Military Service and Lung Disease

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Objective: Lung diseases associated with military service are often a reflection of the conditions seen in the local civilian population, and with a few notable exceptions, are often related to unique environmental and occupational exposures. **Methods:** This article reviews important pulmonary diseases that have been associated with military service in the past 100 years in a question-and-answer format. **Results:** Traditionally, bacterial and viral pneumonias were the most common sources of military morbidity and mortality. With improved preventive medicine and antimicrobial therapy, other diseases related to battlefield injuries or inhalational exposures have assumed greater importance. **Conclusions:** The etiology of military morbidity and mortality has evolved over the past century. Many of the discoveries related to vaccine efficacy, trauma resuscitation, interstitial lung disease, and even carcinomas have a strong military association.

With the introduction of preventive medicine techniques, vaccines, and antibiotics, morbidity and mortality related to pulmonary diseases have dramatically decreased. During World War I, 20% to 40% of US military personnel were devastated by lethal lobar pneumonia caused by the influenza A pandemic. The advent of penicillin in 1928 and influenza vaccine in the 1950s dramatically reduced mortality and morbidity associated with respiratory illnesses.[1,2] Regardless, lung diseases related to military medicine have remained an important topic, with new concerns of chemical warfare, and battlefield exposure to high levels of urban pollution and inhaled particulate matter. This article offers a historical overview of lung diseases associated with US military service from the twentieth century to our most current conflicts in Southwest Asia (SWA).

DID MORE TROOPS DIE IN WORLD WAR I BECAUSE OF INFLUENZA, PNEUMONIA, OR CHEMICAL GASES?

Infectious diseases such as influenza and pneumonia were the predominant cause of death during World War I and took more lives among military personnel than did enemy artillery.[1] The trenches in Europe fostered the spread of the influenza virus and the disease in turn weakened military efforts by depleting resources and rendering troops ineffective. Between 1917 and 1919, 50% of deaths in the US Army were secondary to influenza and pneumonia, whereas 43% of deaths were battle-related. In 1918, at the peak of American involvement in the war, 20% to 40% of US Army and Navy personnel were afflicted with pneumonia and influenza.[1,2] Concurrent with the spread of influenza was the advent of chemical warfare. The release of chlorine gas by German forces over the Western front marked the beginning of chemical warfare. By the end of the war in 1918, 1.3 million chemical weapon casualties resulted in approximately 100,000 deaths.[3,4]

WHAT CHEMICAL AGENT CAUSES THE MOST PULMONARY DAMAGE—MUSTARD, CHLORINE, OR PHOSGENE?

The release of 160 tons of chlorine gas by German forces at the Battle of Ypres in World War I marked the first large-scale use of a chemical weapon in modern warfare. Known as the chemist's war, this gave way to the development of other agents such as phosgene and mustard gas. Phosgene was the most widely used agent and was responsible for a large majority of chemical weapon-related deaths.[5] Phosgene, a choking agent, directly damages lung tissue in a dose-dependent manner and because of its lower solubility also affects the lower airways. Low to moderate concentrations produced cough, dyspnea, and bronchospasm. Higher concentrations were devastating, causing extensive tissue necrosis and alveolar capillary damage leading to noncardiogenic pulmonary edema or over the next 1 to 3 days, progression to acute respiratory distress syndrome (ARDS).[5] These effects were more potent than other choking agents such as chlorine gas. Unlike chlorine gas, the toxic threshold remains below the olfactory threshold and at very high doses (>200 ppm), death from acute cor pulmonale occurred within minutes.[6,7] Mustard gas also produced respiratory symptoms in 70% of casualties. Its effects range from chemical tracheobronchitis and stenosis to pulmonary edema and hemorrhage, with resultant respiratory failure. Long-term sequelae in symptomatic patients exposed to mustard gas during the Iran–Iraq War included constrictive bronchiolitis (CB), an inflammatory and fibrotic narrowing of the airways diagnosed by surgical lung biopsies.[8] Although chemical weapons led to proportionately fewer deaths in World War I, the medical, psychological, and ethical consequences of their use remain relevant to this day.

WHAT ARE SOME RECENT EXAMPLES OF CHEMICAL WARFARE AGENTS THAT CAUSE PULMONARY INJURY TO INCLUDE ARDS?

Multiple attacks using chlorine tanker trucks were executed by Iraqi insurgents during Operation Iraqi Freedom.[9] Chlorine gas exerts its toxic effects when it reacts with water to produce hypochlorous and hydrochloric acid. The acid causes irritation of the airways, leading to cough, dyspnea, and bronchospasm. The subsequent disruption of alveolar and endothelial cell membranes causes pulmonary edema and sets the stage for ARDS.[2,6,7] In mild to moderate exposures, symptoms resolve within 3 to 5 days and patients have normal pulmonary function tests (PFTs). Patients are however at risk for long-term complications, such as reactive airway dysfunction syndrome. This is in contrast to the nerve agent sarin that causes pulmonary injury in a shorter period.[10] Sarin gas, recently used in the Syrian crisis, is a highly volatile agent with a 5-hour half-life that inhibits acetylcholinesterase and produces symptoms of an acute cholinergic crisis. When inhaled, symptoms occur within seconds and can progress to death in minutes; thus, a patient with sarin toxicity is unlikely to present with ARDS 24 hours postexposure. Similarly, blistering agents such as mustard gas are less likely to be the cause of ARDS. Mustard gas was last used in the Iran–Iraq conflict in the 1980s. Respiratory symptoms, while less common than eye and skin effects, can occur but are more commonly limited to the upper airways.[8,11]

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The opinions in this article do not constitute endorsement by the Orlando VA Medical Center, Department of Veterans Administration, or the US Government of the information contained therein.

The authors declare no conflicts of interest.

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DOI: 10.1097/JOM.0000000000000255

WHAT WAS THE IMPACT OF ENHANCED TRIAGE AND AGGRESSIVE FIELD RESUSCITATION ON MORBIDITY AND MORTALITY DURING THE VIETNAM WAR?

World War II saw the first use of antibiotics to treat infections and the establishment of an Army Medical Corps trauma system.[12] The Korean War expanded on this system to create Mobile Army Surgical Hospitals, and helicopters were used for the first time to evacuate injured soldiers.[13] Although these advances contributed to a decrease in combat-related mortality (45% to 66% in World War I, 9% in the Vietnam War), a new phenomenon was developing; soldiers were successfully resuscitated from hemorrhagic and traumatic shock incurred in battle only to have their recovery later interrupted by life-threatening respiratory distress.[12–14] These patients developed a complex of respiratory symptoms including rapid shallow breathing, productive cough, refractory cyanosis, diffuse interstitial infiltrates on radiograph, and pulmonary edema. In severe cases, death occurred from respiratory failure. Injury was initiated by pulmonary hypoperfusion during shock and exacerbated by overaggressive fluid resuscitation and mechanical ventilation. Initially identified as "wet lung" in World War II, these cases became so prevalent during the Vietnam War that the term "DaNang lung" was coined to describe the syndrome.[13] We now recognized this pulmonary disorder as ARDS.

WHICH VIRUS—INFLUENZA, ADENOVIRUS, OR RESPIRATORY SYNCYTIAL VIRUS IS THE MOST COMMON ETIOLOGY OF ACUTE RESPIRATORY ILLNESS IN TROOPS?

Before the identification of its etiology in 1953, the acute respiratory illness caused by adenovirus was referred to as the "acute respiratory disease of recruits" due its predilection for young, healthy military recruits.[15] In the 1950s, adenovirus was consistently isolated in 30% to 70% of trainees with respiratory disease and was associated with 90% of pneumonia cases.[15,16] In 1971, the development and routine use of a vaccine against the most common serotypes of adenovirus, 4 and 7, led to a dramatic shift in its epidemiology, resulting in a reduction in infection rates by 95% to 99%. In 1994, the vaccine's sole manufacturer ceased production citing economic reasons, and since its discontinuation in 1998, the military has experienced a resurgence of adenovirus-related infections.[15,16] A 5-year surveillance study between 1999 and 2004 of eight Army, Navy and Air Force training bases identified adenovirus as the causative agent in 66.8% of cases of febrile respiratory illness in recruits.[16] Furthermore, since 2005, there has been an emergence of infection related to an additional serotype, Ad14.[17] These outbreaks contributed to the reinstitution of the adenovirus vaccine in 2011; subsequent cohort studies from 1999 to 2012 have shown decreased incidence of pneumonia and bronchitis since vaccine reintroduction.[18]

WHAT OCCUPATIONAL LUNG DISEASE AND MALIGNANCY HAS BEEN ASSOCIATED WITH NAVY VETERANS AND SHIPBUILDING?

Asbestos was widely used by the Navy in shipbuilding during World War II because of its incombustibility and low thermal conductivity. During the 1930s, studies demonstrated an association between occupational exposure to asbestos fibers and a fibrosing lung disease known as asbestosis. Aware of the occupational hazard, the Navy established guidelines for the use of asbestos; however, they held the belief that these risks could be adequately controlled with proper training, so use remained high through World War II. In the 1950s, Richard Doll first reported a link between asbestos exposure and lung cancer.[19] With mounting evidence of the risks of lung disease associated with asbestos, the Navy adopted stricter measures such as respirators to counter these risks. Unfortunately, asbestos use and exposure remained prominent until the mid-1980s. The association between asbestos exposure and mesothelioma is now clearly defined, and retrospective case studies continue to provide evidence of increased lung cancer in veterans who worked in these shipyards.[20–22]

HAS EXPOSURE TO AGENT ORANGE BEEN PROVEN TO CAUSE LUNG DISEASE?

No. The US military sprayed millions of gallons of Agent Orange and other herbicides on trees and vegetation during the Vietnam War. Health concerns because of exposures continue. The Veterans Administration (VA) assumes that "presumptive diseases" may be related to a veteran's Agent Orange exposure during both the latter part of the Korean conflict and the Vietnam War. Although the VA has recognized respiratory cancers of the lung, larynx, trachea, and bronchus as eligible for disability compensation or survivors' benefits, no clear link has ever been established.[23] None of the interstitial lung diseases have been associated with Agent Orange exposure. The VA is looking at chronic obstructive pulmonary disease and has a study underway of 4000 American Chemical Corps Veterans who served between 1964 and 1975 to see if they had a significantly higher incidence of chronic obstructive pulmonary disease than their nonexposed counterparts.[24]

A MILITARY TRAINEE STATIONED IN EL PASO, TEXAS, PRESENTS AFTER A 5-WEEK TRAINING EXERCISE WITH SYMPTOMS OF COUGH, DYSPNEA, AND FLU-LIKE SYMPTOMS. THE WORK-UP OF THIS PATIENT WARRANTS TESTING FOR WHAT RESPIRATORY DISEASE?

Military members stationed in the Southwest United States are at risk for coccidioidomycosis infection, secondary to inhalation of spores of *Coccidioides immitis* that is endemic to these regions.[25–28] Symptoms are present in 40% to 60% of cases with the most common involving the respiratory tract and ranging from mild, self-limited respiratory infections to life-threatening pneumonia. Military interest in *Coccidioides* stems back to 1940 to 1941 when US Army Air Forces established training bases in the San Joaquin Valley.[25,27] During World War II, an annual incidence of 8% to 25% was reported among military personnel training in the southwestern United States. A second outbreak occurred in 1977 and affected 18 individuals at the Naval Air Station in Lemoore, California, after a dust storm.[27] The highest attack rate thus far occurred in 2001; 10 of 22 men (45%) in a Navy SEAL unit were diagnosed with acute *Coccidioides* infection during a 6-week training exercise in Coalinga, California.[28]

WHAT INTERSTITIAL LUNG DISEASE RESULTS IN RAPIDLY PROGRESSIVE INFILTRATES, HYPOXIA, AND HIGH EOSINOPHIL LEVELS IN THE BRONCHOALVEOLAR LAVAGE FLUID? WHAT IS THE TREATMENT? WHAT IS A COMMON HISTORICAL FEATURE?

Acute eosinophilic pneumonia (AEP) is a syndrome of unknown etiology characterized by febrile illness, respiratory symptoms, pulmonary eosinophilia, and diffuse infiltrates on chest X-ray.[29] Acute eosinophilic pneumonia can rapidly progresses to acute respiratory distress requiring mechanical ventilation. A study of severe pneumonia among US military personnel serving in Operation Iraqi Freedom identified 18 members with AEP between March 2003 and August 2004 (incidence rate of 9.1 per 100,000 person-years), with two reported deaths. The cause of AEP remains unknown. Nevertheless, in this series all were smokers and 78% had a recent onset of smoking. Military physicians in SWA maintained a high clinical suspicion for AEP, which responds to high-dose steroids, and

despite additional cases, there have been no further deaths attributed to AEP.[30]

WHAT IS THE MOST COMMON PULMONARY DISEASE IN ACTIVE DUTY SERVICE MEMBERS PRESENTING WITH DYSPNEA ON EXERTION?

A prospective comprehensive evaluation of 105 active duty personnel, with dyspnea on exertion, found that obstructive lung disease accounted for 52% of the diagnoses followed by vocal cord dysfunction. Of note, no specific diagnosis was identified in 24% of individuals despite full PFTs, methacholine challenge testing, chest roentgenogram, cardiopulmonary exercise test, arterial blood gas, echocardiogram, laboratory tests, electrocardiogram, and laryngoscopy.[31] Obstructive lung disease, especially asthma, remains the leading cause of dyspnea on exertion in a recent prospective trial of service members returning from deployment to SWA.[32] A diagnosis of asthma does not automatically exclude an individual from military service, and once in the service, medical discharge is not required if the asthma is well controlled with or without medications.[33] A survey of soldiers returning from deployment to SWA revealed that 5% had a preexisting diagnosis of asthma. Respiratory symptoms were common in both asthmatic and non-asthmatic individuals during deployment, especially in asthma patients with poor baseline control.[34] The majority of SWA postdeployment studies have shown an increase in symptoms, but not actual disease.[35,36] Concerning is a case series in which 38 soldiers, many with normal PFTs and high-resolution CT scans, were diagnosed with CB by surgical lung biopsy.[37] In the initial data analysis, a majority of these soldiers had exposure to a sulfur mine fire in Iraq in 2003, but in the final analysis greater than 50% of soldiers biopsied did not have this specific toxic exposure. It remains controversial whether service members without demonstrable significant physiological or radiographic abnormalities should undergo a surgical lung biopsy in the absence of effective treatment or disease progression classically associated with CB.[38] An EPICON study that looked at soldiers exposed to the 2003 Iraqi sulfur fire (time-based and location-based in an unexposed population vs exposed population) showed increased symptoms in the most exposed groups, but not documented respiratory disease.[39] Prospective, randomized trials and longitudinal follow-up of service members with inhalational exposures in SWA are warranted. A diagnostic algorithm or research trial that involves lung biopsy (especially in unexposed or asymptomatic individuals to serve as controls) would be hard to justify on the basis of potential morbidity and mortality associated with video-assisted thoracoscopic surgery lung biopsy to include chronic postthoracotomy pain syndrome, which occurs in 5% to 33% of patients with video-assisted thoracoscopic surgery .[40] Pulmonologists, occupational and preventive medicine specialists, industrial hygienists, and exposure scientists from federal and academic medical centers convened a 2010 Working Group and recommended a comprehensive pulmonary evaluation to ensure that common causes of dyspnea on exertion in young adults were considered, and a case-by-case referral for video-assisted thoracoscopic surgery. The proposed case definition of postdeployment CB was persistent respiratory symptoms and at least two additional findings (unexplained abnormal PFTs, exercise tolerance test, high-resolution CT, or surgical lung biopsy).[41]

WHAT IS THE ASSOCIATION BETWEEN BURN PITS AND PULMONARY DISEASE?

The Department of Defense estimates that in a typical military operation, each US soldier generates 9 to 12 lb of waste a day. Open air burn pits were used extensively throughout SWA, especially in the early years of the conflict to eliminate this waste. These burn pits were gradually phased out and replaced with incinerators.[42] Environmental sampling by the United States Army Public Health Command has revealed that deployed military personnel have been exposed to increased levels of airborne particulate matter that exceed occupational, environmental, and military exposure guidelines. The majority of the particulate matter was from suspended geological dust.[43] The 2011 Institute of Medicine report with a focus on burn pit exposure concluded that the broader exposure to high levels of particulate matter, rather than just burn pits, might be associated with long-term health effects, particularly with high exposure or in susceptible persons.[42] This conclusion was recently substantiated by a retrospective cohort study of military personnel who were deployed to either areas with or without burn pits. Although deployment to Iraq was associated with an increase in respiratory symptoms and asthma compared with US stationed personnel, there was no increase in medical encounters uniquely associated with burn pits.[44] In addition, preexisting abnormalities and exposures may account for many of the abnormalities seen in service members returning from deployment to SWA. Although the military is in general more physically fit than their civilian counterparts, a recent predeployment evaluation of 775 soldiers revealed that predeployment respiratory symptoms and suboptimal exercise endurance along with spirometric abnormalities are common in these soldiers.[45] Fifty-one (6.8%) reported symptoms of dyspnea in the previous 4 weeks, and 27 (3.5%) reported persistent dyspnea for greater than 4 weeks. A total of 182 (23.4%) failed their most recent physical fitness test; 308 (39.9%) had a body mass index of more than 25 kg/m^2 and 15 (1.9%) had a body mass index of more than 30 kg/m^2. Baseline spirometry showed an obstructive ventilatory defect in 67 (8.7%) participants before deployment. In addition, 42% were current or former smokers. Irrespective of these data, the military will need to be more cognizant of environmental exposures to include urban pollution, and focus on preventive pulmonary measures to limit inhalational insults and their potential short- and long-term health consequences. Obviously, smoking cessation must be a part of any respiratory protection plan.

BECAUSE OF CONCERNS ABOUT LUNG DISEASES ASSOCIATED WITH EXPOSURE THAT OCCUR AT THE BATTLE FRONT, SHOULD BASELINE OR SCREENING SPIROMETRY BE CONSIDERED FOR MILITARY PERSONNEL?

A high-quality initial lung function test at the time of military induction might be useful, especially if subsequent studies are significantly reduced but still in a "normal" range; however, implementation would be difficult, costly, and of unknown overall benefit. The routine use of spirometry has many limitations to include overdiagnosis, misinterpretation, technically inadequate studies, and in the case of military personnel–adverse career implications.[46,47] Furthermore, screening spirometry is not advocated for any asymptomatic population except on a longitudinal basis in certain occupations with direct exposure, which are potentially high risk for lung disease. Because all active service members can expect to deploy, it would be difficult to identify and target only the high-risk occupations or exposures. Millions of service members would require baseline and then longitudinal screening. Even with the use of high-tech equipment, well-trained technicians, and coachable subjects, there will be false-positive and false-negative results. Initial evaluations would be compared with reference populations, that if used by themselves and with 80% predicted and fixed cut points would lead to substantial misdiagnosis of disease affecting greater than 20% of subjects. This misclassification may be even higher in a generally asymptomatic population.[48] The perception that military members, because of their cardiovascular fitness, have supranormal PFTs was recently refuted by a study that showed no difference in the prevalence of supranormal PFTs in an active duty cohort as compared with their nonactive duty counterparts.[49] It is also unclear whether spirometry, even in the best circumstances, is the best screening or surveillance tool for

detection of early or preclinical lung disease in patients, to include those who smoke and are at higher risk for development of lung disease.[50] Impulse oscillometry is widely used in the evaluation of airway diseases in children, and provides a rapid, noninvasive, effort-independent, and validated measure of airway impedance that is used as an indicator of lung function.[51] Two feasibility studies, in a new recruit and in a predeployment population, that utilize both spirometry and impulse oscillometry are ongoing and might provide a better understanding of the utility and cost–benefit of a comprehensive spirometry surveillance program.[52]

CONCLUSIONS

Lung disease associated with military service has decreased in overall contributions to mortality because of advances in the prevention and treatment of infectious diseases, and decreased use of chemical weapons. The recognition of ARDS after survival of initial combat injuries led to important advances in our knowledge of trauma resuscitation and mechanical ventilation strategies. Modern warfare with blurred battlefield lines and deployments to places with high levels of particulate matter to include urban pollution make us cognizant of new health threats, diseases, and preventative strategies. Given that the above scenarios show a clear association with tobacco use, an additional military strategy needs to be smoking cessation programs and policies that deter or ban the use of tobacco products.

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