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PULMONOLOGY IN COMBAT MEDICINE (G EAPEN, SECTION EDITOR)

Non-traumatic Pulmonary Emergencies in the Deployed Setting

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Abstract
Purpose of Review Pulmonary disorders accounted for up to 8% of the over 70,000 medical evacuations conducted from Iraq and Afghanistan in the past 15 years. This review of non-traumatic pulmonary emergencies provides an overview of deployed military medical treatment capabilities and highlights pulmonary emergencies requiring aeromedical evacuation from theater.
Recent Findings Recent studies have improved the epidemiologic evaluation of non-traumatic pulmonary disease, highlighted specific parenchymal diseases, and revealed infection pathologies unique to the deployed setting. Literature regarding possible chemical exposures in the current deployed environment remains limited.
Summary Respiratory disorders requiring medical evacuation represent a wide variety of diseases. Complications such as pulmonary emboli, infectious pathogens, and hazardous chemical exposures threaten the deployed warfighter. Adequate medical care requires an understanding of these potential environmental exposures. This review serves as a general overview of this topic; however, more research regarding epidemiologic and environmental exposures is required.

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Introduction

Since the initiation of combat operations in 2001, more than two million military personnel have been deployed to Southwest Asia. From 2003 to 2011, over 70,000 service members were evacuated from combat zones in Southwest Asia for non-combat-related illnesses and injuries. Pulmonary conditions were responsible for 3–8% of these evacuations. The management of pulmonary diseases in the deployed setting is complicated due to numerous environmental hazards, toxic exposures, and limited resources. This review will summarize the respiratory conditions that require aeromedical evacuation (AE). First, in-theater medical capabilities and then the treatment and diagnosis of pulmonary diseases in the deployed setting will be reviewed. Finally, unique infections and inhalational threats to service members in Southwest Asia will be discussed.

Deployed Medical Capabilities

An appreciation of combat medical care is necessary to understand the diagnosis, treatment, and disposition of deployment-related respiratory emergencies. Deployed medical resources are asymmetrically distributed, as the goal of combat medicine is to perform immediate lifesaving interventions and stabilize casualties for transport to higher levels of care. Military doctrine describes a five-tiered disbursement of medical resources through increasing echelons of care [1•]. Echelon I facilities are forward aid medical stations without

surgical capabilities. Echelon II comprises medical facilities that perform lifesaving "damage control" surgery. These facilities have basic laboratory and radiologic capabilities but have minimal holding capacity and lack the ability to perform bronchoscopies or other advanced medical interventions [2]. Echelon III provides surgical subspecialties, robust intensive care capabilities, and more extensive holding capacity. These facilities provide the highest level of in-theater medical care but are often limited by manpower and bed availability. Echelon IV facilities are military hospitals located outside of combat zones, but not within the U.S.A. Echelon V facilities are military hospitals located within the U.S.A. More exhaustive descriptions of AE and military medical doctrine are described elsewhere [1•, 2, 3]. Specialized teams exist to transport patients between higher echelons of care. These include critical care air transport teams (CCATT), a burn flight team, and an acute lung rescue team (ALRT) [1•, 4, 5•].

Epidemiology

Over the past 15 years, more than two million service members have been deployed to Southwest Asia [6]. During a 12-month combat deployment, approximately 4% of army personnel, 2% of marine corps, and 1% of navy and air force members are medically evacuated [7]. From 2003 to 2011, 50,634 service members were evacuated from Iraq. Of those evacuations, 41,690 (82.3%) were for disease and non-battle injuries (DNBI). Pulmonary symptoms and diagnoses accounted for 1167 (2.6%) of evacuations [6]. An echelon II facility in Iraq reported that of 4831 encounters, 4663 (93%) were for DNBI. Of those outpatient encounters, 191 (4%) were for upper respiratory complaints and 56 (1.2%) for asthma or other respiratory issues. During the survey, one soldier was evacuated for asthma [8]. Another analysis of 343 patients evacuated for DNBI showed 3% had underlying pulmonary conditions [9]. Of 875 intensive care unit admissions to a combat support hospital, 125 were admitted for DNBI and 8% were admitted for pulmonary etiologies [10]. Over 11 years, 23,719 personnel were evacuated from Afghanistan. DNBI accounted for 18,144 (76.2%) of these evacuations and 442 (1.9%) were for pulmonary complications [11]. A review of 10 years of CCATT data reveals that of 673 DNBI CCATT missions, 107 (15.9%) were related to pulmonary conditions [12].

Due to the large spectrum of diseases, diverse exposures, variable environments, and sporadic reporting, the precise characterization of non-traumatic pulmonary emergencies in deployed personnel is difficult. However, with nearly 80000 soldier days lost to DNBI over a 2 year period [13] and with pulmonary disease representing 3-8% of DNBI evacuations [6, 7, 11], the impact of non-traumatic downrange pulmonary emergencies is a relevant topic for bother service members and medical providers. For the purposes of this article, pulmonary emergencies will be defined as any pulmonary condition associated with respiratory failure, death or requiring medical evacuation from the theater of operations. Other pulmonary conditions such as altitude illness and thromboembolic disease are addressed elsewhere.

Pulmonary Disorders

Acute Eosinophilic Pneumonia

Acute eosinophilic pneumonia (AEP) is an acute, hypersensitivity-like reaction resulting in eosinophilic infiltration of the lung parenchyma. Common inciting factors include parasitic infection, drug hypersensitivity, tobacco use, and systemic eosinophilic disorders [14]. AEP presents with fever, hypoxia, diffuse pulmonary infiltrates, and eosinophilia. Symptoms develop over the course of hours, days, or weeks [15]. Diagnostic criteria include an acute respiratory illness, bilateral pulmonary infiltrates, hypoxemia, and greater than 25% eosinophils on bronchoalveolar lavage [16, 17]. AEP is treated with corticosteroids and supportive care and frequently requires mechanical ventilation [18–20].

In 2004, 18 cases of AEP were described among personnel evacuated from Iraq. Most of the patients were men and reported dust exposure. All used tobacco [19]. A follow-up study described 43 additional cases of AEP among military members from 2003 to 2014 [20]. Nearly 90% of the patients smoked, and more than 70% had recently started smoking or increased cigarette use. This is consistent with the known association of AEP and tobacco use [21]. All patients required oxygen supplementation or mechanical ventilation and were evacuated from theater.

The diagnosis may be delayed in the deployed setting due to limited access to bronchoscopy and laboratory resources. In patients with a history suggestive of AEP, early empiric antibiotics and steroids should be considered. AE to higher levels of care is mandatory.

Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS) is a clinical syndrome characterized by diffuse lung inflammation and hypoxic respiratory failure [21]. Trauma and trauma resuscitation are the most common etiologies for ARDS in the deployed setting. Other etiologies include inhalational injuries, infection, and pancreatitis [22]. Treatment of ARDS in the deployed setting is similar to that employed elsewhere [22] with an emphasis on treatment of the underlying etiology, lung protective ventilation, and conservative fluid management. In the downrange setting, ARDS is most commonly associated with combat trauma. A review of the U.S. DOD trauma registry revealed approximately 3% of trauma patients developed

ARDS. Risk factors for developing ARDS included female sex, more severe injuries, hypotension, and tachycardia on presentation [23]. Patients with ARDS also had higher risk of death and used more resources. Prolonged respiratory support and rehabilitation preclude maintaining these patients in theater [3]. Severe ARDS is the most common reason for activation of the ALRT [4].

Inhalation Injuries

Chemical Weapon Exposure

Chemical weapons have a storied history. They have been used in numerous conflicts throughout history and were used by Iraq in the Iraq-Iran war and against Kurdish Iraqi citizens [24••]. The threat of chemical weapon exposures played a prominent role in the planning and strategies of the first Gulf War and the most recent conflicts in Iraq and Afghanistan. In these conflicts, there have been multiple reports of individual exposures, but the medical literature regarding these exposures is limited [25–27].

Classically, chemical agents are classified into choking agents, blood agents, blister agents, and nerve agents. Choking agents are sub-categorized into centrally acting agents and peripherally acting agents. Centrally acting agents, such as chlorine, are highly water soluble and mainly affect the upper airway, larynx, trachea, and large airways; symptoms begin soon after exposure. Peripherally acting agents such as phosgene are less water soluble, affect the distal airways, and often have a delayed onset of symptoms [28]. Management of inhalation injuries requires early recognition, limiting additional exposure, and respiratory support. Definitive therapies are limited.

Chlorine Exposure

Chlorine is a readily available chemical used for sanitation and for public works projects. When combined with explosives, chlorine can be vaporized and dispersed as a chemical weapon. Acute exposure causes bronchoconstriction resulting in wheezing, coughing, and chest tightness. Hypoxemia and infiltrates may also be present. Treatments include respiratory support, bronchodilators, and inhaled corticosteroids [28, 29]. Inhaled sodium bicarbonate may scavenge chlorine free radicals and minimize long-term effects [30]. Long-term sequelae from chlorine exposure include reactive airway dysfunction syndrome (RADS) and bronchiolitis obliterans syndrome [31].

The use of chlorine tankers in vehicle-based improvised explosive devices has occurred on multiple occasions during the recent conflicts [29, 32–34]. Additionally, attacks conducted with "chlorine-filled rockets" have been reported [35]. Although the clinical course of service members exposed to chlorine is unavailable, news reports cite substantial civilian and military casualties from chlorine exposure [29, 32, 35, 36]. Given the easy accessibility and facile disbursement, weaponized chlorine has and will continue to play an important role in military operations.

Phosgene

Phosgene is the prototypical peripheral choking agent and has previously been used in numerous conflicts. Phosgene is a chemical solvent widely used by the pharmaceutical and chemical industries [24••, 37]. Nasopharyngeal pain and irritation are the initial complaints. Over several hours, progressive dyspnea, cough, and foamy sputum production will develop. Hypoxia on room air is predictive of lung injury and ARDS [24••]. No specific antidote exists for phosgene toxicity. Treatment is limited to supportive care and lung-protective ventilation [38], though the administration of corticosteroids and nebulized *N*-acetylcysteine may be beneficial [39].

There are no recent reports of phosgene exposure in military members in Southwest Asia; however, due to its relatively common availability and potential for significant harm, phosgene remains an agent of clinical and tactical concern.

Hydrofluoric Acid

Heptafluoropropane is a hydrofluorocarbon used for fire suppression and was part of the automated fire suppression systems in military vehicles. At high temperatures, heptafluoropropane breaks down to hydrofluoric acid. Vaporized hydrofluoric acid from vehicle fires has been linked to two soldier deaths and two patients requiring ALRT evacuation [40]. The soldiers presented in respiratory distress and had diffuse peripheral lung infiltrates on radiograph. Treatments include decontamination and early respiratory support. Nebulized calcium and sodium bicarbonate may be beneficial [40]. A heptafluoropropane-based fire suppression system continues to be used in military vehicles, and therefore, there remains a potential inhalational hazard to military personnel.

Sulfur Exposures

Sulfur dioxide (SO_2) and hydrogen sulfide (H_2S) result from the combustion of elemental sulfur. SO_2 is water soluble and causes skin and airway irritation [41]. H_2S is a toxic gas that is a major contaminant of natural gas reservoirs [42]. Intense exposure to SO_2 and H_2S causes upper airway obstruction, laryngospasm, hypoxemia, and pulmonary edema [41, 43]. Therapy for acute exposure includes minimizing additional exposure, bronchodilators, and respiratory support [41].

In 2003, the Al-Mishraq sulfur mine fire potentially exposed thousands of U.S. troops to high levels of SO_2 and H_2S [44]. Firefighters and support elements had the highest

exposure; however, no serious health consequences were noted [45]. No long-term sequelae were clearly identified in these firefighters; however, there is a controversial association between exposure to this event and constrictive bronchiolitis and RADS [46]. The mine was reignited in October 2016 during the ongoing battle for Mosul [50]. Media reports associated with the 2016 fire indicate there have been 2 civilian deaths and more than 1000 respiratory complaints from the fires [47]. U.S. and coalition troops remain in the area and are at high risk for exposure to these agents.

Mustard Agents

Mustard agents are strongly lipophilic liquids that can be dispersed by explosive blasts and contaminate numerous individuals. Brief high-intensity exposures cause skin and eye injury. Aerosolized exposure can provoke respiratory failure. Tachypnea, bronchospasm, increased secretions, and a productive cough are common respiratory complaints after intense exposures [48]. Treatment includes decontamination, use of sodium thiosulfate, vitamin E, and steroids soon after exposure. Mechanical ventilation is necessary for respiratory compromise [48].

The medical literature cites one case of a soldier being exposed to chemical agents in Iraq [48]; however, there are numerous news reports of other exposures, and weaponized mustard agents have been uncovered throughout Iraq [25–27]. In addition, the Islamic State of Iraq and the Levant (ISIL) have recently used mustard agents against U.S. troops [49, 50]. The non-traditional tactics and adversaries faced in the current conflicts make exposure to banned chemical warfare agents more likely.

Nerve Agents

Nerve agents inhibit synaptic cholinesterase activity and cause unopposed cholinergic activity [51]. Aerosolized agents can be absorbed through the skin or respiratory tract and then distributed throughout the body. Symptoms include meiosis, rhinorrhea, salivation, bronchostriction, and bronchorrhea. Cardiovascular autonomic lability, coma, seizures, and central apnea are also reported [24••, 52]. The diagnosis of nerve agent exposure is based on clinical presentation and history. Therapy involves decontamination, respiratory support, and pharmacologic therapy with atropine, pralidoxime chloride, and diazepam [24••].

During the First Gulf War, service members were exposed to sarin while destroying a munitions depot in Khamisiyah, Iraq, in 1991 [53]. In the current conflict, there are no reports in the medical literature of nerve agents causing military casualties. However, several soldiers attempting to disarm warheads had symptoms suggestive of nerve agent exposure [25, 26]. Sarin was recently used in the region, and there is concern ISIL may acquire old nerve agent munitions [54].

Inhalation Burns

Burn injury often requires extensive resuscitation and hemodynamic monitoring. Although burn injuries often present in patients with traumatic injuries, inhalational burn injuries are not isolated to traumatic incidents. Reviews of military combat trauma and burn care can be found elsewhere [55, 56•].

Inhalational burns are categorized into flash burns or smoke inhalation injuries. Flash burns expose the upper airways to thermal injury. Smoke inhalation injury involves chemical burns of the lower airway. In both scenarios, the airway epithelium is denuded and reactive pulmonary changes occur. The subsequent edema and inflammation can cause an exudative cast formation of denuded epithelium [55]. Common inhalation burn manifestations include laryngeal edema and respiratory compromise. Smoke inhalation can result in bronchoconstriction and pulmonary edema [55]. Individuals with facial burns require airway visualization for assessment of inhalation injury [57]. Treatment options including endotracheal intubation for airway protection, routine use of inhaled heparin/*N*-acetylcysteine, chest physiotherapy, and bronchoscopies to improve airway hygiene have been suggested [58–60].

Between 2003 and 2005, burn injuries accounted for 5% of theater evacuations. Facial burns were seen in 77% of those patients and 26% had inhalation injuries [61]. Given the lack of necessary equipment to diagnose airway injuries at echelon I and II facilities, prophylactic intubation and subsequent AE is necessary in any patient with suspected inhalational injuries [55].

RADS

Reactive airway dysfunction syndrome (RADS) is an acute-onset respiratory syndrome occurring minutes to hours after a single high-intensity exposure to a respiratory irritant. Following the exposure, an asthma-like syndrome with airway hyperreactivity persists. Traditional RADS diagnostic criteria include symptoms within 24 h of exposure and a positive methacholine challenge test [62]. The current diagnostic criteria also require airway hyperreactivity for greater than 3 months [62]. Sulfuric acid, ammonia, chlorine, diesel, fire/smoke, mustard gas, and sulfur dioxide have all been associated with RADS [63, 64]. With an acute presentation, the differential may include pneumonia, asthma, non-cardiogenic pulmonary edema, and AEP.

While the incidence of RADS is difficult to ascertain in deployed personnel, numerous service members have developed symptoms following an acute high-intensity exposure to inhalational irritants. Deployed individuals are frequently exposed to many of the irritants known to cause RADS. RADS

should also be a consideration in any mass causality situation with fire, gas, or munition exposure. Treatment consists of corticosteroids and bronchodilators [65].

Asthma

Prior to 2003, individuals with asthma were not eligible for military service. Currently, individuals with asthma are permitted to enter the military with a proper waiver and may be deployed if their symptoms are well controlled [66]. A post-deployment survey of 1200 soldiers returning from Southwest Asia showed that 61 (5%) reported being asthmatics [67]. Those with asthma experienced more respiratory complaints than non-asthmatics. Half of asthmatics sought treatment for respiratory complaints while deployed, and 10 (16%) reported missing duty days or being hospitalized for respiratory symptoms [67]. A review of ICD-9 codes in 6000 charts at a single VA center suggested there were higher rates of "new asthma" in soldiers who had been deployed between 2003 and 2007 than those who had not been (6.6 vs 4.3%) [68].

Deployed service members are exposed to airborne particulate matter (PM) from geological dusts, burn pit emissions, wasted munitions, and regional pollutants. Many service members complain of respiratory symptoms while deployed [69, 70]; however, this is particularly true for asthmatics [67]. The perpetual exposure to environmental irritants increases the likelihood of asthma exacerbations. Most echelon I and II facilities have the required medications and equipment to treat mild exacerbations; however, these facilities lack the manpower and expertise to manage severe exacerbations. Symptoms will not improve if patients are not removed from the irritation. Given these factors, most moderate and severe asthma exacerbations are evacuated from theater.

Pulmonary Infections

Community-acquired pneumonia, influenza, and theater-specific organisms affect numerous service members in the deployed setting. These infections can cause severe respiratory illnesses leading to septic shock and respiratory failure. At the height of the conflicts in Southwest Asia, there were multiple evacuations every month for severe respiratory infections [71]. The topics included here represent a small subset of potential infectious etiologies that have been linked to respiratory failure and required evacuation from theater. Common infectious agents are not included.

Q-Fever

Q-fever is a zoonotic infection caused by inhalation of *Coxiella burnetti*. It is highly infectious and is considered a category B biologic agent [72]. Q-fever often develops after individuals are in close proximity to cattle, goats, and sheep. Acute infections present with a flu-like illness that can progress to severe pneumonia and acute hepatitis. Other manifestations include meningoencephalitis, endocarditis, and osteomyelitis [73]. Serologic testing is required for a diagnosis; however, the testing is not available in theater [72].

Q-fever markedly impacted military operations in Iraq [74, 75]. Serologic evaluations of 909 service members with febrile illnesses showed 10% likely had Q-fever and 22 members required AE out of theater [76]. In 2005, a marine platoon suffered an outbreak of Q-fever. No marine required evacuation from theater, but 64 duty days were lost due to the illness [74]. Subsequent reports describe Q-fever-associated cholecystitis and ARDS in deployed service members [75]. The treatment of choice is doxycycline. Given the outbreak potential and the difficulty in diagnosing Q-fever, doxycycline is often added empirically to treat respiratory infections of soldiers in theater [66].

Viral Infections

Numerous viral infections have been reported in the deployed environment. Surveillance conducted by the Medical Surveillance Monthly Report has attempted to quantify the prevalence of significant infections, but the data is from retrospective chart reviews without PCR testing. A serologic analysis of 1000 troops deployed to Southwest Asia revealed that while deployed over 30% seroconverted to at least one of the following infections: influenza, *Bordetella pertussis*, *Chlamydia pneumoniae*, *Mycoplasma pneumoniae*, adenovirus, parainfluenza, or respiratory syncytial virus [77•]. During the H1N1 influenza pandemic, 6344 Afghan soldiers were seen for respiratory complaints and 319 were deemed to be infected with H1N1. Sixty-one of these soldiers were evacuated to higher levels of care, and 58 of those 61 required treatment for severe pneumonia [78]. Viral infections decrease individual and unit readiness and in the setting of limited resources often prompt AE to higher levels of care.

Hemorrhagic Fever

Crimean-Congo hemorrhagic fever (CCHF) is a life-threatening viral illness endemic to Africa and the Middle East. Caused by an infection from a tick or contact with blood/bodily fluids of infected animals, the disease presents with high fevers, followed by an altered mental status and hemorrhagic manifestations. There is estimated 30–50% mortality within 14 days of symptom onset. Ribavarin therapy and supportive care are the standard treatments. In addition to therapy for an infected individual, infection control measures must be undertaken to limit exposure.

One case of CCHF has been reported in an American soldier. The soldier was serving in Afghanistan and had been exposed to tick bites and undercooked goat meat. He initially

sought treatment for 4 days of diarrhea and hematemesis. He soon developed bloody diarrhea, gingival bleeding, hypoxia, and alveolar hemorrhage. He was transferred to an echelon IV facility where he received numerous units of blood and was started on Ribavarin therapy. The patient died from massive cerebral edema and herniation after 7 days in the ICU [79].

A high degree of suspicion is required to diagnose and expedite AE of patients with viral hemorrhagic fever. The multi-organ system failure, respiratory support, and intensive laboratory requirements preclude managing these patients in the deployed environment. Additionally, vigorous infection control and outbreak control measures must be managed throughout AE and at higher echelons of care.

Acinetobacter Infection

Acinetobacter is a gram-negative coccobacillus native to the soil of Southwest Asia. It readily colonizes the skin, wounds, and respiratory and gastrointestinal tracts [80]. It readily develops antimicrobial resistance making nosocomial strains particularly concerning. *Acinetobacter* has caused numerous ventilator-associated pneumonias and wound-site infections [80]. Rarely, primary pneumonias are caused by *Acinetobacter*, but it more commonly causes secondary infections in patients being mechanically ventilated or with open wounds [81, 82]. Treatment options are guided based on susceptibilities, most commonly with use of carbapenem and polymyxin therapy [81–83].

Conclusion

In the recent conflicts in Iraq and Afghanistan, nearly 2000 service members have been evacuated for pulmonary-related conditions. With an array of underlying etiologies, the diagnostic and treatment capabilities of these patients in the deployed environment are often limited. Awareness of the potential pulmonary diseases affecting soldiers downrange is crucial to optimizing health care in the deployed environment.

Compliance with Ethical Standards

Conflict of Interest Steven Deas, Andrew Skabelund, and Nikhil Huprikar declare no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1.• Crawley PG. The critical care air transport experience. Current Pulmonology Reports. 2016;5(2):77–85. **This article provides an overview of critical care transport capabilities in U.S. military conflicts but also highlights the limitations of the various echelons of care both in the deployed environment and international locations.**
2. Grathwohl KW, Venticinque SG. Organizational characteristics of the austere intensive care unit: the evolution of military trauma and critical care medicine; applications for civilian medical care systems. Crit Care Med. 2008;36(Suppl):S275–S83. doi:10.1097/CCM.0b013e31817da825.
3. Venticinque SG, Grathwohl KW. Critical care in the austere environment: providing exceptional care in unusual places. Crit Care Med. 2008;36(7):S284–S92.
4. Cancio LC, Horvath EE, Barillo DJ, Kopchinski BJ, Charter KR, Montalvo AE, et al. Burn support for Operation Iraqi Freedom and related operations, 2003 to 2004. Journal of Burn Care & Research. 2005;26(2):151–61.
5.• Bein T, Zonies D, Philipp A, Zimmermann M, Osborn EC, Allan PF, et al. Transportable extracorporeal lung support for rescue of severe respiratory failure in combat casualties. J Trauma Acute Care Surg. 2012;73(6):1450–6. The **article provides a brief overview of the utility of extracorporeal lung support in patients deployed internationally.**
6. Center AFHS. Causes of medical evacuations from Operations Iraqi Freedom (OIF), New Dawn (OND) and Enduring Freedom (OEF), active and reserve components, US Armed Forces, October 2001-September 2010. MSMR. 2011;18(2):2.
7. Center AFHS. Medical evacuations from Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF), active and reserve components, U.S. Armed Forces, October 2001-September 2009. MSMR. 2010;17(2):2–7.
8. Murray CK, Reynolds JC, Schroeder JM, Harrison MB, Evans OM, Hospenthal DR. Spectrum of care provided at an echelon II medical unit during Operation Iraqi Freedom. Mil Med. 2005;170(6):516–20.
9. Harman DR, Hooper TI, Gackstetter GD. Aeromedical evacuations from Operation Iraqi Freedom: a descriptive study. Mil Med. 2005;170(6):521–7.
10. Lundy JB, Swift CB, McFarland CC, Mahoney LCP, Perkins RM, Holcomb JB. A descriptive analysis of patients admitted to the intensive care unit of the 10th combat support hospital deployed in Ibn Sina, Baghdad, Iraq, from October 19, 2005, to October 19, 2006. J Intensive Care Med. 2010;25(3):156–62. doi:10.1177/0885066609359588.
11. Center AFHS. Medical evacuations from Afghanistan during Operation Enduring Freedom, active and reserve components, U.S. Armed Forces, 7 October 2001-31 December 2012. MSMR. 2013;20(6):2–8.
12. Maddry JK, Arana AA, Reeves LK, Perez CA, Mora AG, Savell SC, Bebarta VS. An evaluation of critically ill non-trauma patients evacuated by CCATT. Pending. Pending;In Preparation.
13. Wojcik BE, Humphrey RJ, Czejdo B, Hassell LH. US Army disease and nonbattle injury model, refined in Afghanistan and Iraq. Mil Med. 2008;173(9):825–35.
14. Philit F, Etienne-Mastroianni B, Parrot A, Guerin C, Robert D, Cordier JF. Idiopathic acute eosinophilic pneumonia: a study of

22 patients. Am J Respir Crit Care Med. 2002;166(9):1235–9. doi:10.1164/rccm.2112056.
15. Allen JN, Pacht ER, Gadek JE, Davis WB. Acute eosinophilic pneumonia as a reversible cause of noninfectious respiratory failure. N Engl J Med. 1989;321(9):569–74.
16. Janz DR, O'Neal HRJ, Ely EW. Acute eosinophilic pneumonia: a case report and review of the literature. Crit Care Med. 2009;37(4):1470–4. doi:10.1097/CCM.0b013e31819cc502.
17. Thota D, Latham E. Case report of electronic cigarettes possibly associated with eosinophilic pneumonitis in a previously healthy active-duty sailor. The Journal of Emergency Medicine. 2014;47(1):15–7. doi:10.1016/j.jemermed.2013.09.034.
18. Ajani S, Kennedy CC. Idiopathic acute eosinophilic pneumonia: a retrospective case series and review of the literature. Respiratory Medicine Case Reports. 2013;10:43–7. doi:10.1016/j.rmcr.2013.06.005.
19. Takeuchi A, Nelson C, Yamamoto I, Yamashiro S, Myers J. Acute eosinophilic pneumonia after resumption of cigarette smoking. Mil Med. 2016;181(6):e613–e5. doi:10.7205/MILMED-D-15-00300.
20. Shorr AF, Scoville SL, Cersovsky SB, et al. Acute eosinophilic pneumonia among US military personnel deployed in or near iraq. JAMA. 2004;292(24):2997–3005. doi:10.1001/jama.292.24.2997.
21. Force ADT. Acute respiratory distress syndrome. JAMA. 2012;307(23):2526–33.
22. Morris MJ. Acute respiratory distress syndrome in combat casualties: military medicine and advances in mechanical ventilation. Mil Med. 2006;171(11):1039–44.
23. Park PK, Cannon JW, Ye W, Blackbourne LH, Holcomb JB, Beninati W, et al. Incidence, risk factors, and mortality associated with acute respiratory distress syndrome in combat casualty care. J Trauma Acute Care Surg. 2016;81(5):S150–S6.
24.•• Chalela JA, Burnett WT. Chemical terrorism for the intensivist. Mil Med. 2012;177(5):495–500. **This review provides clinical aspects associated with diagnosis and treating victims of chemical attacks.**
25. Chivers CJ. The secret casualties of Iraq's abandoned chemical weapons. New York Times. 2014. https://www.nytimes.com/interactive/2014/10/14/world/middleeast/us-casualties-of-iraq-chemical-weapons.html?_r=0.
26. Schmitt E. ISIS used chemical weapons in Syria and Iraq at least 52 times, report says. New York Times. 2016. https://www.nytimes.com/2016/11/21/world/middleeast/isis-chemical-weapons-syria-iraq-mosul.html.
27. Agency CI. Detailed preliminary assessment of chemical weapons findings. In: Iraq's chemical weapons program - Annex F. 2007. https://www.cia.gov/library/resports/general-reports-1/iraq_wmd_2004/chap5_annexF.html. Accessed Dec 17 2016.
28. Kales SN, Christiani DC. Acute chemical emergencies. N Engl J Med. 2004;350(8):800–8. doi:10.1056/NEJMra030370.
29. Jones R, Wills B, Kang C. Chlorine gas: an evolving hazardous material threat and unconventional weapon. Western J Emerg Med. 2010; 11(2):151–156
30. Bosse GM. Nebulized sodium bicarbonate in the treatment of chlorine gas inhalation. J Toxicol Clin Toxicol. 1994;32(3):233–41.
31. Jones RN, Hughes JM, Glindmeyer H, Weill H. Lung function after acute chlorine exposure 1–3. Am Rev Respir Dis. 1986;134(6):1190–5.
32. Maher A. Islamic State 'using chlorine gas' in Iraq roadside bombs. BBC. 2015. http://www.bbc.com/news/world-middle-east-31847427.
33. Cave DF, Ahmad. Iraq militants use chlorine in 3 bombings. The New York Times. 2007. http://www.nytimes.com/2007/02/21/world/middleeast/21cnd-baghdad.html.
34. Johnston WR. Summary of historical attacks using chemical or biological weapons. 2016. http://www.johnstonsarchive.net.
35. Batchelor T. ISIS targets civilians with chemical weapon attack in Iraq and Syria. Express. 2015. http://www.express.co.uk/news/world/592379/Islamic-State-chemical-weapon-attack-jihaditerrorists-chlorine-rockets.
36. Cave D, Fadam A. Iraq militants use chlorine in 3 bombings. The New York Times. 2007. http://www.nytimes.com/2007/02/21/world/middleeast/21cnd-baghdad.html.
37. Borak J, Diller WF. Phosgene exposure: mechanisms of injury and treatment strategies. J Occup Environ Med. 2001;43(2):110–9.
38. Parkhouse DA, Brown RF, Jugg BJ, Harban FM, Platt J, Kenward CE, et al. Protective ventilation strategies in the management of phosgene-induced acute lung injury. Mil Med. 2007;172(3):295–300.
39. Grainge C, Rice P. Management of phosgene-induced acute lung injury. Clin Toxicol. 2010;48(6):497–508.
40. Zierold D, Chauviere M. Hydrogen fluoride inhalation injury because of a fire suppression system. Mil Med. 2012;177(1):108–12.
41. Registry AfTSaD. Sulfur dioxide: Center for Disease Control 2015. https://www.atsdr.cdc.gov/toxprofiles/TP.asp?id=253&tid=46.
42. Warenycia MW, Goodwin LR, Benishin CG, Reiffenstein R, Francom DM, Taylor JD, et al. Acute hydrogen sulfide poisoning: demonstration of selective uptake of sulfide by the brainstem by measurement of brain sulfide levels. Biochem Pharmacol. 1989;38(6):973–81.
43. Snyder JW, Safir EF, Summerville GP, Middleberg RA. Occupational fatality and persistent neurological sequelae after mass exposure to hydrogen sulfide. Am J Emerg Med. 1995;13(2):199–203.
44. Affairs USDoV. Sulfur fire at Mishraq State Sulfur Mine. Online 2015. https://www.publichealth.va.gov/exposures/mishraq-sulfur-fire/index.asp.
45. U.S. Army Public Health Command EMP. Health assessment of 2003 Al Mishraq sulfur fire incident. In: USAPHC, editor. Online 2012.
46. King MS, Eisenberg R, Newman JH, Tolle JJ, Harrell Jr FE, Nian H, et al. Constrictive bronchiolitis in soldiers returning from Iraq and Afghanistan. N Engl J Med. 2011;365(3):222–30.
47. BBC. Mosul battle: hundreds treated over toxic fumes in Iraq. BBC. 2016. http://www.bbc.com/news/world-middle-east-37738667.
48. Goverman J, Montecino R, Ibrahim A, Sarhane KA, Tompkins RG, Fagan SP. Sulfur mustard gas exposure: case report and review of the literature. Annals of Burns and Fire Disasters. 2014;27(3):146–50.
49. Phippen JW. A mustard gas attack on U.S. troop: a general confirmed that Islamic State fighters used the chemical weapon this week in an attack on American soldiers in Iraq. The Atlantic. 2016.
50. Michaels J. Pentagon confirms ISIL tried to use mustard gas on U.S. troops. USA Today. 2016.
51. Brown M. Military chemical warfare agent human subjects testing: part 2-long-term health effects among participants of US military chemical warfare agent testing. Mil Med. 2009;174(10):1049–54.
52. Cosar A, Kenar L. An anesthesiological approach to nerve agent victims. Mil Med. 2006;171(1):7–11.
53. Proctor SP, Heaton KJ, Heeren T, White RF. Effects of sarin and cyclosarin exposure during the 1991 Gulf War on neurobehavioral functioning in US army veterans. Neurotoxicology. 2006;27(6):931–9. doi:10.1016/j.neuro.2006.08.001.
54. Charbonneau LN, Michelle. U.N. confirms sarin used in Syria attack; U.S., UK, France blame Assad. Reuters. 2013.
55. Boyer NL, Forbes DA, Chung KK. Practical management of burns and inhalation injury. Current Pulmonology Reports. 2016;5(2):63–9.
56.• Walker PF, Buehner MF, Wood LA, Boyer NL, Driscoll IR, Lundy JB, et al. Diagnosis and management of inhalation injury: an updated review. Crit Care. 2015;19(1):1. **Review of recent advances in diagnosis and treatment of inhalation injuries**

57. Kwon HP, Zanders TB, Regn DD, Burkett SE, Ward JA, Nguyen R, et al. Comparison of virtual bronchoscopy to fiber-optic bronchoscopy for assessment of inhalation injury severity. Burns. 2014;40(7):1308–15. doi:10.1016/j.burns.2014.06.007.
58. Ennis JL, Chung KK, Renz EM, Barillo DJ, Albrecht MC, Jones JA, et al. Joint Theater Trauma System implementation of burn resuscitation guidelines improves outcomes in severely burned military casualties. J Trauma Acute Care Surg. 2008;64(2):S146–S52.
59. Mlcak RP, Suman OE, Herndon DN. Respiratory management of inhalation injury. Burns. 2007;33(1):2–13.
60. Carr JA, Phillips BD, Bowling WM. The utility of bronchoscopy after inhalation injury complicated by pneumonia in burn patients: results from the National Burn Repository. Journal of Burn Care & Research. 2009;30(6):967–74.
61. Kauvar DS, Wolf SE, Wade CE, Cancio LC, Renz EM, Holcomb JB. Burns sustained in combat explosions in Operations Iraqi and Enduring Freedom (OIF/OEF explosion burns). Burns. 2006;32(7):853–7.
62. Brooks SM, Weiss MA, Bernstein I. Reactive airways dysfunction syndrome (RADS): persistent asthma syndrome after high level irritant exposures. Chest. 1985;88(3):376–84.
63. White CW, Martin JG. Chlorine gas inhalation: human clinical evidence of toxicity and experience in animal models. Proc Am Thorac Soc. 2010;7(4):257–63.
64. Alberts WM, Guillermo A. Reactive airways dysfunction syndrome. CHEST Journal. 1996;109(6):1618–26.
65. Bardana EJ. Reactive airways dysfunction syndrome (RADS): guidelines for diagnosis and treatment and insight into likely prognosis. Annals of Allergy, Asthma & Immunology. 1999;83(6):583–6.
66. Regulation A. Standards of medical fitness. Headquarters, Department of the Army Washington, DC; 2004.
67. Roop SA, Niven AS, Calvin BE, Bader J, Zacher LL. The prevalence and impact of respiratory symptoms in asthmatics and nonasthmatics during deployment. Mil Med. 2007;172(12):1264–9.
68. Szema AM, Peters MC, Weissinger KM, Gagliano CA, Chen JJ, editors. New-onset asthma among soldiers serving in Iraq and Afghanistan. Allergy and Asthma Proceedings; 2010: OceanSide Publications, Inc.
69. Smith B, Wong CA, Boyko EJ, Phillips CJ, Gackstetter GD, Ryan MAK, et al. The effects of exposure to documented open-air burn pits on respiratory health among deployers of the Millennium Cohort Study. J Occup Environ Med. 2012;54(6):708–16. doi:10.1097/JOM.0b013e31825107f9.
70. Smith B, Wong CA, Smith TC, Boyko EJ, Gackstetter GD. Newly reported respiratory symptoms and conditions among military personnel deployed to Iraq and Afghanistan: a prospective population-based study. Am J Epidemiol. 2009;170(11):1433–42.
71. Center AFHS. Deployment-related conditions of special surveillance interest, U.S. Armed Forces, by month and service, January 2003 - October 2009 MSMR. 2009; 16(11):16–9.
72. Hartzell JD, Gleeson T, Scoville S, Massung RF, Wortmann G, Martin GJ. Practice guidelines for the diagnosis and management of patients with Q fever by the Armed Forces Infectious Diseases Society. Mil Med. 2012;177(5):484–94.
73. Wallace MR, Hale BR, Utz GC, Olson PE, Earhart KC, Thornton SA, et al. Endemic infectious diseases of Afghanistan. Clin Infect Dis. 2002;34(Supplement 5):S171–207. doi:10.1086/340704.
74. Faix DJ, Harrison DJ, Riddle MS, Vaughn AF, Yingst SL, Earhart K, et al. Outbreak of Q fever among US military in western Iraq, June–July 2005. Clin Infect Dis. 2008;46(7):e65–e8. doi:10.1086/528866.
75. Hartzell JD, Peng SW, Wood-Morris RN, Sarmiento DM, Collen JF, Robben PM, et al. Atypical Q fever in US soldiers. Emerg Infect Dis. 2007;13(8):1247.
76. Anderson AD, Baker TR, Littrell AC, Mott RL, Niebuhr DW, Smoak BL. Seroepidemiologic survey for Coxiella burnetii among hospitalized US troops deployed to Iraq*. Zoonoses Public Health. 2011;58(4):276–83. doi:10.1111/j.1863-2378.2010.01347.x.
77.• Eick AA, Faix DJ, Tobler SK, Nevin RL, Lindler LE, Hu Z, et al. Serosurvey of bacterial and viral respiratory pathogens among deployed US service members. American Journal of Preventive Medicine. 2011;41(6):573–80. **An evaluation of respiratory pathologies responsible for infections in soldiers from 2004 to 2009. This article provides insights regarding potential infectious exposure agents in the deployed environment.**
78. Center AFHS. Preliminary report: investigation and control of novel H1N1 influenza among soldiers of the Afghan National Army, Kabul, Afghanistan, 30 October - 14 November 2009. MSMR. 2009;16(11):8–11.
79. Conger NG, Paolino KM, Osborn EC, Rusnak JM, Günther S, Pool J, et al. Health care response to CCHF in US soldier and nosocomial transmission to health care providers, Germany, 2009. Emerging Infectious Disease journal. 2015;21(1):23. doi:10.3201/eid2101.141413.
80. Munoz-Price LS, Weinstein RA. Acinetobacter infection. N Engl J Med. 2008;358(12):1271–81. doi:10.1056/NEJMra070741.
81. Yun HC, Weintrob AC, Conger NG, Li P, Lu D, Tribble DR, et al. Healthcare-associated pneumonia among U.S. combat casualties, 2009 to 2010. Mil Med. 2015;180(1):104–10. doi:10.7205/MILMED-D-14-00209.
82. Murray CK, Hospenthal DR. Treatment of multidrug resistant Acinetobacter. Curr Opin Infect Dis. 2005;18(6):502–6.
83. Eliopoulos GM, Maragakis LL, Perl TM. Acinetobacter baumannii: epidemiology, antimicrobial resistance, and treatment options. Clin Infect Dis. 2008;46(8):1254–63. doi:10.1086/529198.