Chapter 9

COMMUNICABLE DISEASE CONTROL IN BASIC TRAINING: PROGRAMMATIC ASPECTS

JEFFREY D. GUNZENHAUSER, MD, MPH

INTRODUCTION

A PERSPECTIVE ON THE CONTROL OF COMMUNICABLE DISEASE

PREVENTIVE INTERVENTIONS RELATED TO MODE OF TRANSMISSION

Airborne Direct Contact Foodborne and Waterborne Vector-borne

SURVEILLANCE FOR COMMUNICABLE DISEASE

CONTROL OF SPECIFIC DISEASES Influenza Adenovirus Meningococcal Disease Group A Streptococcal Disease Other Agents of Communicable Disease

SUMMARY

Military Preventive Medicine: Mobilization and Deployment, Volume 1

J. D. Gunzenhauser; Colonel, Medical Corps, US Army; Preventive Medicine Staff Officer, Office of the Surgeon General, 5109 Leesburg Pike, Suite 684, Falls Church, VA 22041

INTRODUCTION

The control of communicable disease in basic training or boot camp represents one of the greatest achievements of military medicine. The magnitude of this accomplishment is difficult to comprehend without first-hand experience of camp-based epidemics or extensive study of the lessons of medical history. This chapter provides a brief outline of the fascinating story of how this achievement has been accomplished. The technological tools and administrative controls that exist at basic training centers today combine to form an efficient, elegant approach to safeguarding the health of military recruits. An appropriate regard for time-worn lessons is the proper starting point for future efforts to raise health status. This chapter will highlight those lessons so that future efforts to minimize the threat of communicable disease to basic training populations can be successful.

Outbreaks of communicable disease at basic training installations have riveted the nation's attention. Not simply another national news item, these incidents have brought terror into the hearts of Americans. Typhoid fever during the Spanish-American War, influenza during World War I, scarlet and rheumatic fever during World War II, and meningococcal disease during the Vietnam War these are a few notorious examples. The absence of significant outbreaks during the past 15 years is evidence of the success of control programs.

The control of communicable diseases in basic training is important for many reasons. Among these is the value of health maintenance to the individual recruit and the benefit of his or her optimal health to the initial training effort. [Note: rather than referring to "basic trainees," "airmen basic," or "soldiers, sailors, airmen, and marines," "recruit" will be used to describe a new members of any of the four services.] The value of sustained health to the individual trainee is paralleled by the tremendous savings accrued by the military services through avoidance of retraining and additional recruitment. Another important reason for disease control is to minimize the potential of communicable disease spread to civilian populations. This is a major public health issue, as well as an item of political interest. Such concern resulted in the suspension of basic training at Fort Ord, Calif, in 1964.¹ Likewise, concern about community spread of measles and rubella was an important factor that led to routine recruit immunization against these infections.² On a grander scale, the National Immunization Program of 1976, which aimed to vaccinate all Americans against swine influenza, was initiated following an outbreak in Army basic trainees at Fort Dix, NJ.³ Communicable disease control also enables military personnel to progress rapidly to more advanced training and immediate deployment, if necessary. Influenza outbreaks during World War I ravaged the health of large cohorts of soldiers who were scheduled to deploy to the front lines; this resulted in a need to reorganize and reconstitute units.⁴ An ability to train and rapidly deploy large numbers of military personnel may be critical in future military campaigns. A final reason to minimize disease among training populations is to enable the service medical departments to be as efficient as possible in providing health service support to deployed forces: healthy trainees need fewer medical resources, resources that can be used by those on the front lines. These reasons underscore the importance of communicable disease control in recruit populations.

A PERSPECTIVE ON THE CONTROL OF COMMUNICABLE DISEASE

Programmatic aspects of communicable disease control are best viewed in terms of modes of transmission.^{5,6} This viewpoint contrasts with the traditional medical curriculum, which normally assumes the perspective of agent taxonomy or organ system involvement. The major modes of disease transmission are the airborne, direct-contact, waterborne, foodborne, vector-borne, blood-borne, and sexual contact routes. Since the last two categories do not pose a substantial risk to today's basic trainee populations, they will not be discussed here. Exhibit 9-1 lists the major modes of transmission and their associated agents of communicable disease. All these modes of transmission have played a major role in epidemics at basic training installations. A few examples will highlight the breadth of this spectrum.

In 1898, typhoid fever epidemics at numerous camps in the United States severely disrupted the operational ability of many commands during the Spanish–American War. Nearly 100 regiments were affected, with an average of more than 200 cases per regiment and a case-fatality rate of 7.6%. The Reed-Vaughan-Shakespeare Typhoid Board determined that person-to-person transmission through direct contact, as well as fly-borne transmission, played a more important role in the outbreaks than

EXHIBIT 9-1

ROUTES OF TRANSMISSION AND THEIR ASSOCIATED AGENTS OF COMMUNICABLE DISEASES

Airborne

Mycobacterium tuberculosis Neisseria meningiditis Influenza viruses Measles virus Varicella-zoster virus Other viruses

Direct Contact

Influenza virus Adenovirus Streptococcus pyogenes Neisseria meningitidis Cold viruses Streptococcus pneumoniae Mycoplasma pneumoniae Chlamydia pneumoniae

Foodborne or Waterborne

Salmonella typhi Hepatitis A virus Salmonella species (non-typhi) Agents of food poisoning Shigella dysenteriae and other Shigella species Vibrio cholerae Campylobacter jejuni

Vector-borne

Yellow fever virus Dengue viruses Plasmodium species Borrelia burgdorferi Rickettsia rickettsii Ehrlichia chafeensis

the waterborne route. The outbreaks were so severe that the US Congress responded after the war by appointing a commission to investigate. The Dodge Commission severely criticized both the Medical Department and the War Department, eventually leading to a reorganization of the Army. Compulsory vaccination against typhoid fever beginning in 1911 and improvements in camp sanitation dramatically reduced its occurrence in subsequent mobilizations.⁷

Tuberculosis was a major health problem for the military during World War I. Thousands of soldiers were hospitalized and more than 2,000 died. Although a substantial effort was made to bar from enlistment all individuals with evidence of pre-existing infection, approximately 5,000 with unrecognized active disease and up to 15,000 with radiologically detectable tuberculous infection were accepted into service. The crowding of basic training afforded prime conditions for transmitting infection from active cases to other recruits and also contributed to the total morbidity and mortality of that period.⁸ Roentgen examination instituted during World War II resulted in reduced tubercular disease rates during that war.⁹

Disease caused by Streptococcus pyogenes exacted a terrible toll on military forces during World War II. Coburn and Young estimated that 21,209 naval personnel developed rheumatic fever during the war;¹⁰ 83% of cases occurred within the continental United States (ie, were associated with initial training). The comparable figures for the Army were 18,339 and 77%, respectively.¹¹ The highest rates of streptococcal disease incidence occurred at the naval training center at Farragut, Idaho, where 2.2% (2,084 cases) and 10.4% (9,589 cases) of military personnel, recruits and cadre were hospitalized with rheumatic fever and scarlet fever, respectively, from 1943 through 1945.¹⁰ In the Army, the highest rates of rheumatic fever were reported at Fort Warren, Wyo, where approximately 5% of soldiers were hospitalized with rheumatic fever during 1943.11 Shortly after the end of the war, studies in military populations demonstrated the effectiveness of penicillin in controlling these types of outbreaks.

These three examples demonstrate subtle, important aspects of the interrelatedness of disease control efforts. The threat of typhoid fever, which was so devastating during the Spanish-American War, was eventually eliminated by development of an effective vaccination program and by general improvements in sanitation. While the vaccination strategy was unique to the military population, the sanitary improvements were largely the by-product of improving sanitary conditions across the United States. The eventual reduction in tuberculosis incidence may also be attributed to general improvements in the health of the nation as a whole. Yet, while these general improvements in the larger society undoubtedly contributed to the reduced incidence of typhoid fever, they appear to have had no or little impact on the occurrence of tuberculosis during World War I, which was then still a universal infection by the age of 20 and remained the nation's leading cause of death. Hence, the military relied on screening procedures to minimize the number of enlistees with active disease. Unfortunately, this approach was ineffective. In addition to the substantial improvements in the quality and completeness of tuberculosis screening procedures between the two world wars, improvements in nutrition and the standard of living in the United States also favorably affected the threat of this disease. But the same improvements in sanitary conditions that eliminated the threat of direct-contact transmission of typhoid fever during the Spanish-American War and helped minimize the tuberculosis problem did not prevent the transmission of S pyogenes during World War II. To this day, environmental control measures (ie, interventions that reduce exposure to the agent) have had little impact on communicable infections that are spread primarily through direct-contact or airborne modes of transmission. These few illustra-

tions demonstrate the interrelationships of communicable disease control efforts in the military and the United States as a whole.

These examples also show several general but critical aspects of disease control. First, communicable disease outbreaks occur with greatest frequency and impact during periods of mobilization. Second, the highest attack rates occur in unseasoned personnel, especially those in the earliest weeks of initial training (ie, basic training, boot camp). Third, during the period of mobilization for a particular campaign (even one of several years' duration), there is not sufficient time to develop means to control large outbreaks of previously unrecognized communicable disease threats. And fourth, current capacities to prevent disease outbreaks involve many components and are based on lessons from earlier periods and benefits accrued from general improvements in the larger community. These aspects will be discussed in the sections that follow.

PREVENTIVE INTERVENTIONS RELATED TO MODE OF TRANSMISSION

Preventive interventions for communicable diseases may target environmental reservoirs, transmission of agents from reservoir to host, or aspects of the agent-host interaction. Interventions may be classified as agent-specific if they target a single microorganism or as agent-generic if they affect multiple organisms. Agent-generic interventions make up much of what has come to be known as the "sanitary revolution" of the late 19th and early 20th centuries and have had a tremendous impact on the health status of all Americans, including trainees. In addition, numerous agent-specific interventions constitute a major portion of current communicable disease control programs in basic training centers.

Exhibit 9-2 lists some of the interventions in use today. For each agent and for each mode of transmission, elimination of the reservoir and immunization of the host—the trainee—are possible approaches. Thus, eradication of smallpox and vaccination against typhoid fever largely eliminate the need to consider how those organisms are transmitted, at least within the basic training environment. Between reservoir elimination and host immunization, however, exists a range of strategies linked to the mode of transmission. Most of these intermediary strategies can be classified as either environmental sanitation, "vector" reduction, or barrier approaches.

In developing and reviewing control programs, depth must be emphasized. The goal in disease control is not merely to identify and implement one effective strategy; rather, it is to implement sufficient layers of prevention so an adequate safety net exists in case one approach fails. Multiple preventive layers should exist for each agent and for each mode of transmission.

Airborne

Airborne contagion is distinguished from directcontact transmission in that the former involves infective organisms suspended in air while the latter involves either immediate contamination of susceptible hosts or secondary transmission by "vectors" (ie, fomites). Of course, infections transmissible through airborne contagion may also be transmitted by direct contact, but environmental or host factors normally dictate a predominant mode. The suspended, infective vehicle of airborne contagion is the "droplet nuclei." Particles with a diameter between 0.1 µm and 50 µm are capable of suspension in air; the lower limit on the size of droplet nuclei is limited by the size of the organism itself.¹² Larger nuclei (10 µm to 50 µm in diameter) will fall to the ground relatively quickly. A 10 µm nuclei, for example, will fall the height of a room in 17 minutes.13

When inhaled, most nuclei larger than 5 μ m in diameter deposit in the upper respiratory tract, while smaller nuclei deposit primarily in the lower respiratory tract. The ability of infectious organisms to be transmitted via this route is a function of the organism's accessibility to sites in the infectious host where droplet formation occurs, the stability and size of suspended particles, and the ability of suspended

EXHIBIT 9-2

PRIMARY APPROACHES TO DISEASE CONTROL BY ROUTE OF TRANSMISSION

Universal Approaches

Reservoir elimination Environmental sanitation Continuous Intermittent Vector reduction^{*} Barriers Reservoir-proximate Host-proximate Host immunization

Airborne

Quarantine Agent removal Agent inactivation Agent dilution Barriers Host immunization

Direct Contact

Reservoir removal (quarantine) Environmental sanitation Personal hygiene Personnel dispersion Bunk spacing and orientation Masks Host immunization

Foodborne

Reservoir elimination Agent removal (filtration) Agent inactivation Restriction of sources Proper waste disposal Host immunization

Waterborne

Reservoir elimination Agent removal (filtration) Agent inactivation Restriction of sources Proper waste disposal Host immunization

Vector-borne

Reservoir elimination Vector elimination Barrier protections against the vector Host immunization

Including such "vectors" as fomites

organisms to remain viable over time. Bacteria, fungi, and human by-products that contain viruses (eg, products of coughing or sneezing, fibers, or fragments of desquamated skin) when expelled into the air form droplet nuclei. Slight drafts or other air disturbances help such nuclei remain suspended for indefinite periods of time.

Airborne infections are a significant threat to the health of basic trainees. Influenza, multidrug-resistant tuberculosis, and varicella are notable concerns. In spite of considerable efforts to develop interventions against all airborne infections, virtually all preventive efforts in place today are agent-specific. There has been, for example, considerable effort to assess the contribution of crowding to the incidence of airborne infections.^{14,15} John Shaw Billings, a distinguished Army physician of the 19th century, felt so strongly about the relationship of crowding to airborne infections that he disseminated a circular, stating "every man should have his sixty feet of floor space as much as his ration."14p419 Designing epidemiological studies to assess the impact of crowding on respiratory disease rates is challenging, though, because "crowding" is a time-variant variable that is difficult to measure and interacts with agent endemicity (ie, if the influenza virus is not present, no one will get the flu, no matter how crowded it is). Despite considerable investigative effort, only scant evidence exists that crowding contributes to respiratory disease rates. Nonetheless, in 1943 the US Army adopted a standard requiring 50 square feet of space for each recruit in reception centers.¹⁴

The role of ventilation on incident infections has also been the subject of considerable study. While evidence exists that shows that ventilation pathways are associated with observed patterns of disease,^{16–18} it is less clear to what extent variations in ventilation flow or in the amount of outside air introduced into a closed environment affect the magnitude of incidence rates. Brundage and colleagues¹⁹ measured an association of modern, energy-efficient barracks with increased rates of respiratory disease. Before the institution of year-round adenovirus and influenza vaccination programs, trainees housed in energy-efficient barracks had a 50% greater risk of respiratory infection compared to trainees living in older, less tightly sealed barracks. Because other potentially confounding variables (eg, crowding) were not measured in the study, the authors were reluctant to endorse a causal relationship.

A variety of techniques have been attempted in the past to interrupt the airborne route of transmission. Quarantine procedures, such as the removal of affected individuals through mandatory hospitalization policies, have been used at basic training installations for many years. The effectiveness of these isolation procedures depends on a variety of factors, including the agent-specific duration of asymptomatic shedding, the willingness of affected trainees to report for medical evaluation, the logistical ease of reporting for evaluation, and the degree to which medical authorities adhere to hospitalization policies.

Studies have also been conducted to assess the effectiveness of procedures to inactivate or remove infectious agents from the environment. Wells²⁰ demonstrated that indoor ultraviolet irradiation has a modest effect in reducing infection rates in various populations. The recent emergence of multidrugresistant Mycobacterium tuberculosis has prompted the Occupational Safety and Health Administration to recommend the use of ultraviolet irradiation as an adjunct in controlling the transmission of respiratory pathogens in health care facilities. Others²¹ have reduced the frequency of nosocomial infections through the use of laminar airflow systems that incorporate filters capable of removing most microorganisms. Some advocates²² recommend the development of techniques using small air ions to reduce the viability of suspended microorganisms. None of these approaches, however, has been applied or studied in military trainee populations.

During World War II, the Commissions on Acute Respiratory Disease and Air-borne Infections investigated the effect of oiling floors and bedding on acute respiratory infections.²³ These experiments were based on the concern that bedding or floor dust might serve as a "reservoir" for hemolytic streptococci that, when disturbed, could become airborne. Floors were treated using a petroleumdistillate floor oil, distributed through buckets with perforated bottoms, and spread with hair brooms.²⁴ Oiling of bedding (ie, blankets, sheets, pillowcases, and mattress covers) consisted of adding an oilemulsion base during the rinse phase of machine laundering and resulted in an oil loading of 2% to 4% in the fabric.^{25,26} General enthusiasm for these procedures was reported from participants due to the absence of dust and the reduced amount of work to maintain barracks cleanliness. In fact, the intervention was so popular that requests for the procedures were received from others not included in the study. Except for occasional staining of feet or clothes with oil, no complaints were received. During periods of endemic disease, this program reduced infection rates by 30% to 40%, but during epidemic periods rates were only reduced 6% to 12%.¹ For reasons that are not clear, the commission did not advocate this method as a means to control disease

rates. Because the commission presumed that *S pyogenes* was transmitted through the airborne route, it is possible that these studies underestimated the effect that oiling procedures may have on true airborne infections. This or other similar modes of intervention may warrant further evaluation.

Concerns over indoor air pollution and the effect that "tight" buildings may have on respiratory disease have resulted in several proposals and actions. Dilution of "polluted" indoor air may be accomplished by mixing with outside air. The American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) publishes ventilation standards for indoor air quality. The standards address microorganisms as well as various gases, vapors, smokes, and other particulate contaminants. Most recently revised in 1989, the ASHRAE standard now requires 15 cubic feet of outside air per minute per person for "dormitory sleeping areas."27 Before this revision, the requirement was only for 5 cubic feet per minute. The extent to which existing recruit barracks conform with the revised ASHRAE standard has not been comprehensively evaluated, and current military policy continues to emphasize minimal square footage requirements.

Control of airborne infections in basic training populations relies almost exclusively on agent-specific interventions. While none can deny the tremendous effect that these interventions have had, the health of trainees is vulnerable to a breakdown in any single strategy or to the emergence of new airborne agents of disease. The vulnerability of this posture was demonstrated in 1989 when a measles outbreak at Fort Leonard Wood, Mo, followed a delay in normal vaccination procedures until several weeks into the training cycle. The simple delay in vaccinations provided a sufficient "window" for a limited outbreak to occur among non-immune recruits. The agent-generic strategies the military has in place to control airborne infections (eg, requirements for segregation of sick trainees, minimal square footage of living space) can be expected to have at most a modest effect on limiting disease.

Direct Contact

Infectious organisms that can be transmitted through direct contact include many that have caused large epidemics in trainee populations in the past: adenovirus, influenza viruses, *S pyogenes*, and *Neisseria meningitidis*. Others in this group that cause respiratory infections include the common cold viruses, *S pneumoniae*, *Mycoplasma pneumoniae*, and *Chlamydia pneumoniae*. Other agents of disease, such as those that cause airborne or gastrointestinal infections, may also be transmitted through the direct contact route.

Direct-contact transmission may occur through several mechanisms. Sneezing, coughing, spitting, talking, and even normal breathing project microorganisms into the air²³ in the form of infectious droplets. These droplets are usually much larger in size than the droplet nuclei of airborne contagion, fall quickly, and come to rest on objects in the immediate environment. Susceptible persons become infected if the organisms enter directly into the body through portals such as the eye, nose, or mouth. Alternatively, viable organisms on the surface of environmental objects (fomites) may serve as a source of secondary, hand-inoculated infection. Direct-contact transmission may also result from physical contact between persons in either a primary (eg, kissing) or a secondary (eg, hand-shaking) mode.

The basic training environment (see Chapter 8) provides countless opportunities for direct-contact transmission. In fact, it is difficult to imagine conditions more conducive to contagion: crowding, groups containing individuals from diverse geographic locations, mandatory and continuous concentration of personnel, and vigorous activities involving physical contact both with other persons and with objects in the environment.

Control of infections transmitted through direct contact is similar in many aspects to the control of airborne infections. Reliance on vaccination is the primary mode of intervention for most organisms in this class. Year-round influenza immunizations appear to be very effective at preventing large outbreaks, such as those that have occurred in the past. Vaccines were developed to protect trainees from the tremendous morbidity of adenovirus infections and the risk of mortality from meningococcal disease. In contrast, outbreaks of S pyogenes infections have been controlled through the broad use of penicillin. Other agents of disease in this category have contributed to trainee morbidity in the past but have not caused repeated outbreaks over time or been associated with substantial mortality.

Quarantine of affected individuals with conditions transmissible by direct contact is normally routine and frequently mandatory. US Army policy, for example, has required the hospitalization of any trainee with an influenza-like illness, a temperature of 38°C or higher, and one or more respiratory symptoms. The degree to which these procedures are implemented determines the overall effectiveness of the policy. In addition, adherence to minimal square footage requirements in barracks areas probably decreases case reproduction rates. Any measure that increases the distance between trainees could theoretically reduce the likelihood that infectious droplets expelled during a sneeze or cough would fall on and infect a susceptible person. Head-to-foot arrangements of bunks in sleeping quarters, for example, could have such an effect. One study²⁸of the effect of doublebunking (ie, using bunkbeds) on the incidence of respiratory disease noted a 50% reduction in cases of acute respiratory disease (ARD) (excluding influenza, atypical pneumonia, and hemolytic streptococcal infections) in the intervention group in comparison to a control population.

Other measures of prevention are theoretically possible. Direct transmission of infectious droplets from one individual to another could be minimized, for example, through the use of masks, by dispersion of personnel, or by separation of symptomatic personnel from others. During the influenza epidemics of 1918 and 1919, several communities in the United States adopted programs of mask-wearing. There is some evidence that these programs had a modest effect.⁴ Secondary transmission through fomite contact could be minimized through intermittent sterilization of environmental surfaces. Ultraviolet irradiation of air spaces or environmental surfaces, especially when personnel are absent, could be an efficient strategy in certain situations. Alternatively, surfaces might be developed that have inherent anti-microbiological properties. Finally, disease transmission rates could be reduced through changes in behavior. Mandatory, frequent handwashing and training to reduce contact between hands and facial portals of entry could have some effect.

In contrast to these potentially beneficial practices, one common practice probably has no effect. Often, as rates of respiratory disease increase, local personnel will open barracks windows and doors to increase ventilation. Because the most commonly encountered pathogens are transmitted through direct contact rather than airborne transmission, increased ventilation has no effect on this mode. It is conceivable that "fresh" air sterilizes the environment through an unspecified mechanism or that "fresh" air somehow enhances natural host immune defenses. However, these conceivable benefits have not been substantiated by trials.

Foodborne and Waterborne

Little information will be provided here on the threat of foodborne and waterborne infections to trainee populations. Other chapters in this volume address these diseases in detail (see Chapter 37, Diseases Spread by Food, Water, and Soil). Nonetheless, these conditions are of historical importance in the trainee environment. Intestinal infections were a horrendous problem in training camps before the sanitary revolution. Typhoid fever outbreaks during the Spanish-American War are the most notable example. Diarrheal diseases were also a common affliction that had a severe impact on training.

With the exception of typhoid fever, virtually all conditions in this foodborne and waterborne illness category are prevented through intermediary, agent-generic measures. These include policies to guarantee the procurement of safe water and food; routine inspection of food service facilities to ensure proper handling, preparation, and storage of food items; sanitary procedures for disposal of all forms of waste; and emphasis on handwashing and other personal hygiene measures. The success of these sanitary procedures to affect foodborne and waterborne disease transmission stands in stark contrast to the failure of all these approaches to affect transmission of airborne and direct-contact diseases.

Of course, sporadic outbreaks of foodborne or waterborne infections may occur in trainee populations whenever lapses occur in preventive strategies. The universal susceptibility of trainees to most agents of foodborne and waterborne disease and an inherent potential for diseases transmitted by this

Control of communicable disease does not rely solely on the continuous implementation of proven strategies. It also requires a vigilant watchfulness for breakdowns in control practices and the emergence of previously unrecognized threats through the implementation of a comprehensive surveillance program.

A surveillance program for communicable diseases will have several goals. The first is, of course, to verify that prevention strategies are effective. While assuring the health of individual trainees, this goal also maintains accountability of the medical department to the public interest. A corollary of this goal is to identify outbreaks as soon as possible so that immediate corrective actions may be taken. Disease control programs should be designed in layers, with a pre-planned capability to add layers of prevention in response to an increased threat of disease. Another goal of surveillance is to trigger investigations that may identify risk factors, causes, and possible control strategies. While formal investigations are usually initiated only after one or more control strategies have failed, such investigations

mode to affect many persons underscore the significance of these conditions. Thus, while the threat of foodborne and waterborne outbreaks may be small in comparison to the threat of diseases transmitted via other modes of transmission, it is imperative that the disease control officer emphasize procedures to prevent their occurrence.

Vector-borne

Other chapters in this volume have detailed discussions of the issues related to conditions in this category. While serious vector-borne infections, such as malaria, dengue fever, and yellow fever, were major problems in years past, their threat to current military trainee populations is not considered serious. These infectious agents were eliminated from the continental United States through vector control programs. Current trainee populations benefit from this situation, but re-establishment of viable vectors has occurred and warrants ongoing surveillance. Other vector-borne conditions, such as Lyme disease, Rocky Mountain spotted fever, and ehrlichiosis, are endemic in certain training locations. Successful control of vectorborne disease will, therefore, depend on avoiding re-introduction of previously eliminated organisms, as well as using personal protective measures.

SURVEILLANCE FOR COMMUNICABLE DISEASE

often identify shortcomings in these attempts or identify other alternatives for control that have not been considered. Another purpose of surveillance is to define disease trends so that resources may be efficiently distributed. Changes in the distribution of resources may be required both to meet the clinical health care needs of affected personnel and to assure disease prevention programs. A fourth goal of surveillance is to monitor the relative morbidity of various conditions so that prevention research can target those that cause the greatest problem. The ability of research and development agencies to revise and focus their efforts on conditions of greatest significance to the military community depends on their having current information concerning the health threat. All of these goals must be borne in mind when designing and evaluating surveillance systems.

Potentially, communicable disease surveillance programs may monitor any or all of the following elements: hazardous agents in the environment, exposures to those agents, and adverse health outcomes resulting from those exposures.²⁹ In addition,

surveillance programs should collect and archive detailed information on potentially affected populations. Military basic training installations currently conduct only rudimentary surveillance programs. The Air Force conducts limited influenza surveillance in basic training, with collection of throat washings for viral cultures. Neither the Navy nor the Army performs routine environmental monitoring to identify circulating agents of respiratory disease. In the absence of information about which agents are present, it is impossible to monitor potential exposures of individual trainees to those agents.

All military services require medical personnel to report selected infectious conditions, including many that are of concern during basic training. In addition, personnel at Navy and Marine Corps training centers monitor total counts of positive cultures for *S pyogenes*. Personnel at Army basic training installations monitor trainee segregations for infectious respiratory conditions and laboratoryconfirmed infections with *S pyogenes* among hospitalized trainees. Information on potentially exposed trainee populations is usually collected in aggregate form and does not normally provide more detail than the number and sex of trainees assigned to each company-sized training unit.

The current Army ARD surveillance program is an offspring of the now defunct adenovirus surveillance program.³⁰ In the early 1980s, the program was modified to collect specific information on streptococcal infections. A key component of the altered program has been the requirement to segregate trainees with an influenza-like illness, fever in excess of 38°C, and any respiratory symptom. Before the 1990s, this was usually on ARD wards, but with the arrival of managed care, cases are now often segregated in less-formal housing arrangements. Each week, surveillance personnel collect the following information for each training company: the number of recruits in training by sex, the number of trainees segregated with acute respiratory infections, the number of segregated trainees with at least one culture obtained for S pyogenes, and the number of positive throat cultures. These numbers are the building blocks for three indices, which are calculated each week (Table 9-1).

The ARD rate is a general measure of respiratory disease activity in trainee populations. The term "acute respiratory disease" as applied here is distinct from the term "acute respiratory disease of recruits," which from the 1940s to the 1970s applied to adenovirus infections.^{30,31} ARD now means any acute, febrile condition primarily involving the respiratory system. As recently as 1986, the nominal epidemic threshold for the ARD rate was 2 hospitalizations per 100 trainees per week. When persistently low hospitalization rates were observed at all installations, the threshold was lowered to 1.5.

TABLE 9-1

MEASURES OF .	ACUTE RESPIR	RATORY DIS	EASE ACTI	VITY AT US	5 ARMY BASI	C TRAINING
INSTALLATION	[S					

Rate or Index	How Calculated	Threshold
ARD rate	100 x HOSP / POP	1.5
Streptococcal recovery rate	100 x GABHS / CULT	None [*]
Streptococcal-ARD surveillance index	ARD rate x streptococcal recovery rate	25

ARD: Acute respiratory disease

POP: The total number of individuals in the basic training population

HOSP: The number of trainees segregated with acute febrile respiratory conditions[†]

CULT: The number of throat cultures performed on hospitalized trainees

GABHS: The number of throat cultures positive for group A beta-hemolytic streptococci

^{*}During outbreaks of virulent streptococcal disease, this rate often exceeds 50%. However, such high recovery rates may also be observed during periods of infrequent hospitalizations or during periods of hyperendemic infections with nonvirulent organisms. Therefore, this "rate" should never be used alone as a measure of streptococcal disease.

[†]Hospitalizations with any of the following diagnoses at the time of admission: acute respiratory disease, streptococcal pharyngitis, influenza-like illness, tonsillitis, upper respiratory infection, bronchitis, acute pharyngitis, pneumonia, peritonsillar abscess, retropharyngeal abscess, bacterial meningitis, mononucleosis, sinusitis, mycoplasma, otitis media, chickenpox (or varicella), or acute viral syndrome or illness. Interpretation of ARD rates for groups of trainees smaller than the entire installation is problematic. Although the origins of large outbreaks can usually be traced to individual units, most elevations of ARD rates in less-than-installation-sized groups usually resolve spontaneously. For this reason, interpretation of the ARD rate for small-sized units is not recommended. Before the use of adenovirus vaccines, weekly ARD rates approached 10 per 100 during peak epidemic periods.³⁰ Following the initiation of year-round adenovirus 4 and 7 vaccination, ARD rates normally did not exceed 1 and only rarely approached or exceeded 2.³²

Although the ARD rate is sensitive to outbreaks of adenovirus and influenza, it is a poor indicator of virulent streptococcal disease activity. This was demonstrated during an outbreak of acute rheumatic fever (14 cases) at Fort Leonard Wood in 1987 during a period when ARD rates remained well below the epidemic threshold.³³ Similar observations were made during subsequent outbreaks at other Army installations.³⁴

In 1959, the Armed Forces Epidemiological Board suggested that weekly streptococcal pharyngitis rates in excess of 10 cases per 1,000 trainees may serve as an indicator of an increased risk for acute rheumatic fever.³⁵ The Navy applied this criterion in determining the need to reinstitute benzathine penicillin G prophylaxis at the Naval Training Center in San Diego, Calif, in 1987.³⁶ In contrast, the Army monitors the streptococcal-ARD surveillance index (see Table 9-1). This index includes a criterion for morbidity (defined as hospitalization) and, as such, is theoretically less susceptible to false alarms resulting from hyperendemic infections with nonvirulent strains and variations in screening behaviors of health care providers. In practice, either measure of streptococcal disease activity is sufficient as a tool for initiating penicillin prophylactic programs, especially when elevated rates are accompanied by independent evidence of virulent strain circulation in the trainee population (ie, cases of rheumatic fever or invasive sequelae). In some situations, penicillin prophylaxis may be indicated simply to prevent morbidity associated with streptococcal pharyngitis.

The ARD rate and either of these measures of *S* pyogenes activity are efficient indicators of outbreaks. In particular, because both adenovirus and streptococcal disease outbreaks require several weeks to reach peak activity and may extend over several months, sufficient time is normally available to interrupt an epidemic once the threshold is passed. In contrast, influenza epidemics start, reach their peak, and begin to subside within a 1- to 2week period. The rapidity with which influenza can pass through a large population is not generally recognized. This contrast is demonstrated in Figure 9-1. For the purposes of influenza control, the currently implemented surveillance systems are only useful in that they may serve as sentinel indicators for unaffected installations.

CONTROL OF SPECIFIC DISEASES

This section builds on concepts already presented by discussing a handful of diseases that are of greatest concern today or for which unique control programs have been developed in the past. Fortunately, preventive strategies that affect the pathway along a particular mode of transmission are likely to affect more than one disease. Current environmental sanitation and personal hygiene practices have minimized waterborne, foodborne, and vector-borne infections to a level where they are not considered a substantial threat to the health of trainees. In contrast, virtually no effective agent-generic strategies have been developed for airborne and direct-contact contagion. All of the conditions discussed below fall into one of these two categories. Because this discussion highlights programmatic aspects of disease control, the reader is referred to Chapter 38, Diseases Spread by Close Personal Contact, for more information on the diseases themselves.

Influenza

The impact of influenza on US military operations during World War I was devastating. During 1918, over 43,000 members of the military died from influenza or pneumonia: 38,000 in the Army and over 5,000 in the Navy. These totals do not include deaths in 1919 for which statistics are somewhat less reliable. Of course, the tragedy wrought by influenza in US military forces was just a small part of the worldwide epidemic, in which up to 25 million died. In the United States, over 650,000 died during the two years 1918 and 1919.⁴

The rapidity with which influenza strains spread across the globe not only is a defining characteristic but also may be an important determinant of virulence enhancement. The rapid changes in the global distribution of influenza viruses were evident in the military population during World War



Fig. 9-1. These two graphs demonstrate the condensed epidemic curve of influenza virus outbreaks when compared with streptococcal-associated respiratory disease outbreaks. Graph **a** shows that an outbreak of influenza occurring at Camp Funston, Ks, in 1918 occurred during a 3-week period in March. In contrast, Graph **b** shows a rheumatic fever–associated outbreak of streptococcal disease among trainees at Fort Leonard Wood, Mo, during 1987 to 1988 that extended over a multimonth period (September 1997 to February 1988). Because of the extended duration of streptococcal-associated outbreaks, control programs can rely on surveillance and prompt intervention as an effective strategy, whereas the short duration of influenza outbreaks requires an emphasis on primary prevention (eg, vaccination) or prior preparation (eg, prepositioned medications).

Sources: (a) Opie EL, Freeman AW, Blake FG, Small JC, Rivers TM. Pneumonia at Camp Funston. *JAMA*. 1919;72:114. (b) Centers for Disease Control. Acute rheumatic fever among Army trainees—Fort Leonard Wood, Missouri, 1987-1988. *MMWR*. 1988;37:519–522.

I. An epidemic of non-lethal influenza affected military units in the continental United States during the spring of 1918. This original, less-virulent strain of virus subsequently spread around the world before being reintroduced into the United States in a more virulent form in the late summer. Almost instantaneously, the most-lethal form of influenza erupted at several sites around the world in September 1918. It exacted a horrible toll in human lives and misery for several months before beginning a prolonged, dwindling spiral that lasted into the second half of 1919. The fact that this form of influenza affected persons in the prime of life and that US forces in the continental United States were affected to a greater extent than those overseas ensured that trainee populations were among those most greatly affected. At Fort Devens, Mass, for example, up to 60 trainees died each day during the peak of the epidemic.

While many important questions concerning the epidemic of 1918-1919 remain unanswered, the effectiveness of current disease control practices can be interpreted in light of recent observations. Universal immunization of all military personnel with influenza vaccine is a Department of Defense policy. While seasoned personnel normally are vaccinated annually during the fall months, recruits receive the immunization in their first week of training, no matter what time of year. "Mass" prophylaxis of the entire force each year and continuous "tandem" prophylaxis of accessions results in a population that probably experiences the lowest age-adjusted attack rates in the world. The effectiveness of this approach, though, depends on the degree to which influenza vaccine components protect against circulating strains. The near complete absence of outbreaks of influenza in US armed services personnel during recent years³⁷ indicates that this strategy has been very effective. This success is probably attributable to a variety of factors. World Health Organization procedures for selecting strains for inclusion

in vaccine products have been remarkably accurate. Studies by Meiklejohn and colleagues^{38,39} at Lowry Air Force Base, Colo, have documented that vaccines produced in industrial quantities for use in the elderly and other high-risk populations are highly immunogenic in military trainee populations. And notwithstanding the great number of deaths influenza continues to cause in susceptible populations, no recent strain of influenza has had a level of virulence comparable to that which ravaged the military force during World War I.

If a pandemic of influenza caused by a strain similar to the one that circulated in 1918 recurs, it is doubtful that current disease control strategies could provide much protection for military trainees. As previously discussed, no preventive strategies are currently in place that can interrupt the airborne or direct-contact modes of transmission, both of which are important routes for influenza.³⁹ Currently produced vaccines have low overall vaccine efficacy rates (70%-80%) and would not provide a substantial herd immunity barrier40 against the spread of a virulent, highly transmissible strain. Even if strategies such as mask-wearing and environmental sanitation (eg, ultraviolet radiation) could provide protection, the absence of current materiel, training, and policy to direct these activities assures they will have a minimal role during early phases of a large-scale outbreak. One currently circulating proposal suggests stockpiling amantidine or rimantidine for use at basic training installations in the event of a large outbreak. Improvements in vaccine efficacy rates and reduced vaccine production times may be feasible in the near future. Such improvements in plans and technology could reduce the impact of a pandemic.

The National Influenza Immunization Program of 1976 highlights many of the difficulties that an effort to prevent a "killer flu" epidemic would entail. The origins of that effort^{41–45} and the events that followed⁴⁶ (including cases of Guillain-Barré syndrome^{47,48}) have caused many to reconsider how such national decisions are made.⁴⁹ The potential political liability of making an incorrect decision may make future efforts to respond to such a national threat extremely difficult. Nonetheless, efforts are in progress to outline such a strategy for the nation.⁵⁰

Adenovirus

Adenovirus infections in military populations are of tremendous significance. Illness caused by adenovirus is characterized by high attack rates of a short-term, febrile, debilitating illness. Studies have shown that adenovirus types 3, 4, and 7 (and less commonly types 14 and 21) are the primary cause of febrile, acute respiratory disease in military trainees.^{30,31,51,52} This organism was a nearly ubiquitous cause of outbreaks during the fall and winter seasons at basic training installations before the development of effective vaccines.⁵³ Typically, 50% or more of the entire trainee population would acquire this infection during the first few weeks of training, and most would be hospitalized. Thus, adenovirus infections took a heavy toll on the health of most trainees and cost the military many dollars in health care requirements, lost time from training, and the need to recycle trainees.

Isolation of the virus in the late 1950s and demonstration that enteric infections could induce protective immunity were milestones in the development of the live, enteric-coated vaccines. While some studies have demonstrated that live virus can be secondarily transmitted from the bowel of the vaccinated to the oropharynx of the susceptible,¹ such transmissions are relatively infrequent and have not been reported as a significant consequence of vaccination. Clinical trials conducted at Army basic training installations in the 1960s^{54,55} demonstrated that vaccines against adenovirus types 4 and 7 were safe and efficacious, and they eliminated most acute respiratory disease in trainees. The tremendous savings afforded by these vaccines was summarized in a cost-benefit analysis performed by Collis and colleagues in 1973.⁵⁶ They showed that use of the type 4 and 7 vaccines at eight Army installations during just a few months in 1970 and 1971 prevented nearly 27,000 hospitalizations.

By the 1990s, all services except the Air Force required vaccination of male trainees within the first few days of accession to military service. Some installations have vaccinated female recruits, but installations that provide vaccine to males alone have not experienced outbreaks of adenovirus among non-immunized females. In 1984 (after observation of summertime cases), the US Army instituted a program of year-round vaccination. Surveillance for emerging strains of adenovirus into the late 1980s demonstrated that the two vaccines were causally sufficient to prevent nearly all adenovirus infections (JDG, unpublished data, 1990). When production of the vaccines by their sole supplier lapsed in 1996, a policy of seasonal administration of vaccines was resumed and continued until supplies ran out in 1999.

The greatest current threat to the continued prevention of adenovirus-associated illness in military trainee populations is the lack of a commercial market for the vaccine outside of the armed services. In contrast to influenza, military problems with adenovirus infections have had little relevance for civilian populations. The sole US producer of adenovirus vaccines has disassembled its production facility and current lots of vaccine expired in the spring of 1997. In 1997 and 1998, the Food and Drug Administration extended the expiration date of existing vaccine lots, allowing vaccination through the 1998-1999 winter season. Despite current concerted efforts to re-establish a production capacity, it is inevitable that vaccines will not be available to protect trainees for a period of at least 2 years and that epidemics will recur.

While the commercial aspects of adenovirus vaccine production are unique among the conditions discussed in this section, the current crisis vividly demonstrates the vulnerability inherent in a communicable disease control program that is agent-specific and one layer thick. The absence of other preventive strategies, in particular the absence of mode-specific interventions for airborne and direct-contact transmission, is a continuing source of vulnerability.

Meningococcal Disease

Disease caused by N meningitidis has a longstanding relationship with the US military.⁵⁷ Notwithstanding recent policies to vaccinate military personnel deployed to parts of the world where meningococcal disease is endemic,⁵⁸ nearly 200 years of observations have demonstrated that meningococcal infections occur predominantly in recruits. In general, the prime determinant of large military epidemics has been mobilization for war, although smaller outbreaks may have been fueled by contemporaneous epidemics in civilian communities. During World War I and World War II, 2,279 and 559 deaths were attributable to meningococcal disease, respectively. The case-fatality rate during World War I was 39%.57 Although disease caused by N meningitidis was the second leading cause of infectious disease death during World War II,⁵⁹ the overall case-fatality rate was substantially reduced through the use of sulfonamide prophylaxis.⁶⁰

While many strains of meningococcus circulate freely among trainee populations, most "carriage" is asymptomatic. In fact, carriage rates among unaffected populations are similar to those observed during epidemics (20% to 80%).⁵⁷ Early suggestions that carriage rates in excess of 20% indicate a high risk of subsequent morbid disease have not been substantiated.⁶⁰ Transmission occurs presumably via respiratory droplets (airborne and direct contact) originating primarily from individuals with asymptomatic carriage. Fomites play a negligible role in disease spread.⁶¹ Transmission among asymptomatic populations is very efficient and rapid, as documented by a 92% culture-positive rate in one study of 99 men over a 68-day period.⁶⁰

The ability of meningococcal strains to circulate widely but cause disease only rarely may cause substantial psychological stress in affected populations. From the community viewpoint, the organism appears to strike randomly at helpless victims, a large proportion of whom succumb quickly. As a result, many community members may wait in fear to see who will be struck down next. This viewpoint infers both that the organism is extremely virulent and that susceptibility to life-threatening disease is high. In fact, both inferences are false. By the time cases occur, a large proportion of the population has probably already been exposed, but only those with the rare (and largely unknown) host susceptibility factors manifest disease. Agent-associated virulence factors undoubtedly exist, but these remain largely undescribed.

Antibiotic prophylaxis and vaccination have been the only two strategies in the prevention of meningococcal disease. From World War II through the early 1960s, strains of *N* meningitidis remained sensitive to sulfonamides. Complete reliance on this intervention and the eventual development of widespread resistance led to an 8-year period (1963–1971) during the Vietnam War era when no effective preventive strategies were available.57 One of the initial outbreaks in this period—which occurred at Fort Ord—resulted in much local hysteria, considerable political pressure, and the eventual suspension of basic training at that installation. In the late 1960s, military investigators demonstrated that rifampin could effectively eliminate carriage, but the widespread development of resistance to this antibiotic limited its usefulness as a tool in outbreak interruption.¹

Efforts in vaccine development intensified and eventually were fruitful. In October 1971, vaccination against serotype C was begun for all trainees; vaccines against serogroups A, Y, and W-135 were subsequently developed and fielded during the late 1970s and early 1980s. Since the development and routine use of the tetravalent vaccine (A/C/Y/W-135), the occurrence of meningococcal disease has become extremely rare among recruits and due exclusively to serogroup B strains.⁵⁷ Several group B vaccines have been developed, but none demonstrates more than partial (approximately 50%) efficacy and all have only investigational new drug status.

As with other airborne infections, no effective prevention strategies have been developed that control meningococcal infections along its route of transmission. Although studies have shown associations of disease incidence with crowding, ventilation, and microclimate, few trials have been attempted to evaluate the effectiveness of modifying these factors.⁶² Efforts attempted during World War I included quarantine, isolation of carriers, reduction of crowding, and increased ventilation.⁶⁰ The practical requirements of mobilizing a million soldiers during a very short period of time limited these attempts. Subsequent investigations focused almost entirely on chemoprophylaxis and immunoprophylaxis.

Nonavailability of a vaccine against serogroup B meningococcus remains a major threat to the health of trainees. The potential for this serogroup to cause substantial outbreaks was demonstrated in the early 1960s. The recent occurrence of large outbreaks of group B disease in other parts of the world highlights this potential threat. There is no logical reason to believe that these strains will not reappear. Furthermore, the causal connection between the fielding of vaccines and the disappearance of meningococcal disease in military recruits has been questioned by at least one prominent authority.⁵⁷ These shortcomings suggest that further inquiry into strategies to interrupt the transmission of meningococcal organisms within trainee populations may be warranted.

Group A Streptococcal Disease

The reemergence of rheumatic fever⁶³ and the identification of a previously unidentified syndrome (toxic streptococcal syndrome)⁶⁴ during the 1980s sparked substantial, renewed interest in *S pyogenes* infections. Increases in the occurrence of many of the known suppurative complications of streptococcal infections were reported from many locations. During this period, outbreaks of group A streptococcal infections occurred within the trainee populations of all military services.^{33,35,37,65} Both the Army and Navy reported clusters of rheumatic fever cases. These outbreaks resulted in increased hospitalizations, substantial morbidity, and even death. Penicillin prophylaxis programs, which had been discontinued years before, were reinstituted to control disease.

Strategies to control streptococcal disease outbreaks in the military derive from the large outbreaks during World War II. As a result of those outbreaks, the armed services conducted numerous investigations to identify methods of disease control.⁶⁶ An early, remarkable victory was the demonstration that treatment of streptococcal pharyngitis with benzathine penicillin G (BPG) within 9 days of the onset of symptoms could prevent the development of rheumatic fever.⁶⁷ This intervention provided dramatic relief for installations where rheumatic fever attack rates after streptococcal pharyngitis approached 5%. However, because S pyogenes strains circulated widely among trainee populations and because many persons with rheumatic fever reported no antecedent episode of pharyngitis, treatment of symptomatic trainees was not effective in controlling outbreaks. Subsequent investigations at the Streptococcal Disease Laboratory at Fort Warren, Wyo, 67,68 and elsewhere 69-⁷² demonstrated that combined mass and tandem prophylaxis could control outbreaks in trainee populations.

Combinations of mass and tandem prophylaxis programs with BPG have been the mainstay of streptococcal disease control programs for the military ever since. Mass prophylaxis in this context consists of administering antibiotics to all trainees on an installation or in an affected group over a relatively short period of time. Tandem prophylaxis consists of routinely administering antibiotics to new cohorts of trainees shortly after arrival. An attempt to contain a broad-based disease outbreak with tandem prophylaxis alone³⁴ demonstrated the non-effectiveness of this approach. Current Army policies direct the administration of the tandem prophylactic dose (when used) within the first few days of arrival at reception stations, while at Marine Corps and naval training centers, this dose is administered on the 17th day. Hyperendemic infections among a Marine Corps trainee population receiving repeated courses of penicillin prophylaxis (on days 17 and 55) led Gray and colleagues to recommend erythromycin prophylaxis for individuals with penicillin allergy.⁷³

The role of surveillance in the early identification of streptococcal disease outbreaks has already been discussed. Both the streptococcal-ARD index (Army) and the proportion of all throat cultures positive for *S pyogenes* (Navy and Marine Corps) serve as sensitive indicators of evolving outbreaks. A continuing difficulty, particularly at Army installations, is the lack of a reliable indicator to signal when prophylaxis programs may be terminated. A recurrent outbreak at Fort Leonard Wood in 1989 following 19 months of BPG prophylaxis demonstrated that absence of streptococcal disease in the local community was not a reliable indicator that prophylaxis could be safely discontinued.³⁴

Although BPG may have benefits that extend beyond the prevention of streptococcal disease,⁷⁴ use of penicillin prophylaxis for disease control is not an optimal strategy. The potential for allergic reactions, the threat of developing resistant organisms, and the logistical burden of providing deep intramuscular inoculations warrant development of alternative control strategies. Although *S pyogenes* organisms have not yet demonstrated true resistance to penicillin, that is no guarantee that such resistance will not develop in the future. Selection of an alternative chemoprophylactic regimen may be problematic. While some suggest that development of a multi-M-type vaccine is a real possibility,^{75,76} other experts believe that will not happen soon.

Efforts to contain the organism within the environment were attempted by investigators in the years immediately following World War II. Unfortunately, blanket- and floor-oiling experiments were predicated on the assumption that streptococci are transmitted through the airborne route. Subsequent investigations showed that direct contact transmission was, in fact, the principal mode. Environmental factors clearly play an important role in ongoing outbreaks of *S pyogenes*. For example, of 6,710 admissions for rheumatic fever reported in the Army during 1943, 43% occurred in the five states of Colorado, Utah, Idaho, Montana, and Wyoming.¹¹ This region of the country is recognized as an area of increased risk for streptococcal infections, but the reasons for this remain unknown.

Current approaches to the control of streptococcal infections suffer from the same limitations of all diseases discussed in this section. Reliance on chemoprophylaxis is agent-specific and at some point is liable to fail. Ongoing problems with streptococcal infections among Marine Corps trainees (eg, pharyngitis and less commonly suppurative sequelae) demonstrate that approaches that implement a single, final barrier within the susceptible host may not succeed.

Other Agents of Communicable Disease

Influenza viruses, adenovirus, *N* meningitidis, and *S* pyogenes are only four of numerous infectious

agents that are a current threat to the health of military trainees. These organisms have had special significance in the past and remain among the most dangerous threats in the near future, but other organisms deserve at least brief mention.

Pneumococcal disease has been a major problem for military populations in the past, particularly in the 1940s.⁷⁷ Moreover, pneumonia caused by *S pneumoniae* is a significant threat to trainee health. Although these organisms were susceptible to penicillin in the past and an effective, multivalent vaccine⁷⁸ is available for long-term immunoprophylaxis, recent difficulties at the Naval Training Center indicate that prevention strategies are not likely to be simple. Reports of increasing resistance to multiple antimicrobials⁷⁹ and continuing outbreaks in closed populations⁸⁰⁻⁸³ are a cause for concern.

Varicella infections continue as an unmanageable problem in recruit populations. Trainees arriving from tropical locations (eg, Puerto Rico) have low

Rates of communicable disease among military trainee populations have been brought to historical lows. Ongoing programs of environmental sanitation prevent the threat of vector-borne, foodborne, and waterborne diseases that plagued Army camps during the 19th century. Vaccinations against influenza, adenovirus, and the meningococcus have minimized the occurrence of these diseases. Nonetheless, the threat of virulent influenza, the lapse in adenovirus vaccine coverage, and the B serogroup "gap" in the tetravalent meningococcal armamentarium remain substantial threats to future trainee health. Similarly, the resurgence of *S pyogenes* infections during the 1990s and the sputtering patchwork approach of administering penicillin to massive populations of trainees suggests that a less-than-optimal strategy of disease prevention is being pursued. Other agents of communicable disease, which have not quite become leading players, loom in the background and are within an arm's reach of trainee wellness.

A currently prevailing attitude of complacency toward the prospect of trainee outbreaks probably has multiple causes. First, there are other aspects of military medicine and modern medicine, in general, that more easily attract and sustain attention and resources. These more-flashy areas compete with the ever-present and rather mundane need to sustain and enhance the health and readiness of that valuable asset: the newly enlisted sailor, soldier, airman, and marine. A second reason for the current lack of initiatives in the control of trainee communicable disease rates of childhood infection and therefore remain susceptible. Since the licensure of a varicella-zoster vaccine in 1995, its use in recruit populations has been endorsed by several services and may soon become a universal requirement for nonimmunes.

Chlamydia pneumoniae is a widely recognized cause of acute respiratory disease.⁸⁴ This organism has been identified as a significant cause of respiratory disease in recruit populations in other countries.⁸⁵ Retrospective analysis of one outbreak of pneumonia in Army recruits suggests that this organism has caused disease in this country as well. Chlamydial organisms are unique in their mechanisms of pathogenesis and could potentially cause unique problems in future trainee populations.

Streptococcal species other than group A could emerge as significant causes of respiratory disease in the future. Multiple serogroups have been incriminated as causes of disease outbreaks in other closed populations in the 1980s and 1990s.^{86–90}

SUMMARY

may derive from the belief that methods of immunoprophylaxis and chemoprophylaxis can inevitably be found to control all emerging threats. Yet it is this very belief that has perpetuated the emphasis on agent-specific strategies as the solution for disease control. These strategies have produced remarkable results, but they provide only limited solutions for what are essentially larger issues relating to modes of transmission. The need for renewing, revising, and recreating vaccines and antibiotic prophylactics will be as endless as the ability of organisms to emerge, adapt, and mutate. The continuing struggle to control agents transmitted by the airborne and direct-contact routes contrasts so clearly with the successes attained in the control of agents associated with other modes of transmission that the biomedical community should pause to consider redirecting at least some of its research efforts.

Environmental solutions that eliminate the threat of airborne and direct-contact contagion will not be easily obtained. Others who have investigated environmental factors related to the transmission of cold viruses eventually closed their laboratory with little to offer the world against its most common affliction.⁹¹ The agenda of research that was left unfinished by the Commission on Airborne Infections when Sampson Air Force Base, NY, closed in June 1956 is a reasonable starting point for resuming work that is largely unfinished.¹ As one consultant familiar with these issues stated, "The field is wide open and merely awaits the arrival of some genius."^{92(p768)}

REFERENCES

- Jordan WS. History of the Commission on Acute Respiratory Diseases, Commission on Air-Borne Infections, Commission on Meningococcal Meningitis, and Commission on Pneumonia. In: Woodward TE, ed. *The Armed Forces Epidemiological Board, The Histories of the Commissions*. Washington, DC: Office of The Surgeon General, Dept of the Army: 1994; 5–137.
- 2. The Centers for Disease Control. Measles Surveillance Report No. 11, 1977–1981. Atlanta: Public Health Service; September 1982.
- 3. Boffey PM. Anatomy of a decision: how the nation declared war on swine flu. Science. 1976;192:636-641.
- 4. Crosby AW. America's Forgotten Pandemic: the Influenza of 1918. New York: Cambridge University Press; 1989.
- 5. Dunham GC. Basic principles of military epidemiology. *Military Preventive Medicine*. 3rd ed. Harrisburg, Penn: Military Service Publishing; 1940.
- 6. Chapin CV. The Sources and Modes of Infection. New York: John Wiley & Sons; 1910.
- 7. Bayne-Jones S. *The Evolution of Preventive Medicine in the United States Army*, 1607–1939. Washington, DC: Office of the Surgeon General, Dept of the Army; 1968.
- 8. Bushnell GE. Tuberculosis. Siler JF, ed. *Communicable and Other Diseases*. Vol 9. In: *The Medical Department of the United States Army in the World War*. Washington, DC: US Government Printing Office; 1928: 171–202.
- 9. Long ER. Tuberculosis. Coates JB, Hoff EC, Hoff PM, eds. *Communicable Diseases Transmitted Chiefly through Respiratory and Alimentary Tracts.* Vol 4. In: *Preventive Medicine in World War II.* Washington, DC: Office of The Surgeon General, Dept of the Army; 1958.
- 10. Coburn AF, Young DC. *The Epidemiology of Hemolytic Streptococcus during World War II in the United States Navy*. Baltimore: Williams and Wilkins; 1949.
- 11. Rantz LA. Hemolytic streptococcal infections. Coates JB, Hoff EC, Hoff PM, eds. *Communicable Diseases Transmitted Chiefly through Respiratory and Alimentary Tracts.* Vol 4. In: *Preventive Medicine in World War II*. Washington, DC: Office of The Surgeon General, Dept of the Army; 1958.
- 12. Morrow PE. Physics of airborne particles and their deposition in the lung. Ann NY Acad Sci. 1980;353:71–80.
- 13. Knight V. Viruses as agents of airborne contagion. Ann NY Acad Sci. 1980;353:147–156.
- 14. Brodkey C, Gaydos JC. United States Army guidelines for troop living space: a historical review. *Mil Med.* 1980;145:418–421.
- 15. The Personnel of Naval Laboratory Research Unit No. 1. Air-borne infections—a review. War Med. 1943;4:1–30.
- 16. Riley EC. The role of ventilation in the spread of measles in an elementary school. Ann NY Acad Sci. 1980;353:25–34.
- 17. Gundermann KO. Spread of microorganisms by air-conditioning systems. Ann NY Acad Sci. 1980;353:209-217.
- 18. Houk VN. Spread of tuberculosis via recirculated air in a naval vessel: the Byrd study. Ann NY Acad Sci. 1980;353:10–24.
- 19. Brundage JF, Scott RM, Lednar WM, Smith DW, Miller RN. Building-associated risk of febrile acute respiratory diseases in Army trainees. *JAMA*. 1988;259:2108–2112.
- 20. Riley RL. Airborne contagion: historical background. Ann NY Acad Sci. 1980;353:3-9.
- 21. Demling RH, Maly J. The treatment of burn patients in a laminar airflow environment. *Ann NY Acad Sci.* 1980;353:294–299.

- 22. Krueger AP, Reed EJ. Biological impact of small air ions: despite a history of contention, there is evidence that small air ions can affect life processes. *Science*. 1976;193:1209–1213.
- 23. Robertson OH, Hamburger M Jr., Loosli CG, Puck TT, Lemon HM, Wise H. A study of the nature and control of air-borne infection in Army camps. *JAMA*. 1944;126:993–1000.
- 24. Commission on Acute Respiratory Disease and The Commission on Air-Borne Infections. A study of the effect of oiled floors and bedding on the incidence of respiratory disease in new recruits. *Am J Hyg.* 1946;43:120–144.
- 25. Puck TT, Robertson OH, Wise H, Loosli CG. The oil treatment of bedclothes for the control of dust-borne infection, I: principles underlying the development and use of a satisfactory oil-in-water emulsion. *Am J Hyg.* 1946;43:91–104.
- 26. Loosli CG, Wise H, Lemon HM, Puck TT. The oil treatment of bedclothes for the control of dust-borne infection, II: the use of triton oil emulsion (T-13) as a routine laundry procedure *Am J Hyg.* 1946;43:120–144.
- 27. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE). Ventilation for acceptable indoor air quality. ASHRAE, Atlanta, Ga, 1989.
- 28. Commission on Acute Respiratory Disease and The Commission on Air-Borne Infections. The effect of doublebunking in barracks on the incidence of respiratory disease. *Am J Hyg.* 1946;43:65–81.
- 29. Centers for Disease Control and Prevention. National surveillance for infectious diseases, 1995. *MMWR*. 1995;44:737–739.
- 30. Dudding BA, Top FH Jr, Winter PE, Buescher EL, Lamson TH, Leibovitz A. Acute respiratory disease in military trainees: the adenovirus surveillance program, 1966–1971. *Am J Epidemiol*. 1973;97:187–198.
- 31. Dingle JH, Langmuir AD. Epidemiology of acute, respiratory disease in military recruits. *Am Rev Respir Dis.* 1968;97(suppl):1–65.
- 32. Brundage JF, Gunzenhauser JD, Longfield JN, et al. Epidemiology and control of acute respiratory diseases with emphasis on group A beta-hemolytic streptococcus: A decade of U.S. Army experience. *Pediatrics*. 1996;97:964–970.
- 33. Centers for Disease Control. Acute rheumatic fever among Army trainees—Fort Leonard Wood, Missouri, 1987-1988. *MMWR*. 1988;37:519–522.
- 34. Gunzenhauser JD, Longfield JN, Brundage JF, Kaplan EL, Miller RN, Brandt CA. Epidemic streptococcal disease among Army trainees, July 1989 through June 1991. *J Infect Dis.* 1995;172:124–131.
- 35. Armed Forces Epidemiological Board. Recommendations of the ad hoc committee on prophylaxis of streptococcal infections of the commission on streptococcal disease. Washington, DC: Armed Forces Epidemiological Board, 1959.
- 36. Centers for Disease Control. Acute rheumatic fever at a Navy training center—San Diego, California. *MMWR*. 1988;37:101–104.
- 37. Meiklejohn G, Zajac RA, Evans ME. Influenza at Lowry Air Force Base in Denver, 1982-1986. J Infect Dis. 1987;156:649–651.
- 38. Meiklejohn G. Viral respiratory disease at Lowry Air Force Base in Denver, 1952–1982. J Infect Dis. 1983;148:775–784.
- 39. Cliff AD, Haggett P, Ord JK. Spatial Aspects of Influenza Epidemics. London: Pion Ltd; 1986.
- 40. Fine PE. Herd immunity: history, theory, practice. *Epidemiol Rev.* 1993;15:265–302.
- 41. Goldfield M, Bartley JD, Pizzuti W, Black HC, Altman R, Halperin WE. Influenza in New Jersey in 1976: isolations of influenza A/New Jersey/76 virus at Fort Dix. *J Infect Dis.* 1977;136(suppl):S347–S355.

- 42. Gaydos JC, Hodder RA, Top FH Jr, et al. Swine influenza A at Fort Dix, New Jersey (January-February 1976), I: case finding and clinical study of cases. *J Infect Dis.* 1977;136(suppl):S356–S362.
- 43. Gaydos JC, Hodder RA, Top FH Jr, et al. Swine influenza A at Fort Dix, New Jersey (January-February 1976), II: transmission and morbidity in units with cases. *J Infect Dis.* 1977;136(suppl):S363–S368.
- 44. Hodder RA, Gaydos JC, Allen RG, Top FH Jr, Nowosiwsky T, Russell PK. Swine influenza A at Fort Dix, New Jersey (January-February 1976), III: extent of spread and duration of the outbreak. J Infect Dis. 1977;136(suppl):S369–S375.
- 45. Top FH Jr, Russell PK. Swine influenza A at Fort Dix, New Jersey (January-February 1976), IV: summary and speculation. J Infect Dis. 1977;136(suppl):S376–S380.
- 46. Neustadt RE, Fineberg H. *The Swine Flu Affair: Decision Making in a Slippery Case*. Washington, DC: US Dept of Health, Education, and Welfare; 1978.
- 47. Roscelli JD, Bass JW, Pang L. Guillain-Barré syndrome and influenza vaccination in the US Army, 1980-1988. *Am J Epidemiol*. 1991;133:952–955.
- 48. Safranek TJ, Lawrence DN, Kurland LT, et al. Reassessment of the association between Guillain-Barré syndrome and receipt of swine influenza vaccine in 1976–1977: results of a two-state study. *Am J Epidemiol*. 1991;133:940–951.
- 49. Schoenbaum SC, McNeil BJ, Kavet J. The swine-influenza decision. N Engl J Med. 1976;295:759–765.
- 50. Hoke C, Division of Communicable Diseases and Immunology, Walter Reed Army Institute of Research. Personal Communication, 1995.
- 51. Buescher EL. Respiratory disease and the adenoviruses. Med Clin North Am. 1967;51:769–779.
- 52. Commission on Acute Respiratory Diseases. Acute respiratory disease among new recruits. *Am J Public Health Nation's Health*. 1946;36:439–450.
- 53. Bloom HH, Forsyth BR, Johnson KM, et al. Patterns of adenovirus infections in Marine Corps personnel. *Am J Hyg.* 1964;80:328–342.
- 54. Top FH Jr, Grossman RA, Bartelloni PJ, et al. Immunization with live types 7 and 4 adenovirus vaccines, I: safety, infectivity, antigenicity, and potency of adenovirus type 7 vaccine in humans. *J Infect Dis.* 1971;124:148–154.
- 55. Dudding BA, Top FH Jr. Scott RM, Russell PK, Buescher EL. An analysis of hospitalizations for acute respiratory disease in recruits immunized with adenovirus type 4 and type 7 vaccines. *Am J Epidemiol*. 1972;95:140–147.
- 56. Collis PB, Dudding BA, Winter PE, Russell PK, Buescher EL. Adenovirus vaccines in military recruit populations: a cost-benefit analysis. *J Infect Dis.* 1973;128:745–752.
- 57. Brundage JF, Zollinger WD. The epidemiology of meningococcal disease in the US Army. In: Vedros NA, ed. *Evolution of Meningococcal Disease*. Vol 1. Boca Raton, Fla: CRC Press; 1987: 5–25.
- 58. Moore PS, Harrison LH, Telzak EE, Ajello GW, Broome CV. Group A meningococcal carriage in travelers returning from Saudi Arabia. *JAMA*. 1988;260:2686–2689.
- 59. Gordon JE. General consideration of modes of transmission. In: Coates JB, Hoff EC, Hoff PM, eds. *Communicable Diseases Transmitted Chiefly through Respiratory and Alimentary Tracts.* Vol 4. In: *Preventive Medicine in World War II.* Washington, DC: Office of The Surgeon General, Dept of the Army; 1958: 3–52.
- 60. Phair JJ. Meningococcal meningitis. In: Coates JB, Hoff EC, Hoff PM, eds. *Communicable Diseases Transmitted Chiefly through Respiratory and Alimentary Tracts*. Vol 4. In: *Preventive Medicine in World War II*. Washington, DC: Office of The Surgeon General, Dept of the Army; 1958: 191–209.

- 61. Benenson AS, ed. *Control of Communicable Diseases in Man*. Washington, DC: Dept of the Army; 1995: 280–284. Army Field Manual 8–33.
- 62. Cvjetanovic B. Strategy for control. In: Vedros NA, ed. *Evolution of Meningococcal Disease*. Vol 1. Boca Raton, Fla: CRC Press; 1987: 135–143.
- 63. Veasy LG, Wiedmeier SE, Orsmond GS, et al. Resurgence of acute rheumatic fever in the intermountain area of the United States. *N Engl J Med.* 1987;316:421–427.
- 64. The Working Group on Severe Streptococcal Infections. Defining the group A streptococcal toxic shock syndrome: rationale and consensus definition. *JAMA*. 1993;269:390–391.
- 65. Centers for Disease Control. Group A beta-hemolytic streptococcal pharyngitis among U.S. Air Force trainees—Texas, 1988–89. *MMWR*. 1990;39:11–13.
- 66. Denny FW Jr, Houser HB. Commission on Streptococcal and Staphylococcal Diseases. In: Woodward TE, ed. *The Armed Forces Epidemiological Board: The Histories of the Commissions.* Washington, DC: Office of The Surgeon General, Borden Institute; 1994.
- 67. Davis J, Schmidt WC. Benzathine penicillin G: on effectiveness in the prevention of streptococcal infections in a heavily exposed population. *N Engl J Med.* 1957;256:339–342.
- 68. Morris AJ, Rammelkamp CH. Benzathine penicillin G in the prevention of streptococcic infections. *JAMA*. 1957;165:664–667.
- 69. Frank PF. Streptococcal prophylaxis in Navy recruits with oral and benzathine penicillin. *US Armed Forces Med J.* 1958;9:543–560.
- 70. Schreier AJ, Hockett VE, Seal JR. Mass prophylaxis of epidemic streptococcal infections with benzathine penicillin G, I: experience at a naval training center during the winter of 1955–56. *N Engl J Med.* 1958;258:1231–1238.
- 71. McFarland, Colvin VG, Seal JR. Mass prophylaxis of epidemic streptococcal infections with benzathine penicillin G, I: experience at a naval training center during the winter of 1956–57. *N Engl J Med.* 1958;258:1277–1284.
- 72. Frank PF, Stollerman GH, Miller LF. Protection of a military population from rheumatic fever: routine administration of benzathine penicillin G to healthy individuals. *JAMA*. 1965;193(10):119–127.
- 73. Gray GC, Escamilla J, Hyams KC, Struewing JP, Kaplan EL, Tupponce AK. Hyperendemic *Streptococcus pyogenes* infection despite prophylaxis with penicillin G benzathine. *N Engl J Med.* 1991;325:92–97.
- 74. Gunzenhauser JD, Brundage JF, McNeil JG, Miller RN. Broad and persistent effects of benzathine penicillin G in the prevention of febrile, acute respiratory disease. *J Infect Dis.* 1992;166:365–373.
- 75. Dale JB. Vaccine for strep A closer to reality. US Med. 1990;August:47-48.
- 76. Fischetti VA, Hodges WM, Hruby DE. Protection against streptococcal pharyngeal colonization with a vaccinia: M protein recombinant. *Science*. 1989;244:1487–1490.
- 77. Hodges RG, MacLeod CM. Epidemic pneumococcal pneumonia, I: description of the epidemic. *Am J Hyg.* 1946;44:183–192.
- 78. Shapiro ED, Berg AT, Austrian R, et al. The protective efficacy of polyvalent pneumococcal polysaccharide vaccine. *N Engl J Med.* 1991;325:1453–1460.
- 79. Breiman RF, Butler JC, Tenover FC, Elliott JA, Facklam RR. Emergence of drug-resistant pneumococcal infections in the United States. *JAMA*. 1994;271:1831–1835.
- 80. Hoge CW, Reichler MR, Dominguez EA, et al. An epidemic of pneumococcal disease in an overcrowded, inadequately ventilated jail. *N Engl J Med*. 1994;331:643–648.

- 81. Eskola J, Takala AK, Kela E, Pekkanen E, Kalliokoski R, Leinonen M. Epidemiology of invasive pneumococcal infections in children in Finland. *JAMA*. 1992;268:3323–3327.
- 82. Cherian T, Steinhoff MC, Harrison LH, Rohn D, McDougal LK, Dick J. A cluster of invasive pneumococcal disease in young children in child care. *JAMA*. 1994;271:695–697.
- 83. Centers for Disease Control. Outbreak of invasive pneumococcal disease in a jail—Texas, 1989. MMWR. 1989;38:733-734.
- 84. Grayston JT, Wang SP, Kuo CC, Campbell LA. Current knowledge on *Chlamydia pneumoniae*, strain TWAR, and important cause of pneumonia and other acute respiratory diseases. *Eur J Clin Microbiol Infect Dis*. 1989;8:191–202.
- 85. Kleemola M, Saikku P, Visakorpi R, Wang SP, Grayston JT. Epidemics of pneumonia caused by TWAR, a new *Chlamydia* organism, in military trainees in Finland. *J Infect Dis.* 1988;157:230–236.
- 86. Turner JC, Hayden GF, Kiselica D, Lohr J, Fishburne CF, Murren D. Association of group C beta-hemolytic streptococci with endemic pharyngitis among college students. *JAMA*. 1990;264:2644–2647.
- 87. Benjamin JT, Perriello VA Jr. Pharyngitis due to group C hemolytic streptococci in children. J Pediatr. 1976;89:254–256.
- 88. McCue JD. Group G streptococcal pharyngitis: analysis of an outbreak at a college. JAMA. 1982;248:1333–1336.
- 89. Meier FA, Centor RM, Graham L Jr, Dalton HP. Clinical and microbiological evidence for endemic pharyngitis among adults due to group C streptococci. *Arch Intern Med.* 1990;150:825–829.
- 90. Hill HR, Caldwell GG, Wilson E, Hager D, Zimmerman RA. Epidemic of pharyngitis due to streptococci of Lancefield group G. *Lancet*. 1969;2:371–374.
- 91. Andrewes CH. Adventures among viruses, III: the puzzle of the common cold. Rev Infect Dis. 1989;11:1022–1028.
- 92. Andrewes CH. The common cold: prospects for its control. Med Clin North Am. 1967;51:765-768.