

INDEPENDENT MEDICAL EXPERT (IME) NEXUS-OPINION

19 August 2014

To: Veterans Administration (VA)

Re: [REDACTED]

As my attached curriculum vitae indicates [EXHIBIT 1], I am a surgeon with almost thirty years of medical experience. I was Clinical Associate Professor of Surgery and Attending Surgeon in Transplantation at SUNY at Stony Brook. I served as President of the New York Transplantation Society and as Assistant Editor of Transplantation Proceedings. I hold three patents. I have authored three book chapters and 106 research papers published in peer reviewed medical journals.

Mr. [REDACTED] ("Veteran") served in the Navy during the Gulf War Era, from December 4, 2002, to April 30, 2012. The rating decision of July 3, 2013, granted service connection for lumbar spondylolysis L5-S1 with a 10% disability rating. Service connection for allergic rhinitis and deviated septum were denied; the VA stated that these conditions did not exist during his service. The veteran also suffers from obstructive sleep apnea.

After reviewing the veteran's medical records, including service records, I find that the conditions of allergic rhinitis and deviated septum were indeed present during his military service. I also opine that it is more likely than not that the veteran's obstructive sleep apnea is secondary to nasal obstruction due to allergic rhinitis and deviated septum.

Review of the medical records

In 2007, while in service, the veteran was prescribed nasal sprays for the treatment of his nasal condition. A medical record from February 5, 2008, shows that Dr. Perrin Clark saw the veteran for nasal obstruction. On exam the veteran was found to have a deviated septum and the nasal turbinates were hypertrophied. The record also shows that the veteran was treated with nasal drops; the prescription was last filled in May of 2007. [EXHIBIT 2]

In 2009, while still in service, the veteran received care at Makalapa Clinic and the Tripler Army Medical Center (TAMC) in Honolulu, Hawaii. He was examined by Dr. Gwendolyn Nishimura on March 4, 2009, who diagnosed deviated nasal septum and recommended an evaluation at the ENT Clinic. He was also prescribed Nasonex. [EXHIBIT 3]

On March 11, 2009, the veteran was seen at the Ear, Nose and Throat Clinic at TAMC by Dr. Mitchell Ramsey for deviated septum. The veteran complained of a long history of difficulty breathing due to nasal obstruction, worse on the right; with complaints of sneezing, watery rhinorrhea, itchy eyes and palate. The ENT physician stated that the veteran has clear symptomatology of allergic rhinitis and a deviated septum to the right. He recommended medical management for two months, and if the nasal obstruction persisted then septoplasty/turbinoplasty would be considered. [EXHIBIT 4]

On November 2, 2009, the veteran was seen by Dr. Nathaniel Duff for allergy testing. Dr. Duff noted that the veteran was scheduled for a septo/turbino-plasty. On examination, he found the nasal septum deviated to the right with a spur on the left floor, and pale, swollen and edematous nasal mucosa. [EXHIBIT 5]

On March 9, 2012, the veteran was seen for a severe case of allergic rhinitis, sinus pain, cough, congestion, and nasal drainage. [EXHIBIT 6]

The veteran's military service separation physical on April 5, 2012, shows that he again complained of problems with breathing and allergies, "had septoplasty but c/o recurrent sinus pain and difficulty breathing secondary to obstruction. [EXHIBIT 7]

Veteran underwent a sleep study on June 12, 2014, by Dr. Sunit Patel who is Board Certified in Pulmonary Critical Care and Sleep Medicine. The polysomnography report revealed mild snoring, a total of one obstructive apnea and 37 hypopneas. The apneic event was 20.9 seconds in duration and the longest hypopnea was 30.5 seconds in duration. The lowest oxygen desaturation was 92%. These findings indicate a moderate form of obstructive sleep apnea. CPAP was initiated at a pressure of 4 cm and titrated up to 10 cm where improvement was noted. The veteran was also found to have an abnormal sleep architecture characterized by reduced

sleep efficiency, reduced sleep latency, increased stage R latency, reduced amount of stage R sleep, and sleep fragmentation. Treatment recommendations included BPAP therapy with 10/6 cm water. [EXHIBIT 8]

Review of the medical literature

Conceptually, the upper airway is a compliant tube and, therefore, is subject to collapse.ⁱ [EXHIBIT 9] OSA is caused by soft tissue collapse in the pharynx. Transmural pressure is the difference between intraluminal pressure and the surrounding tissue pressure. If transmural pressure decreases, the cross-sectional area of the pharynx decreases. If this pressure passes a critical point, pharyngeal closing pressure is reached. Exceeding pharyngeal critical pressure (Pcrit) causes a juggernaut of tissues collapsing inward. The airway is obstructed. Until forces change transmural pressure to a net tissue force that is less than Pcrit, the airway remains obstructed. OSA duration is equal to the time that Pcrit is exceeded.

The Bernoulli effect plays an important dynamic role in OSA pathophysiology. In accordance with this effect, airflow velocity increases at the site of stricture in the airway. As airway velocity increases, pressure on the lateral wall decreases. If the transmural closing pressure is reached, the airway collapses. The Bernoulli effect is exaggerated in areas where the airway is most compliant. Loads on the pharyngeal walls increase adherence and, hence, increase the likelihood of collapse. This effect helps to partially explain why obese patients, and particularly those with fat deposition in the neck, are most likely to have OSA.ⁱⁱ [EXHIBIT 10]

Given this information, it is abundantly clear that even a small reduction in a diameter of the upper airway will cause a collapse of the upper airway during sleep.

The effect of nasal breathing on sleep apnea was studied by Fitzpatrick et al., *Effect of nasal or oral breathing route on upper airway resistance during sleep*. [EXHIBIT 11] The author reports that healthy subjects with normal nasal resistance breathe almost exclusively through the nose during sleep. The researchers studied the resistance to the upper airway through either nasal or oral breathing and found that upper airway resistance during sleep and the propensity to obstructive sleep apnea are significantly lower while breathing nasally rather than orally. Nasal obstruction during sleep results in mouth opening and mouth opening has been

shown to increase the propensity to upper airway collapse. It has been shown that jaw opening is associated with posterior movement of the angle of the jaw, thus compromising the oropharyngeal airway diameter. This is caused by shortening of the upper airway dilator muscles located between the mandible and the hyoid bone. In addition, jaw opening profoundly affects the diameter of the retroglossal airway. The author has shown that there are two distinct sites of airway obstruction during sleep with oral breathing, when nasal breathing is not efficient.

Conclusion

It is clear from the veteran's service records that his medical conditions of deviated nasal septum and allergic rhinitis existed while he was in service. After review of the pertinent medical literature, I opine that the veteran's obstructive sleep apnea is secondary to nasal obstruction due to allergic rhinitis and deviated septum.

Sincerely,



David Anaise, JD, MD
Attorney at Law

DA /scb

Enclosures: EXHIBITS: (see attached list of exhibits)

ⁱ Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. Chest. Jul 2007;132(1):325-37. [Medline]. [Full Text].

ⁱⁱ Schwab RJ, Pasirstein M, Pierson R, Mackley A, Hachadoorian R, Arens R, et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. Am J Respir Crit Care Med. Sep 1 2003;168(5):522-30. [Medline].