. Diagnosis of extrapulmonary TB is more difficult because it requires tissue biopsies or body fluids (e.g., spinal fluid) that usually contain only a few organisms.

Epidemiology of TB in the United States

TB in the United States has declined approximately 6% per year since nationwide reporting began in 1953. However, in 1986, the morbidity rate for TB increased slightly to 9.4/100,000, a rate 82% lower than that for 1953 but 1.1% higher than the 1985 rate. A total of 22,768 cases were reported *(5)*, and approximately 80% were pulmonary disease.

Untreated TB is fatal in up to 50% of cases. However, chemotherapy has helped reduce the case-mortality rate 94% since 1953. In 1984, the most recent year for which final mortality data are available, 1729 deaths were attributed to TB, representing a mortality rate of 0.7/100,000 population.

Prevalence of TB infection and disease varies for different segments of the population. Disease rates are twice as high in males as in females and increase sharply with age in both sexes and all races. Groups at high risk for TB include most racial/ethnic minorities, immigrants from countries with a high prevalence of TB, the homeless population, close contacts of persons with pulmonary TB, and persons with HIV infection. In 1986, 62% of all TB cases occurred in racial/ethnic minorities, and over 20% of all cases were in foreign-born persons *(5)*. Although the prevalence of active TB in the homeless population is difficult to assess, surveillance of selected clinics and shelters showed infection rates between 1.6% and 6.8% *(6)*. Based on 1985 data from U.S. health departments, 29% of close contacts of TB patients were infected at the time the patients were diagnosed *(7)*. In addition, the estimated risk for active TB in persons with symptomatic HIV infection is 100–200 times greater than that of persons in the general population *(8)*. Persons with asymptomatic HIV infection and *M. tuberculosis* infection may have an equally high risk for developing clinical disease.

In 1985, the 1261 cases of TB in children <15 years of age accounted for 5.7% of cases in all age groups. Eighty percent of these were among racial/ethnic minorities *(9)*. One fourth (315) of all childhood cases were extrapulmonary; of these, 41 cases were meningeal, and 17 were miliary. Childhood cases of TB meningitis and miliary TB remained stable between 1981 and 1985, averaging 55 cases annually.

In the past, TB was regarded as an occupational hazard for health-care workers, who had higher rates of infection and disease than persons of the same age groups in the general population. Although these rates have decreased over time, persons who work with high-risk patients or in high-prevalence communities still may be at risk for new infection, defined as conversion from a negative to a positive tuberculin skin test *(10–18)*. However, in recent studies, which found increased conversion rates among health-care personnel, rates were highest in health-care workers who did not have patient contact *(10, 11)*, suggesting that conversion resulted from community-acquired infection with *M. tuberculosis* or exposure to nontuberculous mycobacteria rather than from occupational exposure.

Control of TB

There are four general strategies for controlling TB:

1. The most important and universally applied strategy is the early identification and treatment of persons with infectious TB. This strategy not only cures the affected person but also renders the patient noncontagious within a few weeks. Thus, case-finding and treatment programs have both clinical and public health benefits *(19)*.
2. Identifying and treating persons with noncontagious TB (such as extrapulmonary disease, primary pulmonary disease in children, bacteriologically unconfirmed pulmonary disease, and tuberculous infection) can prevent infectious cases *(20)*. Therapy to prevent progression of infection to clinical disease is particularly useful in countries, such as the United States, where the risk of new infection is low.
3. Use of ventilation and ultraviolet lights will decontaminate air containing infectious droplet nuclei. Because sites of potential transmission of tubercle bacilli are numerous and difficult to identify in advance, this strategy is used routinely only where the risk of transmission is known to be exceptionally high. Some of these areas include mycobacteriology laboratories, sputum induction cubicles, chest clinic waiting areas, and selected shelters for the homeless. To be effective, ventilation systems and ultraviolet lights must be properly maintained.
4. In the United States, BCG vaccination is recommended only for uninfected children who are at unavoidable risk of exposure to TB and for whom other methods of prevention and control have failed or are not feasible.

BCG Vaccines

BCG was derived from a strain of *M. bovis* attenuated through years of serial passage in culture by Calmette and Guérin at the Pasteur Institute in Lille, France. It was first administered to humans in 1921. Many BCG vaccines are available worldwide; all are derived from the original strain but vary in cultural characteristics and in ability to induce sensitization to tuberculin. BCG vaccines vary because of genetic changes in the bacterial strains and because of differences in techniques of production, in methods and routes of vaccine administration, and in characteristics of the populations and environments in which BCG vaccines have been studied.

Production standards for BCG vaccines, set by the Food and Drug Administration, specify that they be freeze-dried products containing live bacteria from a documented strain of BCG. The strain must demonstrate

Continued to page 4

Continued from page 3

various specified characteristics of safety and potency in animals and induce tuberculin sensitivity in guinea pigs and humans. The vaccines currently available in the United States have been evaluated only for their ability to induce a delayed hypersensitivity state.

Vaccine Efficacy Studies

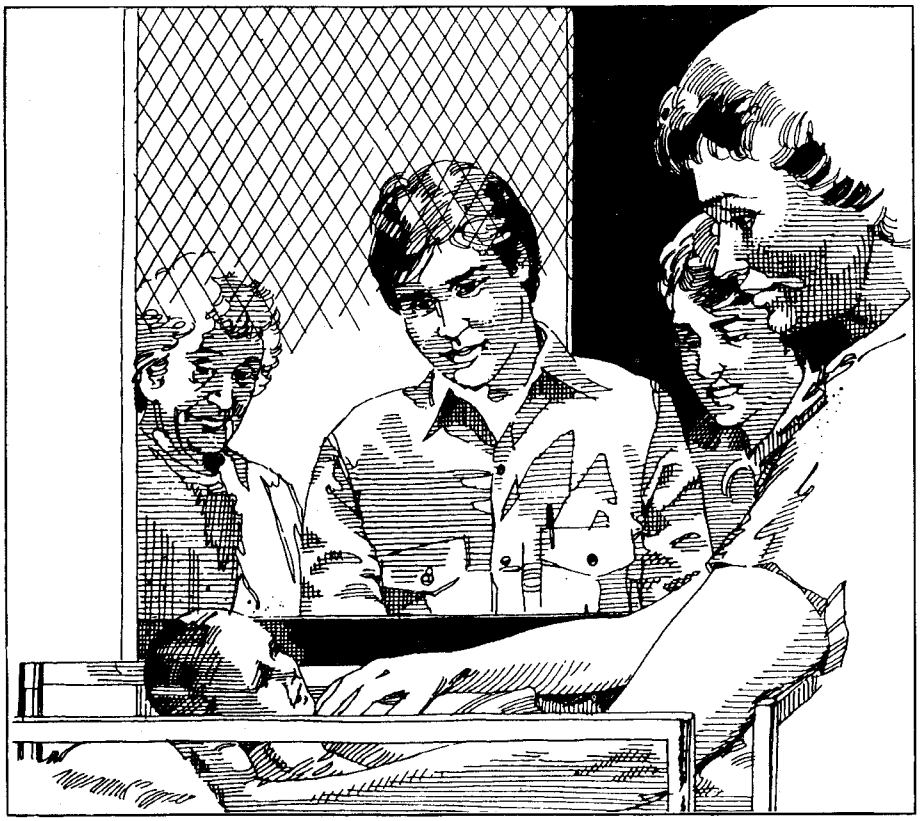
BCG vaccines vary substantially in efficacy. Different preparations of liquid BCG used in controlled community trials conducted before 1955 gave estimated efficacies ranging from −56% and 80% *(21)*. In 1969, a large controlled trial was begun in Madras (Chingleput) in south India to estimate the efficacy of two strains of freeze-dried BCG vaccine at two different doses. After 15 years of follow-up, the risk of sputum-positive pulmonary TB in persons vaccinated with BCG was not lower than that in persons given placebo *(22)*.

Although randomized controlled trials are the most reliable method for assessing vaccine efficacy, less precise estimates can be obtained more quickly and less expensively by observational studies (case-control, historical cohort, and cross-sectional studies) in areas where vaccination is performed at birth. Data from such studies show that the incidence of tuberculous meningitis and miliary TB is 52%–100% lower and that the incidence of pulmonary TB is 2%–80% lower in vaccinated children <15 years of age than in unvaccinated controls *(1–4,23,24)*. However, because vaccination is not allocated randomly in observational studies, disproportionate exposure to TB may distort the estimates of vaccine efficacy.

Side Effects and Adverse Reactions

BCG rarely causes serious complications. Side effects vary by vaccine strain; they also vary for the same strain over time. Side effects occur in 1%–10% of vaccinated persons and usually include severe or prolonged ulceration at the vaccination site, lymphadenitis, and lupus vulgaris. The risk of side effects is greater with more potent vaccines. Some vaccine strains have caused osteomyelitis in one case per million doses administered. Disseminated BCG infection and death have occurred in one to 10 cases per 10 million doses administered, although this problem is restricted almost exclusively to persons with impaired immunity.

Data on adverse reactions may pertain to the vaccines licensed in the United States. The reported frequency of complications has varied, depending in part on the intensity of the surveillance effort.



In persons with tuberculous infections, the response to BCG vaccine is accelerated. This accelerated response is generally characterized by the appearance of induration >5 mm in diameter within 24–48 hours after vaccination, formation of a pustule within 5–7 days, and scab formation and healing in 10–15 days *(25)*. The normal response to BCG vaccine begins 2–3 weeks after vaccination. Scar formation and healing occur within 3 months.

Interpretation of Tuberculin Test Following BCG Vaccination

The size of tuberculin skin test reactions caused by BCG vaccination (i.e., postvaccination sensitivity) varies by strain and dose of vaccine, age and nutritional status at vaccination, number of years since vaccination, and frequency of tuberculin testing. Mean size of skin test reactions in BCG-vaccinated children range from 3 mm to 19 mm *(26–35)*. The presence or size of postvaccination tuberculin skin test reactions does not reliably predict the degree of protection afforded by BCG *(36)*.

After BCG vaccination, it is usually not possible to distinguish between a tuberculin skin test reaction caused by virulent mycobacterial infection or by vaccination itself *(37)*. Therefore, TB should be included in the differential diagnosis of any TB-like illness, especially if the person has been recently exposed to a person with infectious TB or received BCG several years before being tuberculin tested *(38)*.

General guidelines exist for interpreting tuberculin skin test reactions in BCG vaccine recipients. The probability that a skin test reaction results from exposure to *M. tuberculosis* increases 1) as the size of the reaction increases, 2) when the patient is a contact of a person with TB, especially if that person has infected others, 3) when there is a family history of TB or when the patient's country of origin has a high TB prevalence, and 4) as the length of time between vaccination and tuberculin testing increases *(38)*. For example, a positive skin test (>10 mm) usually can be attributed to *M. tuberculosis* infection if the vaccinated person is in a group at high risk for TB or has known exposure

to a person with infectious TB. However, in vaccinated persons who do not belong to groups at high risk for infection and have no known exposure, a positive skin test reaction probably does *not* indicate recent infection with *M. tuberculosis*.

General Recommendations

In the United States, the general population is at low risk for acquiring tuberculous infection. Furthermore, TB can be controlled successfully in most high-risk groups by modern methods of case detection, chemotherapy, and preventive therapy. In most population groups, prevention of TB is most reliably accomplished by periodic Mantoux testing with PPD tuberculin for high-risk children and adults and with administration of preventive therapy to those whose skin test reactions convert from negative to positive. Preventive chemotherapy should also be given to tuberculin-positive persons who are contacts of persons with infectious TB and to other high-risk tuberculin-positive persons *(39)*. Therefore, a BCG vaccination policy for the entire population is not indicated. However, BCG vaccination may contribute to TB control in selected population groups. For example, it may benefit uninfected children who are at high risk for continuous or repeated exposure to infectious persons who remain undetected or untreated.

Recommended Vaccine Recipients

Exposed tuberculin skin-test-negative infants and children. BCG vaccination is strongly recommended for infants and children with negative tuberculin skin tests who 1) are at high risk of intimate and prolonged exposure to persistently untreated or ineffectively treated patients with infectious pulmonary TB, cannot be removed from the source of exposure, and cannot be placed on long-term preventive therapy, or 2) are continuously exposed to persons with TB who have bacilli resistant to isoniazid and rifampin.

Groups with an excessive rate of new infections. BCG vaccination is also recommended for tuberculin-negative infants and children in groups in which the rate of new infections exceeds 1% per year *(40)* and for whom the usual surveillance and treatment programs have been attempted but are not operationally feasible. These groups include persons without regular access to health care, those for whom usual health care is culturally or socially unacceptable, or groups who have demonstrated an inability to effectively use existing accessible care.

Discontinued Recommendation for Health-Care Workers

In the past, BCG vaccine was recommended for health-care workers, who as a group experienced high rates of new infection. However, BCG is *no longer recommended* for this group. Instead, health-care workers should be protected by adequate surveillance by periodic tuberculin skin testing *(41)* and isoniazid preventive therapy for all skin-test-positive health-care workers who are at high risk for developing disease. These persons include recent skin test converters and workers who are close contacts of TB patients or those who have medical conditions such as diabetes, renal failure, or immunosuppression associated with therapy or disease *(39)*. In addition, hospital infection control measures, especially the prompt identification and implementation of precautions for patients with suspected TB, will help reduce the risk of TB transmission to health-care workers *(42)*.

Vaccine Availability

Two BCG vaccine strains licensed in the United States are available. The Glaxo strain is available from Quad Pharmaceuticals, Inc., Indianapolis. The Tice strain is available from Bionetics Research, Inc. Chicago, or Antigen Supply House, Northridge, California.

Vaccine Dose and Administration

BCG should be reserved for persons whose skin test is negative to 5 tuberculin units of PPD tuberculin. The Glaxo strain is administered intradermally and the Tice strain percutaneously. Vaccination should be administered only by the route indicated in the package labeling and only in the suggested dose.

Infants <30 days old should receive one half the usual dose. If the indications for vaccination persist, they should receive a full dose at 1 year of age.

Freeze-dried vaccine should be reconstituted, protected from exposure to light, refrigerated when not in use, and used within 8 hours.

Contraindications to Use

BCG should not be given to persons 1) whose immunologic responses are impaired because of congenital immunodeficiency, HIV infection, leukemia, lymphoma, or generalized malignancy or 2) whose immunologic responses have been suppressed by steroids, alkylating agents, antimetabolites, or radiation.

BCG vaccine should be administered with caution to persons in groups at high risk for HIV infection. An AIDS patient was reported to have developed disseminated *M. bovis* disease after vaccination with BCG *(43)*. Three infants with symptomatic HIV infection were reported to have developed BCG adenitis after vaccination *(44)*; however, disseminated BCG disease has not been reported in persons with asymptomatic HIV infection.

Theoretically, persons with asymptomatic HIV infection may be at greater risk for complications from BCG vaccine, but data are inconclusive regarding this elevated risk. The World Health Organization has recommended that in populations where the risk of tuberculosis is high, HIV-infected children who are asymptomatic should receive BCG vaccine at birth or as soon as possible thereafter. BCG vaccine should not be given to children with symptomatic HIV infection *(45)*. In populations where the risk of TB is low, BCG vaccine should be withheld from persons known or suspected to be infected with HIV *(45)*. The latter recommendation would apply to most populations in the United States for whom BCG might be considered.

Continued to page 6

Continued from page 5

Use in Pregnancy

Although harmful effects of BCG on the fetus have not been observed, women snould avoid vaccination during pregnancy.

Surveillance

All suspected adverse reactions to BCG should be reported to the manufacturer and to the Office of Biologics Research, Center for Biologics Evaluation and Research, Food and Drug Administration, Bethesda, Maryland. These reactions occasionally occur >1 year after vaccination.

References

1. Romanus V. Tuberculosis in *Bacillus Calmette-Guérin* -immunized and unimmunized children in Sweden: a ten-year evaluation following the cessation of general *Bacillus Calmette-Guérin* immunization of the newborn in 1975. Pediatr Infect Dis 1987; 6:272–80.
2. Smith PG. Case-control studies of the efficacy of BCG against tuberculosis. In: International Union Against Tuberculosis, ed. Proceedings of the XXVIth IUAT World Conference on Tuberculosis and Respiratory Diseases. Singapore, Japan: Professional Postgraduate Services, International, 1987:73–9.
3. Padungchan S, Konjanart S, Kasiratta S, Daramas S, ten Dam HG. The effectiveness of BCG vaccination of the newborn against childhood tuberculosis in Bangkok. Bull WHO 1986; 64:247–58.
4. Tidjani O, Amedome A, ten Dam HG. The protective effect of BCG vaccination of the newborn against childhood tuberculosis in an African community. Tubercle 1986;67:269–81.
5. CDC. Tuberculosis, final data—United States, 1986. MMWR 1987;36:817–20.
6. Slutkin G. Management of tuberculosis in urban homeless indigents. Public Health Rep 1986; 101:481–5.
7. CDC. Tuberculosis statistics: states and cities, 1985. Atlanta: US Department of Health and Human Services, Public Health Service, 1986:84–5; HHS publication no. (CDC)87-8249.
8. CDC. Tuberculosis and AIDS—Connecticut. MMWR 1987;36: 133–5.
9. Hayden CH, Bloch AB, Snider DE. Tuberculosis among children: United States, 1985 [Abstract]. Am Rev Respir Dis 1987;135(suppl 2):A74.
10. Vogeler DM, Burke JP. Tuberculosis screening for hospital employees: a five-year experience in a large community hospital. Am Rev Respir Dis 1978;117: 227–32.
11. Ruben FL, Norden CW, Schuster N. Analysis of a community hospital employee tuberculosis screening program 31 months after its inception. Am Rev Respir Dis 1977;115:23–8.
12. Bass JB Jr, Serio RA. The use of repeat skin tests to eliminate the booster phenomenon in serial tuberculin testing. Am Rev Respir Dis 1981;123:394–6.
13. Lé CT. Cost-effectiveness of the two-step skin test for tuberculosis screening of employees in a community hospital. Infect Control 1984;5:570–2.
14. Thompson NJ, Glassroth JL, Snider DE Jr, Farer LS. The booster phenomenon in serial tuberculin testing. Am Rev Respir Dis 1979;119:587–97.
15. Valenti WM, Andrews BA, Presley BA, Reifler CB. Absence of the booster phenomenon in serial tuberculin skin testing. Am Rev Respir Dis 1982;125:323–5.
16. Catanzaro A. Nosocomial tuberculosis. Am Rev Respir Dis 1982;125:599–62.
17. Geiseler PJ, Nelson KE, Crispen RG, Moses VK. Tuberculosis in physicians: a continuing problem. Am Rev Respir Dis 1986;133:773–8.
18. Chan JC, Tabak JI. Risk of tuberculous infection among house staff in an urban teaching hospital. South Med J 1985;78: 1061–4.
19. American Thoracic Society, CDC. Control of tuberculosis. Am Rev Respir Dis 1983;128: 336–42.
20. Farer LS. Chemoprophylaxis. Am Rev Respir Dis 1982;125(Pt 2):102–7.
21. Clemens JD, Chuong JJH, Feinstein AR. The BCG controversy: a methodological and statistical reappraisal. JAMA 1983; 249:2362–9.
22. Tripathy SP. Fifteen-year follow-up of the Indian BCG prevention trial. In: International Union Against Tuberculosis, ed. Proceedings of the XXVIth IUAT World Conference on Tuberculosis and Respiratory Diseases. Singapore, Japan: Professional Postgraduate Services, International, 1987:69–72.
23. Young TK, Hershfield ES. A case-control study to evaluate the effectiveness of mass neonatal BCG vaccination among Canadian Indians. Am J Public Heath 1986;76:783–6.
24. Shapiro C, Cook N, Evans D, et al. A case-control study of BCG and childhood tuberculosis in Cali, Colombia. Int J Epidemiol 1985;14:441–6.
25. Myint TT, Yin Y, Yi MM, Aye HH. BCG test reaction in previously BCG vaccinated children. Ann Trop Paediatr 1985;5:29–31.
26. Karalliedde S, Katugaha LP, Uragoda CG. Tuberculin response of Sri Lankan children after BCG vaccination at birth. Tubercle 1987;68:33–8.
27. Bahr GM, Stanford JL, Rook GAW, Rees RJW, Abdelnoor AM, Frayha GJ. Two potential improvements to BCG and their effect on skin test reactivity in the Lebanon. Tubercle 1986;67: 205–18.
28. Heyworth B. Delayed hypersensitivity to PPD-S following BCG vaccination in African children—an 18-month field study. Trans R Soc Trop Med Hyg 1977;71:251–3.
29. Baily GVJ, Narain R, Mayurnath S, Vallishayee RS, Guld J. Trial of BCG vaccines in south India for tuberculosis prevention: tuberculosis prevention trial, Madras. Indian J Med Res 1980;72(suppl).
30. Abrahams EW. Tuberculin hypersensitivity following BCG vaccination in Brisbane school children. Tubercle 1979;60:109–13.
31. Comstock GW, Edwards LG, Nabangxang H. Tuberculin sensitivity eight to fifteen years after BCG vaccination. Am Rev Respir Dis 1971;103:572–5.
32. Stewart CJ. Skin sensitivity to human, avian and BCG PPDs after BCG vaccination. Tubercle 1968;49:84–91.

33. Guld J, Waaler H, Sundaresan TK, Kaufmann PC, ten Dam HG. The duration of BCG-induced tuberculin sensitivity in children, and its irrelevance for revaccination: results of two 5-year prospective studies. Bull WHO 1968;39:829–36.
34. Horwitz O, Bunch-Christensen K. Correlation between tuberculin sensitivity after 2 months and 5 years among BCG vaccinated subjects. Bull WHO 1972; 47:49–58.
35. Orefici G, Scopetti F, Grandolfo ME, Annesi I, Kissopoulos A. Study of a BCG vaccine: influence of dose and time. Boll Ist Sieroter Milan 1982;61:24–8.
36. Fine PEM, Pönnighaus JM, Maine NP. The relationship between delayed type hypersensitivity and protective immunity induced by mycobacterial vaccines in man. Lepr Rev 1986; 57(suppl 2):275–83.
37. American Thoracic Society, CDC. The tuberculin skin test. Am Rev Respir Dis 1981;124: 356–63.
38. Snider DE Jr. Bacille Calmette-Guérin vaccinations and tuberculin skin tests. JAMA 1985; 253:3438–9.
39. American Thoracic Society, CDC. Treatment of tuberculosis and tuberculosis infection in adults and children. Am Rev Respir Dis 1986;134:355–63.
40. Springett VH. The value of BCG vaccination. Tubercle 1965;46: 76–84.
41. Snider DE Jr, Cauthen GM. Tuberculin skin testing of hospital employees: infection, "boosting," and two-step testing. Am J Infect Control 1984;12:305–11.
42. CDC. Guidelines for prevention of TB transmission in hospitals. Atlanta: US Department of Health and Human Services, Public Health Service; HHS publication no. (CDC)82-8371.
43. CDC. Disseminated *Mycobacterium bovis* infection from BCG vaccination of a patient with acquired immunodeficiency syndrome. MMWR 1985;34:227–8.
44. Blanche S, Le Deist F, Fisher A, et al. Longitudinal study of 18 children with perinatal LAV/HTLV III infection: attempt at prognostic evaluation. J Pediatr 1986;109:965–70.
45. World Health Organization. Special Programme on AIDS and Expanded Programme on Immunization—joint statement: consultation on human immunodeficiency virus (HIV) and routine childhood immunization. Wkly Epidemiol Rec 1987;62: 297–9.

Reprinted from MMWR 1988;37: 663–664, 669–675.

Index To Volume 88

Have an Idea for the *Bulletin?*

The editor welcomes any reports of cases, outbreaks, or public health problems of interest to the Bulletin's *readers. Such accounts and any other comments or suggestions regarding the* Bulletin *should be addressed to: Editor,* Epidemiology Bulletin, *Office of Epidemiology, Room 700, 109 Governor Street, Richmond, Virginia 23219.*

Cases of selected notifiable diseases, Virginia, for the period November 1, through November 30, 1988.

Disease	State					Regions				
			Total to Date		Mean 5 Year To Date	This Month				
	This Month	Last Month	1987	1988		N.W.	N.	S.W.	C.	E.
Measles	20	34	1	220	23	0	0	20	0	0
Mumps	2	15	80	136	45	0	0	0	2	0
Pertussis	2	0	52	23	36	2	0	0	0	0
Rubella	0	0	1	11	1	0	0	0	0	0
Meningitis—Aseptic	45	43	262	194	298	13	15	2	7	8
*Bacterial	24	23	158	163	207	1	4	4	2	13
Hepatitis A (Infectious)	15	33	221	341	147	1	4	0	4	6
B (Serum)	33	34	405	306	475	1	4	3	9	16
Non-A, Non-B	6	10	44	73	72	0	0	0	0	6
Salmonellosis	129	310	1717	1645	1449	14	36	23	25	31
Shigellosis	39	56	213	433	157	4	4	4	15	12
Campylobacter Infections	87	105	589	665	589	16	20	13	11	27
Tuberculosis	39	31	397	372	397	4	12	3	7	13
Syphilis (Primary & Secondary)	36	46	297	399	363	0	5	3	24	4
Gonorrhea	1014	1318	13332	12957	17320	—	—	—	—	—
Rocky Mountain Spotted Fever	0	3	21	18	41	0	0	0	0	0
Rabies in Animals	30	24	340	338	297	7	6	5	6	6
Meningococcal Infections	8	3	67	54	65	1	1	2	1	3
Influenza	15	19	1270	2478	1686	0	0	0	2	13
Toxic Shock Syndrome	0	0	1	1	6	0	0	0	0	0
Reye Syndrome	0	0	0	0	3	0	0	0	0	0
Legionellosis	1	1	11	11	22	0	0	0	1	0
Kawasaki's Disease	1	1	25	13	25	0	0	0	1	0
Acquired Immunodeficiency Syndrome	31	26	225	345	—	4	8	0	10	9

Counties Reporting Animal Rabies: Albemarle 1 raccoon; Amelia 1 bat, 1 skunk; Arlington 1 raccoon; Augusta 1 raccoon; Botetourt 1 skunk; Chesapeake 1 bat; Chesterfield 1 bat, 2 raccoons; Clarke 1 fox; Craig 1 skunk; Fairfax 1 raccoon; Fauquier 1 skunk; Frederick 1 raccoon; Henry 1 horse; James City 1 raccoon, 1 skunk; Lancaster 1 fox, 1 skunk; Loudoun 3 raccoons, 1 skunk; Richmond City 1 raccoon; Rockbridge 1 skunk; Warren 1 raccoon; Washington 2 skunks, Williamsburg 1 raccoon.

Occupational Illnesses: Asbestosis 29; Carpal Tunnel Syndrome 2; Loss of Hearing 17; Mesothelioma 1; Occupational Asthma 1; Pneumoconioses 27; Poisoning-Metal 1.

*other than meningococcal

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

Bulk Rate
U.S. POSTAGE
PAID
Richmond, Va.
Permit No. 1225