

# Tinnitus, a Military Epidemic: Is Hyperbaric Oxygen Therapy the Answer?

LCDR Thomas M. Baldwin, MD, MPT

## ABSTRACT

Tinnitus is the phantom perception of sound in the absence of overt acoustic stimulation. Its impact on the military population is alarming. Annually, tinnitus is the most prevalent disability among new cases added to the Veterans Affairs numbers. Also, it is currently the most common disability from the War on Terror. Conventional medical treatments for tinnitus are well documented, but prove to be unsatisfying. Hyperbaric oxygen (HBO<sub>2</sub>) therapy may improve tinnitus, but the significance of the level of improvement is not clear. There is a case for large randomized trials of high methodological rigor in order to define the true extent of the benefit with the administration of HBO<sub>2</sub> therapy for tinnitus.

## THE PHYSIOLOGY OF HEARING

Hearing is a series of events in which sound waves in the air produce electrical signals and cause nerve impulses to be sent to the brain where they are interpreted as sound. The auditory system consists of the external, middle, and inner ears, as well as the central auditory pathways in the brain. Sound waves enter the external ear via the pinna and reach the middle ear where they strike the eardrum and cause it to vibrate. The vibrations set the middle-ear bones (malleus, incus, stapes) in motion. Movement of the stapes causes pressure waves in the fluid contained within the cochlea, which contains the organ of Corti, the sensory organ for hearing. The primary sensory receptors for hearing, the inner hair cells, are found within the organ of Corti as are the outer hair cells, which primarily facilitate the sensory response of the inner hair cells.<sup>1</sup> The fluid in the cochlea moves the top portion of the hair cells, called the hair bundle, which initiates the changes that lead to the production of the nerve impulses. The nerve fibers connected to the hair cells, primarily the inner hair cells, are excited and transfer the auditory information to the brain where they are interpreted as sound.<sup>1,2</sup>

## THE ETIOLOGY OF TINNITUS

Tinnitus, the perception of sound in the absence of an external source, is a chronic and debilitating condition often described as ringing, hissing, buzzing, chirping, high-pitched squealing, or roaring in the ears or in proximity to the head.<sup>2,4</sup> According to the National Research Council, tinnitus is considered a symptom rather than an illness.<sup>2</sup> The perceived noise can be within one or both ears, within or around the head, or perceived as an outside distant noise. It can be pulsatile or nonpulsatile and be continuous or occur in-

termittently. Tinnitus can be caused by or accompany many conditions, including presbycusis, Meniere's disease, otosclerosis, head injury, cerebellar-pontine angle tumors, otitis media, meningitis, dental disorders, and certain medications. However, most tinnitus is due to noise induced sensorineural hearing loss with resulting dysfunction within the auditory system.<sup>2,5,6</sup>

The presence of tinnitus often is an early indicator of cochlear hair cell dysfunction or loss, as in the case of excessive noise exposure.<sup>3</sup> The pathogenesis is assumed to consist of micromechanical traumatic and biochemical-metabolic damage to the outer hair cells.<sup>3</sup> Studies have shown how hair cells of the inner ear react to damage caused by noise.<sup>7,8</sup> In acoustic trauma, defined as an acute impairment of hearing caused by sharp sounds, like that of a gun going off, the partial pressure of oxygen decreases significantly in the fluid spaces of the inner ear.<sup>7</sup> Morphological damage results, leading to intra and extracellular ion imbalances and hearing damage. Histological findings are swelling and structural damage of the dendrites, alterations of mitochondria and the cell-structure, separation of hair-cells from tectorial membrane, oedema of the endothelium, and oedematous closure of functional endarteries with blocking of the microcirculation.<sup>7</sup> If the swelling persists for a prolonged period, the hair cells degenerate and are replaced by non-functioning endothelial cells.<sup>7,9</sup> PET scanning and functional MRI studies indicate that the loss of cochlear input to neurons in the central auditory pathways (such as occurs with cochlear hair cell damage due to noise trauma) can result in abnormal neural activity in the auditory cortex.<sup>3</sup> Such activity has been linked to tinnitus. It is important to note, that sounds of moderate intensity as encountered in everyday life usually do not affect the

oxygen tension within the cochlea.<sup>7</sup> As tinnitus is usually accompanied by hearing loss, similar mechanisms are likely involved.

### THE CHARACTERISTICS OF NOISE

Noise, defined medically as an intense sound capable of producing damage to the inner ear, leads to one of the most common conditions evaluated by otolaryngologist: noise induced hearing loss (NIHL). Noise can be further categorized as impulse noise, the product of explosive devices, or impact noise, caused by a collision of two hard surfaces. However described, both are produced by a sudden intense sound wave capable of causing inner ear damage. Excessive noise exposure is the most common cause of hearing loss.<sup>2,5,6</sup> When an individual is exposed to sounds that are too loud or loud sounds over a long period of time, sensitive structures of the inner ear can be damaged, resulting in NIHL. In humans, outer hair cells are usually the first type of sensory cell to be damaged.<sup>2</sup> As the hearing loss progresses and becomes more permanent, the degeneration involves both outer and inner hair cells. As the number of hair cells decreases, so does an individual's hearing. With severe permanent hearing losses, both sensory and supporting cells of the organ of Corti are missing. In these cases, the degenerative layer of the organ of Corti is replaced by an undifferentiated layer of squamous epithelium and the sensory nerve fibers are destroyed.<sup>9</sup> The type and amount of the resulting hearing loss are typically determined by the following acoustic parameters: the intensity of the noise, the duration of exposure to the noise, and the type of noise.

Intensity of sound is measured in units called decibels (dB), a measurement of the amount of energy or air pressure moving from the source to our ear.<sup>10,11,20</sup>

The faintest sound humans with normal hearing can detect has a value between zero and ten decibels, and the loudest sound the human ear can tolerate without pain is about 120 decibels.<sup>10</sup> Decibels are measured logarithmically, being 20 times the log of the ratio of a particular sound pressure to a reference sound pressure. This means that as decibel intensity increases by units of 20, each increase is 10 times the lower figure. Thus, 20 decibels is 10 times the intensity of 0 decibels, and 40 decibels is 100 times as intense as 20 decibels.<sup>20</sup> The Navy considers any sound above 84dB as noise hazardous and having the potential to cause hearing damage if it is loud enough or lasts long enough.<sup>4,5,10,12</sup> The higher the intensity of the sound, the greater its potential to cause hearing damage. Single exposures to impulse noises above 140 decibels have the potential to cause permanent damage.<sup>4</sup> According to the U.S. Army Center for Health and Preventative Medicine, a gunner on a 105 millimeter towed howitzer experiences an impulse noise of 183dB.<sup>2,4</sup> A servicemember who shoots a rifle is ex-

posed to 157-163dB and a gunner with a machine gun, 145dB.<sup>2,5,10,12</sup> Those suffering from an improvised explosive device (IED) are exposed to impulse noise in excess of 180dB.<sup>13</sup>

Duration is defined as the length of time you are exposed to a noise. The louder the sound and more prolonged the exposure, the shorter amount of time it takes to cause hearing damage. For unprotected ears, the allowed exposure time decreases by one half for each 5dB increase in the average noise level.<sup>5,14</sup> For instance, exposure is limited to eight hours per day at 90dB, four hours per day at 95dB, and two hours per day at 100dB. The highest permissible noise exposure for the unprotected ear is 115dB for 15 minutes per day.<sup>5,14</sup> Sounds of less than 75dB, even after long exposure, are unlikely to cause hearing loss.

Hearing loss that results from exposure to sound with energy spread across a wide range of frequencies, such as impulses common to military settings, is often characterized by a gradual increase in threshold as frequencies increase. The hearing loss typically reaches a maximum between 3000 and 6000 hertz (Hz), followed by a return toward normal hearing at still higher frequencies. This pattern of hearing loss is often referred to as the "noise-notch" audiogram and is a clinical hallmark often used to distinguish noise-related hearing loss from that associated with other etiologies, such as ototoxic medications or aging.<sup>2</sup>

### CHRONIC NOISED-INDUCED HEARING LOSS AND ACOUSTIC TRAUMA

Chronic NIHL is a disease process that occurs gradually over many years of exposure to less intense noise levels. It is generally caused by long term exposure to high intensity continuous noise with superimposed episodic impact or impulse noise. The hearing loss associated with chronic NIHL is variable between individuals, but the principal characteristics remain relatively consistent:<sup>14-17</sup>

- It is always sensorineural affecting the hair cells in the inner ear.
- It is nearly always bilateral and symmetric.
- It will only rarely produce a profound loss.
- It will not progress once noise exposure is stopped.
- The higher frequencies (3000-6000Hz) are more affected than the lower frequencies, with the greatest loss usually occurring at 4000Hz.
- Continuous noise is more damaging than intermittent noise.
- Tinnitus is often associated with NIHL.

One exception to these features would be the individual who had significant noise exposure secondary to rifle shooting. In this case, an asymmetrical loss, with the ear nearest the gun barrel demonstrating slightly worse hearing, would be expected.<sup>14</sup>

The development of chronic NIHL progresses through two phases. A brief hearing loss, more commonly referred to as a temporary threshold shift (TTS), characterizes the first stage. It occurs after noise exposure and completely resolves after a period of rest. Often reported as auditory fatigue, most studies indicate that it is associated with no sensory cell damage or minimal, reversible cell changes.<sup>14,18</sup> Eventually, after repeated exposure to noises intense enough to produce TTS, a permanent threshold shift (PTS) will occur. This is an irreversible increase in hearing thresholds and defines the second stage of chronic NIHL. At this point, there has been irreversible hair cell damage.<sup>5,14,18</sup>

In contrast to chronic NIHL, acoustic trauma refers to a sudden permanent hearing loss caused by a single exposure to an intense sound. It occurs when excessive sound energy strikes the inner ear. Exposure to noise from firearm use during military service is probably the most frequent etiology of acute acoustic trauma worldwide; therefore, it may be regarded as a professional disease in military populations.<sup>19</sup> The sound pressure levels capable of causing acoustic trauma vary between individuals but average around 130-140dB.<sup>14</sup> The hearing loss is sudden, sometimes painful, and is often followed by a new onset of tinnitus. For the vast majority of patients, tinnitus presents as the most annoying symptom, with the risk for permanent tinnitus being considered more critical for the patient than any degree of hearing loss resulting from acoustic trauma.<sup>19</sup> Although the audiogram may show the typical 3000-6000Hz sensorineural notch seen with chronic NIHL, down-sloping or flat audiograms that affect a broad range of frequencies are more common.<sup>2,14,20</sup> Direct mechanical injury to the sensory cells of the cochlea is thought to be the mechanism of injury in acoustic trauma.

Noise exposure and NIHL are the most common cause of tinnitus.<sup>2,5,6</sup> The relationship between noise exposure, NIHL and tinnitus has been addressed in a number of articles. A review of these studies was presented by Axelsson & Barrenas, 1991, and it was found that noise exposure and NIHL were by far the most common cause of tinnitus; if “acoustic trauma” was included, at least one-in-three cases were caused by noise.<sup>6</sup> Tinnitus may occur following a single exposure to high-intensity impulse/impact noise (a short burst of acoustic energy which can either be a single burst or multiple bursts of energy), long-term exposure to repetitive impulses, long-term exposure to continuous noise, or exposure to a combination of impulses and continuous noise.<sup>2,4,6</sup>

#### **THE “IMPACT” OF NOISE ON THE MILITARY**

A staggering number of Soldiers and Marines caught in roadside bombings and firefights in Iraq and Afghanistan are coming home with ringing in their ears. High rates of tinnitus among patients exposed to

gunfire and explosive detonations suggest that impulse/impact noise is likely to precipitate tinnitus associated with acoustic trauma, excessive noise exposure, and NIHL.<sup>2,4,6,10,12</sup> According to research published in the December 2005 issue of *American Journal of Audiology* (AJA), Soldiers sent to battle zones are over 50 times more likely to suffer NIHL loss and/or tinnitus than Soldiers who do not deploy.<sup>21</sup> According to a report released in 2007 by the House Appropriations Subcommittee on Military Quality of Life, as a result of ongoing combat operations, one in three post-deploying Soldiers report acute acoustic trauma and one in four report hearing loss and/or hearing complaints to include tinnitus.<sup>12</sup>

From World War II and well through the Vietnam War, hearing damage has been a leading disability. According to the Department of Veterans Affairs, hearing damage is the number one disability in the War on Terror, with some experts predicting the true toll could take decades to become clear.<sup>13</sup> According to the American Tinnitus Association (ATA), more will be spent on veterans’ disability compensation for tinnitus and other hearing conditions over the coming years than for any other medical injuries from the Iraq and Afghan wars.<sup>4</sup> Between 2000 and 2005, the number of veterans with tinnitus disabilities more than doubled and the amount paid to veterans with tinnitus disabilities went up more than two-and-a-half times.<sup>4,22</sup> Presently, tinnitus is the most prevalent disability among new cases added to Veterans Affairs numbers; nearly 70,000 of the more than 1.3 million troops who have served in Afghanistan and Iraq are collecting disability for tinnitus.<sup>13,23</sup> In fact, recent studies demonstrated that 49-50% of all Soldiers exposed to explosive blasts in Iraq and Afghanistan had tinnitus and 60% had tinnitus, often related to hearing loss.<sup>4,13,22,24</sup> The number of servicemembers on disability because of hearing damage is expected to grow 18% a year, with payments totaling \$1.1 billion annually by 2011.<sup>4,13</sup>

The economic consequences to the military for hearing impairment, to include tinnitus, include lost time and decreased productivity, loss of qualified workers through medical disqualification, military disability settlements, retraining, and expenses related to medical treatment such as hearing aids and audiometric testing.<sup>10</sup> While the economic consequences are significant, the military implications in a combat zone can be deadly.

A study published in the Army RD&A Bulletin in 1990, concluded that those with hearing impairments were 36% more likely to hear the wrong command, and 30% were less likely to correctly identify their target.<sup>4</sup> Additionally, it was noted that Soldiers with hearing impairments only hit the enemy target 41% of the time, while Soldiers without hearing impairments hit the enemy target 94% of the time. Those with hearing impairments were 8% more likely to take the wrong tar-



get shot and 21% more likely to have their entire tank crew killed by the enemy.<sup>4</sup>

Hearing damage has been a battlefield risk ever since the introduction of explosives and artillery, and the U.S. military recognized it in Iraq and Afghanistan and issued earplugs early on. But the sheer number of injuries and their nature, particularly the high incidence of tinnitus, came as a surprise to military specialists and outside experts. According to VA figures, despite all that has been learned over the years, U.S. troops are suffering hearing damage at about the same rate as World War II veterans.<sup>13,21</sup> Given today's unpredictable weaponry (i.e. roadside bombs), even the best hearing protection is only partly effective, and only if it's properly used.

It makes more sense to prevent hearing damage than to provide a lifetime of disability, but even hearing protection has its limits and it is important to note that some hearing impairments are unavoidable despite use of hearing protection and other measures. Some exposures are so extreme that they will exceed the protective capability of hearing protective devices. As previously noted, damage can occur at 85 decibels. The best protection cuts that by only 20-25dB.<sup>13</sup> That is not enough to protect the ears against an explosion or a firefight, which can range upwards of 180+ dB. Furthermore, much of the fighting consists of ambushes, bombings, and firefights, which come suddenly and unexpectedly, giving Soldiers little time to use their issued hearing protection. In addition, some Infantrymen resist or refuse to wear their hearing protection for fear of dulling their senses and missing critical commands or sounds that can make the difference between life and death.

### **HBO<sub>2</sub> THERAPY EFFICACY IN TINNITUS**

Medical treatments for tinnitus are well documented and there is probably no other disease for which such a variety of treatments have been proposed. Yet, still today, many different treatment regimens are being propagated. Vasodilators, vitamins, steroids, anticoagulants, heparin, histamine, tranquilizers, diuretics, prostacyclin, hypervolemic hemodilution, carbogen, and stellate ganglion block.<sup>3,25</sup> Whether applied separately or together, all have demonstrated limited effectiveness at best. Experimentally, rheological agents and plasma expanders neither cause an improvement in inner ear blood supply nor result in a higher oxygen supply in the inner ear.<sup>26</sup> In addition, two forms of tinnitus rehabilitation are currently being prescribed, tinnitus masking and psychological treatment; both offer symptomatic treatment, with the goal of treatment being only to lessen the awareness of tinnitus and its impact on quality of life.

Since the end of the 1960s, hyperbaric oxygen (HBO<sub>2</sub>) therapy has been used experimentally for certain acute and chronic illnesses of the inner ear.<sup>25</sup> The

role of HBO<sub>2</sub> in the treatment of tinnitus was investigated in the past: Pilgramm et al. in 1985, firstly, and Schumann et al. in 1990, secondly, reported about HBO<sub>2</sub> usefulness in tinnitus treatment, reporting an improvement of 62.2% in 557 patients' tinnitus after receiving 10 applications of HBO<sub>2</sub> therapy.<sup>25,27</sup> While skepticism remains high in the United States, physicians in Germany and Japan continue to recognize its clinical applications in diseases of the inner ear and have demonstrated improved outcomes in the treatment of acute acoustic trauma, NIHL, and tinnitus using HBO<sub>2</sub> therapy.<sup>7,26</sup> The rationale for this therapy is based on the oxygen transportation mechanism in human organisms.

The basis for hyperbaric oxygenation is the breathing of pure oxygen at a pressure which is increased compared to atmospheric pressure (1.0 ATA).<sup>9,26,28-30</sup> The effectiveness of high pressure oxygen therapy is based on raising the partial pressure of oxygen in the blood and thus the pressure difference to tissue. The concentration of oxygen in the atmosphere is 21%. At 1.0 ATA, the oxygen in blood is almost entirely carried by hemoglobin. Because hemoglobin is approximately 97% saturated under normal conditions, greatly increasing the oxygen-carrying capacity of blood by increasing hemoglobin saturation is not possible.

During hyperbaric oxygen therapy the patient sits inside a pressurized chamber. Air pressure inside the chamber is increased up to 2.5 times normal atmospheric pressure at sea level (2.5 ATA). The patient then breathes pure oxygen from a mask. Inhalation of hyperbaric oxygen can enhance the amount of oxygen carried in blood by increasing the quantity of oxygen dissolved in plasma. When breathing 100% oxygen at a surrounding pressure of 2.5 ATA, the quantity of dissolved oxygen in 100ml of plasma increases from 0.3ml, to 6.8ml, which is approximately 20 times higher than normal.<sup>9,26</sup>

The driving force for oxygen diffusion from the capillaries to tissue can be estimated by the difference between the partial pressure of oxygen on the arterial side and the venous side of the capillaries. The difference in the partial pressure of oxygen from the arterial side to the venous side of the capillary system is approximately 37 times greater when breathing 100% oxygen at 3.0 ATA than air at 1.0 ATA.<sup>29</sup>

The increased tissue oxygenation achieved during HBO<sub>2</sub> therapy can support poorly perfused and hypoxic areas. Under this increased pressure, the amount of dissolved oxygen is sufficient, even without hemoglobin, to supply body tissues with oxygen by diffusion. With an increase of the pressure of oxygen in the inner ear, it is possible to influence the auditory sensory cells (inner and outer hair cells) and the peripheral auditory nerve fibers.<sup>30</sup> These cells have no direct vascular supply and depend entirely on oxygen

supplied by diffusion. During exposure to HBO<sub>2</sub> therapy, the oxygenation in the cochlea increases by 460-600% and is still 60% above normal one hour after termination of the therapy.<sup>9,26,30</sup> An increase in oxygen pressure can compensate for oxygen deficiency caused by trauma and gives rise to biological mechanisms which can facilitate cellular and vascular repair.<sup>9,30</sup> Additionally, HBO<sub>2</sub> therapy has been shown to improve hemorheology by causing a reduction in hematocrit, a reduction of platelet aggregation, and an increase in the flexibility of erythrocytes.<sup>31</sup> Hyperoxia has also been shown to reduce edema by reducing vascular permeability and causing a rapid and significant vasoconstriction.<sup>29</sup>

HBO<sub>2</sub> is considered a relatively benign intervention with few adverse effects. Visual disturbance, usually reduction in visual acuity secondary to conformational changes in the lens, and barotrauma, affecting the middle ear, are the most frequently reported complications.<sup>15,16,29</sup> The majority of patients recover spontaneously over a period of days to weeks from their visual disturbances and most episodes of barotrauma do not require the therapy be abandoned. Barotrauma of the middle ear can be treated by placement of pressure equalization tubes or milder cases with decongestants and/or instruction regarding pressure equalization techniques. Less commonly, estimated only to occur in one in 5,000 to 11,000 treatments, HBO<sub>2</sub> may be associated with acute central nervous system oxygen toxicity.<sup>15,16,29</sup> Exposure to 100% oxygen at 3.0 ATA for three hours induces grand mal seizures in most people; at less than 3.0 ATA, seizures are rare.<sup>29</sup> Oxygen-induced seizures are typically benign and produce no long-term sequelae. Additional complications include barotraumas affecting the dental cavities and sinuses, pulmonary barotraumas, drug reactions, and injuries or death related to chamber fires. Decompression sickness can also occur, though rare in patients breathing 100% oxygen with short air breaks.

#### CURRENT LITERATURE

The evidence for HBO<sub>2</sub> therapy for acute and chronic tinnitus based on randomized controlled trials is poor. In July 2004, Bennett et al. underwent an extensive search of what they considered to be "suitable" randomized human trials assessing the outcome of tinnitus with HBO<sub>2</sub> therapy. The inclusion criteria consisted of a randomized controlled study, a review with new data, was not a comparative trial in which all subjects/groups received HBO<sub>2</sub> therapy, subjects were randomly allocated, and report was not a case study.<sup>15,16</sup> The initial search identified six randomized human trials meeting the criteria. However, after appraisal of the full report, three articles were excluded because they did not contain new data. A follow-on search was conducted by Bennett et al. in 2006; no additional studies were identified.<sup>16</sup> Using the same inclusion criteria, the

author of this paper was unable to identify any additional studies that met all the criteria, but was able to find a number of prospective and retrospective studies evaluating the benefits of HBO<sub>2</sub> therapy for the treatment of tinnitus.

#### PROSPECTIVE STUDIES

Two of the randomized controlled trials, identified by Bennett et al., reported on improvements in tinnitus for patients with an early/acute presentation.<sup>15,16,26</sup> The Hoffmann et al. 1995a trial contributed 20 subjects with idiopathic sudden sensorineural hearing loss (ISSHL) with or without tinnitus; all subjects had no improvement after 14 days of pharmacological treatment with hydroxyethyl starch, pentoxifylline, and cortisone. The Schwab et al. 1998 trial contributed 33 subjects with sudden hearing loss and tinnitus seen within two weeks of onset of tinnitus and without any prior therapy. In each study the HBO<sub>2</sub> group's therapy consisted of 100% oxygen at 1.5 ATA for 45 minutes daily, five days each week for two to four weeks (10 to 20 sessions). The control groups underwent no treatment. While the two trials reported a greater mean improvement in tinnitus (using a visual analogue scale between 0 and 10) in the HBO<sub>2</sub> arm compared to the control arm, statistical pooling was not possible due to the authors neglecting to report the standard deviation around the means. As a consequence, clinical significance could not be determined.

The third article considered suitable, by Hoffmann et al. 1995b, was the only randomized human controlled trial reporting on improvements in tinnitus for patients with a chronic presentation.<sup>15,16</sup> This study contributed 44 subjects with ISSHL and tinnitus for longer than six months. HBO<sub>2</sub> therapy consisted of 100% oxygen at 1.5 ATA for 45 minutes daily, five days each week for three weeks. The control group breathed air at 1.5 ATA on the same schedule as the HBO<sub>2</sub> group. While the HBO<sub>2</sub> therapy group did demonstrate some improvement in tinnitus, the improvement did not reach statistical significance:  $p=0.12$ .<sup>15,16</sup>

In each of these studies the HBO<sub>2</sub> therapy consisted of breathing 100% oxygen at 1.5 ATA for 45 minutes. In studies reporting significant improvements, HBO therapy consisted of breathing 100% oxygen at 2.0 to 2.5 ATA for 90 minutes.

In 2007, a comparative trial by Porubsky et al. evaluating the influence of time interval from the onset of tinnitus until the first HBO<sub>2</sub> therapy was published.<sup>31</sup> In addition to time interval, the study compared the influence of other factors: treatment protocols, gender, noise characteristic, and pretreatment expectations. This author will only comment on treatment protocols and time interval from tinnitus onset to treatment.

In this study, 360 patients suffering from tinnitus were randomized into two HBO<sub>2</sub> treatment pro-

tocols: group A: 2.2 ATA for 60 minutes and group B: 2.5 ATA for 60 minutes. Both series were administered once a day for 15 consecutive days; 156 patients underwent protocol A and 156 protocol B. Forty-eight patients were treated inconsistently, leaving out single days of treatment. No patient had less than twelve HBO<sub>2</sub> sessions. One month after the end of HBO<sub>2</sub> treatment, the therapeutic effect was evaluated according to the patient's subjective assessment of tinnitus. A non-treatment control group was not identified. In 92 patients HBO<sub>2</sub> therapy was started within the first two weeks after the onset of tinnitus; in 93 there was a delay between two weeks and six months; in 41 cases the delay was 6-12 months; and in 126 patients more than one year elapsed between the onset of tinnitus and HBO<sub>2</sub> treatment. Eight patients did not answer the question.

A complete remission of tinnitus was reported by 12 (3.3%) subjects, 122 (33.9%) felt a decrease in intensity, 157 (56.3%) patients did not notice any changes and 25 (6.9%) patients complained that their tinnitus became louder after HBO<sub>2</sub>.<sup>31</sup> Out of the 12 patients who had a complete remission of tinnitus, 10 (83.3%) had HBO<sub>2</sub> within the first two weeks after the onset of tinnitus and two (16.6%) later than two weeks but within the first six months. Out of the 122 patients who felt that their tinnitus had lessened, 37 (30.3%) had HBO<sub>2</sub> therapy within the first two weeks after the onset and 39 (31.9%) were treated within the first six months. Only nine (7.4%) who started HBO<sub>2</sub> six to twelve months after the onset of tinnitus had improvement and thirty-four (27.9%) felt a lessening of tinnitus after more than twelve months delay until HBO<sub>2</sub>.<sup>31</sup>

The authors determined there was no statistically significant difference between treatment groups A and B ( $p > .05$ ). Furthermore, they concluded there is no statistically significant difference between the time intervals until the start of HBO<sub>2</sub> therapy.<sup>31</sup>

This study compared a treatment protocol of 2.2 ATA for 60 minutes to a treatment protocol of 2.5 ATA for 60 minutes. This study could have been enhanced if it would have compared two groups in which there was a bigger difference between treatment protocols (i.e. one group breathing 100% oxygen at less than 2.0 ATA) and/or included a non-HBO<sub>2</sub> therapy control group. Additionally, the study grouped patients into a treatment group receiving therapy within the first two weeks after onset of tinnitus and one in which the patient received therapy later than two weeks but within the first six months. Again the authors reported no statistical significant difference between the time intervals until the start of HBO<sub>2</sub> therapy. Most studies group subjects into those suffering from tinnitus for three months or less and those suffering from tinnitus for greater than three months, but less than six months. Of the 122 patients treated, 39 (31.9%) who were treated within the first six months, but after two weeks

from the onset of their tinnitus, reported improvement. Of the 39, how many were treated within three months from the onset of their tinnitus, and if added to those who demonstrated improvement if treated within two weeks from the onset of their tinnitus, would a significant difference between time intervals be seen?

In a study published in 2003 by Narozny et al., 61 patients with tinnitus (29 acute, 32 chronic) underwent HBO<sub>2</sub> therapy with simultaneous pharmacotherapy (group A).<sup>25</sup> HBO<sub>2</sub> therapy was administered once daily at a pressure of 2.5 ATA for 90 minutes (three periods of 20 minutes with two five-minute air breaks and 20 minutes needed for compression and decompression). The patients breathed 100% oxygen throughout the treatment with exception of the two five-minute air breaks. Patients with acute tinnitus underwent  $15 \pm 6$  HBO<sub>2</sub> exposures, patients with chronic tinnitus  $18 \pm 6$  exposures. Before, immediately, and six months after the end of treatment, the level of tinnitus was assessed by means of a visual analog scale (VAS), Vernon's tinnitus severity scores (VTSS), and questionnaire by Tyler and Baker. The obtained results were compared with 122 patients (group B) with tinnitus (70 acute and 52 chronic) treated only pharmacologically. Tinnitus improvement after therapy was stated by comparison of tinnitus level before and after therapy (in percentage).

Satisfactory improvement of tinnitus loudness (more than 50% in comparison to primary state), using the VAS, was demonstrated in 58.6% of patients with acute tinnitus in group A. Of the 58.6% who demonstrated satisfactory improvement, 41.4% showed excellent improvement (75% to 100%) and 17.2% showed some improvement (50% to 75%). No improvement (less than 50%) was seen in 41.4% of the acute tinnitus patients in group A. Comparative analysis of group B subjects with acute tinnitus reflected 41.4% with satisfactory improvement, 30.0% with excellent improvement, and 11.4% with some improvement. No improvement was noted in 58.6% of the acute tinnitus patients in group B. Satisfactory tinnitus improvement in patients with chronic tinnitus (group A) was 81.3%, 6.3% with excellent improvement, and 75.0% with some improvement. No improvement was noted in 18.7%. Comparative analysis of group B subjects with chronic tinnitus revealed 65.4% with satisfactory improvement, 25.0% with excellent improvement, and 40.4% with some improvement. No improvement was noted in 34.6% of the chronic tinnitus patients in group B.<sup>25</sup> Similar results were obtained by VTSS and questionnaire. After six months, there was an inconsiderable regression of the positive effect of therapy, especially in patients with chronic tinnitus, in group A as well as in group B.

The authors (Narozny et al.) concluded HBO<sub>2</sub> therapy may contribute to the treatment of tinnitus, particularly its chronic severe form. Their results were



similar to those of other authors, indicating that HBO<sub>2</sub> therapy can reduce tinnitus even if it has been present for a long time.<sup>25-27</sup>

The authors reported the wrong data for the acute tinnitus group B patients in their results section and unfortunately based their conclusions using the incorrect data. Using the correct data (shown in Table 2 of this study) HBO<sub>2</sub> therapy is shown to be more beneficial in the acute tinnitus stage (group A compared to group B) than it is in the chronic stage (group A compared to group B). While there is a 17.2% difference in satisfactory improvement in acute tinnitus patients comparing group A to group B, there is only a 15.9% difference in satisfactory improvement in chronic tinnitus patients comparing group A to group B.

In a prospective controlled study conducted by Biesinger et al. (1998), 211 cases of acute tinnitus (tinnitus for less than three months) were assessed after receiving one of three treatment protocols.<sup>32</sup> Of the 211 cases of acute tinnitus, 69 patients were treated with haemodilution and cortisone alone and had no HBO<sub>2</sub> therapy. Of the 142 patients that had HBO<sub>2</sub> therapy, 72 of these were after unsuccessful haemodilution.

Of the 69 cases in which the patients received haemodilution only, 36.2% healed completely, 38.8% cases did not notice a change, and 25% of the patients reported a decompensation. Of the 142 cases receiving HBO<sub>2</sub> therapy, 64.1% healed completely, 17.9% experienced no change, and 18% reported a decompensation. Out of the 72 cases receiving HBO<sub>2</sub> therapy after failure of haemodilution, 51.4% healed completely, whereas 37.5% reported improvement, 11.1% experienced no change in their tinnitus, and 0% of the cases worsened.<sup>7,32</sup> The results demonstrated a better outcome for patients with acute tinnitus if they received HBO<sub>2</sub> therapy, especially the high rate of decompensated tinnitus in patients receiving solely haemodilution.

In a study published in 1997 by Delb et al.,<sup>33</sup> a total of 193 patients, having undergone and failed primary intravenous hemorrheologic therapy, were treated with HBO<sub>2</sub> therapy. Tinnitus was evaluated before, after ten sessions, and after 15 sessions using a tinnitus questionnaire. Measurable improvements of the tinnitus occurred in 22% of the patients, moderate improvement in 17% of cases, excellent improvement in 10.4% of cases and complete resolution in two patients.<sup>33</sup> Though clinical significance was not reported, the improvement rate decreased in those cases where the time from onset of tinnitus exceeded 40 days. In addition, while the improvement rate slightly increased in patients receiving 15 sessions compared to those receiving 10 sessions, the clinical significance, once again, was not reported. The authors concluded that HBO<sub>2</sub> therapy seems to be a moderately effective additional treatment in the therapy of tinnitus after primary hemorrheologic therapy, provided the time from onset of tinnitus is less than one month.<sup>33</sup>

In another study published in 1997 by Kau et al.,<sup>26</sup> 355 patients with tinnitus, who had not responded to treatment with medications, were given HBO<sub>2</sub> therapy. Of the 355 patients, 192 suffered from tinnitus for less than three months and 163 suffered from tinnitus for more than three months. HBO<sub>2</sub> therapy consisted of a pressure increase phase of 20 minutes, at the end of which a diving depth of 2.5 ATA was reached. This pressure was held for 70 minutes which was then followed by an ascent phase lasting 20 minutes. Pure oxygen was inhaled by mask during the entire treatment period. The number of treatments was not reported and a non-HBO<sub>2</sub> therapy control group was not identified. Subjective evaluation of tinnitus was expressed by means of a visual analog scale.

For the patients in whom the first episode of tinnitus was within three months before HBO<sub>2</sub> therapy, excellent improvement was seen in 6.7%, noticeable improvement in 44.3%, unchanged in 44.3%, and a temporary increase in the severity of tinnitus in 4.7%.<sup>26,30</sup> Patients who had tinnitus for more than three months before HBO<sub>2</sub> therapy showed a less favorable response. In none of the patients did the tinnitus fully resolve. Noticeable improvement was reported by 34.4% of the patients, no change in tinnitus was appreciated by 62% of the patients and an intermittent increase was reported by 3.6% of the patients.<sup>26,30</sup>

The authors feel the results justify the position that patients, who have been treated unsuccessfully by "conventional" means, may still have a chance of improvement in their symptoms when they can be given HBO<sub>2</sub> therapy within three months of the onset of their tinnitus.<sup>26</sup>

In 1997, an article by Bohmer was published reporting on two prospective studies conducted at the Institute for Hyperbaric Medicine, Orthopaedic University Clinic, Frankfurt, Germany.<sup>28</sup> In the first study, 47 patients received HBO<sub>2</sub> therapy within three months of tinnitus first occurring. In each case they received pharmacotherapy often combined with cortisone prior to undergoing HBO<sub>2</sub> therapy. In 64% of the cases an improvement was attained. During the follow-up examinations 27% of the patients confirmed a further decrease of the ringing in their ears during the two months following treatment.

In the second study, 381 patients underwent HBO<sub>2</sub> therapy for the treatment of their tinnitus. On average 15 single treatments for 90 minutes with a pressure of 2.2 to 2.5 ATA were carried out. Daily, at the same time each day, the patients were asked to subjectively annotate their sound volume. Complete resolution of tinnitus was seen in 3.9% of the patients. Noticeable improvement was seen in 34.1%, slight improvement in 31.8%, no improvement in 28.1%, and worsening of tinnitus in 2.1% of the patients.<sup>28</sup> With HBO<sub>2</sub> therapy, the improvement of tinnitus sound from "becoming less" to "being completely healed" was ap-

preciated in the first six months of tinnitus first occurring. The major advances starting with “unbearably loud” to “bearable” were made during the first two to three months.

The author recommends that HBO<sub>2</sub> therapy should be liberally applied when infusion therapy shows no success. Even after four to six months successful results were obtained with tinnitus patients.<sup>28</sup>

### RETROSPECTIVE STUDIES

In 1998, Lamm et al., and in 2003, Lamm reported on a retrospective meta-analysis of 50 clinical studies carried out on a total of 4,109 patients who received HBO<sub>2</sub> therapy following unsuccessful conventional treatment with drugs for patients suffering from tinnitus.<sup>30,34</sup> Providing the onset of the disorder was longer than two weeks but not longer than six weeks, 4% of the patients suffering from tinnitus reported complete resolution, 81.3% observed a decrease in tinnitus intensity, 13.5% reported no change and 1.2% reported a temporary increase in tinnitus.<sup>30,34</sup> These results were confirmed in some of the prospective studies described above as well as additional studies by Nakiashima et al. (1998), Shiraishi et al. (1998) and Murakawa et al. (2000).<sup>26,33</sup> The authors concluded that HBO<sub>2</sub> therapy is recommended and warranted in those patients treated within three months of the onset of symptoms.<sup>30,34</sup>

In a retrospective evaluation of 7766 patients in 13 publications showed reduction of the molestation and intensity of tinnitus by 50% in approximately 70% of the cases (30%-88%) if treated within the first three months.<sup>7,9,35</sup> Chronic tinnitus with duration of more than three months or bilateral manifestation showed improvement rates of 50% in around 30% of the cases after ineffective conservative treatment. Follow-ups showed no change in 12 months.

An additional retrospective study published by Hoffmann et al.<sup>28</sup> 250 patients who had been treated unsuccessfully with infusion therapy received HBO<sub>2</sub> therapy. These subjects were compared to patients who did not receive HBO<sub>2</sub> therapy. The subjects were under observation for 21 months. In this study, 60% of patients undergoing HBO<sub>2</sub> therapy ascertained a steady tinnitus improvement. Other HBO<sub>2</sub> therapy centers have also shown good results; Almeling et al. (1996), Dauman et al. (1985), Meazza et al. (1996), and Takahashi et al. (1989).

### DISCUSSION

Tinnitus is the phantom perception of sound in the absence of overt acoustic stimulation.<sup>36</sup> Its impact on the military population is alarming. Annually, tinnitus is the most prevalent disability among new cases added to Veterans' Affairs rolls and is currently the number one disability in the War on Terror. There is more being spent on veterans' disability compensation

for tinnitus than on any other disability, with payments expecting to reach \$1.1 billion annually by 2011.<sup>4,13</sup>

A considerable number of therapies have been proposed since tinnitus first appeared in medical literature. However, the results of established, conservative medical treatment regimes for tinnitus are unsatisfying. It has been shown that common pharmacological treatment does not yield better results than placebo therapy.<sup>30,31,36</sup> The knowledge of hyperbaric oxygen therapy for the hyperoxygenation of tissue has led to further development of medical indications over the past 50 years. Indications for ENT therapy include decompression trauma of the inner ear, idiopathic sudden hearing loss, acute acoustic trauma, acute noise-induced hearing loss, osteoradionecrosis and osteomyelitis, otogenic infection of the skull base, and otitis externa maligna.<sup>30</sup> HBO<sub>2</sub> treatment increases the inner ear pO<sub>2</sub>; decreases hematocrit, plasma viscosity, and platelet aggregation, and improves microcirculation.<sup>29,30,37</sup> In spite of its clear-cut rationale, an effectiveness of HBO<sub>2</sub> therapy has not been objectively documented for tinnitus and its use in the United States has not been widely applied (this is not approved by the Undersea and Hyperbaric Medicine Society). Due to the low number of recognized, controlled, double-blind clinical trials demonstrating the effectiveness of HBO<sub>2</sub> therapy for tinnitus, this therapy lacks official recognition and skepticism remains high. Poor methodological quality in many of the reported trials, variability and poor reporting of entry criteria, the inconsistent nature and timing of outcomes, and poor reporting of both outcomes and methodology make comparisons and meta-analysis impossible. In addition, treatment protocols and patient inclusion criteria are not standard, and poorly reported in some trials. No standard severity scale is employed across these trials, and the time to entry varies from within hours to years. Many of the patients were negatively selected, they had already been treated by various methods and only those who had not responded to these therapies were treated with HBO<sub>2</sub> therapy. Moreover, many of the studies neglected to identify a control group and many did not assess HBO<sub>2</sub> as a monotherapy.

### CONCLUSION

Many of the reports indicate the effectiveness of HBO<sub>2</sub> therapy for tinnitus, but a majority of them are retrospective and many suggest using HBO<sub>2</sub> therapy as an adjuvant to standard medical treatment. Nonetheless, the results justify the position that patients with tinnitus, who have been treated conventionally, may still have a chance of improvement of their condition when they can be given HBO<sub>2</sub> therapy within three to six months. These studies have shown that hyperbaric oxygenation treatment can suppress acute and even longer existing tinnitus. It appears that during the first six months, HBO<sub>2</sub> therapy has a positive and



promising effect on tinnitus. However, the most significant improvement in tinnitus is notable when HBO<sub>2</sub> therapy is administered within the first three months at pressures between 2.0 and 2.5 ATA.

#### FUTURE RESEARCH

Because of its subjective nature, assessing the level of distress remains the primary impediment in the appraisal of tinnitus studies. In patient studies, differences in the level of tinnitus, duration, medical history, and involvement of etiological factors in the initiation and mental habituation may obscure any correlation with a treatment outcome.<sup>38</sup> There is a case for large randomized trials of high methodological rigor in order to define the true extent of the benefit (if any) from administration of HBO<sub>2</sub> therapy to patients suffering from tinnitus. A critical multicenter analysis with identical documentation of a large number of patients should establish the therapeutic value of HBO<sub>2</sub> therapy for well defined groups of patients. In addition, further studies to evaluate the actual effect of HBO<sub>2</sub> therapy should concentrate on the development of double-blind, case controlled trials.

Though the authors of several studies report various degrees of improvement in up to 50% to 70% of patients undergoing HBO<sub>2</sub> therapy, actual cure of tinnitus is rare. In no study was it reported to be greater than 3.9%. If HBO<sub>2</sub> therapy is scientifically established to be beneficial in the treatment of tinnitus, cost analysis for treating tinnitus versus paying out VA benefits should be conducted. Will curing approximately 4% of cases significantly reduce VA compensation for tinnitus and/or will a significant reduction in a patient's tinnitus affect VA compensation? A final evidence based recommendation will be possible after conclusion of several randomized, controlled, double-blind studies. Currently, there are six major prospective trials being carried out in Germany.<sup>7,9</sup>

#### ACKNOWLEDGEMENTS

The opinions or assertions contained herein are the private views of the author and are not to be constructed as official or reflecting the views of the Department of the Navy, Department of Defense, nor the U.S. Government.

#### REFERENCES

1. Medical College of Wisconsin (2001). Noise-induced hearing loss. Retrieved March 16, 2009, from Healthlink Medical College of Wisconsin Website: <http://healthlink.mcw.edu/article/965928293.html>
2. Humes, LE, Joellenbeck, LM, Durch, JS (2006). Noise and military service: Implications for hearing loss and tinnitus. Washington D.C.: The National Academies Press.
3. Deschler, DG, (2008). Pathogenesis, diagnosis and treatment of tinnitus. Retrieved December 2008, from Up-to-date Website: <http://utdol.com/online/tinnitus/treatment.html>
4. Lite, J. (2007). Iraq & Afghanistan war vets suffer from hearing loss, tinnitus. The New York Daily News, November 11.
5. American Hearing Research Foundation (2008). Noise-induced hearing loss. Retrieved March 16, 2009, from American Hearing Research Foundation Website: [http://www.americanhearing.org/disorders/hearing/noise\\_induced.html](http://www.americanhearing.org/disorders/hearing/noise_induced.html)
6. Axelsson, A; Prasher, D. (2000). Tinnitus induced by occupational and leisure noise. *Noise & Health: A Quarterly Inter-disciplinary International Journal*; 2(8): 47-54.
7. Reimer Hyperbaric of Canada (1998). Hyperbaric oxygen in the treatment of sudden deafness, acute tinnitus and acute acoustic trauma. Retrieved March, 5 2009, from Reimer Hyperbaric of Canada Website: <http://www.reimerhbo.com/tinnitus.html>
8. Oeken, Jens. (1998). Distortion product otoacoustic emissions in acute acoustic trauma. *Noise & Health: A Quarterly Inter-disciplinary International Journal*; 2(1): 56-66.
9. Keate, B. (2008). Hyperbaric oxygen therapy for tinnitus. Retrieved March 16, 2009, from Arches Tinnitus Formulas Website: <http://www.tinnitusformula.com/infocenter/articles/treatments/hyperbaric.aspx>
10. Naval Safety Center (2008). Acquisition safety – noise control aboard ships. Retrieved December 2, 2008, from Navy Safety Center Website: <http://www.safetycenter.navy.mil/acquisition/noise/index.aspx>
11. National Institute of Health (2008). Noise-induced hearing loss Retrieved March 16, 2009, from National Institute on Deafness and Other Communication Disorders Website: <http://www.nidcd.nih.gov/health/hearing/noise.aspx>
12. Whittle, R. (2006). Hearing loss on rise among troops: Army plans to reduce military audiologist, add civilians. The Dallas Morning News, March 5, p.A10.
13. MSNBC (2008). Hearing loss is silent epidemic in U.S. Troops: Soldiers coming home with permanent hearing damage and ringing in ears. Retrieved December 2, 2008, from MSNBC Website: <http://www.msnbc.msn.com/id/23523729/>.
14. Rosen, E.J.; Vrabec, J.T.; Quinn, F.B.. (2001). Noise-induced hearing loss. Grand Rounds Presentation, UTMB, Dept. of Otolaryngology; January 2001.
15. Bennett, MH; Kertesz, T; Yeung, P. (2005). Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss and tinnitus: A systematic review of randomized controlled trials. *The Journal of Laryngology and Otolaryngology*; 119(10): 791-798.
16. Bennett, MH; Kertesz, T; Yeung, P. (2007). Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss and tinnitus. *Cochrane Database of Systematic Reviews*; Issue 1. Art.
17. Rabinowitz, PM. (2000). Noise-induced hearing loss. Retrieved March 16, 2009, from *The American Family Physician* Website: <http://www.aafp.org/afp/20000501/2749.html>
18. Bredenkamp, JK; Schoenfield. (2008). Noise-induced hearing loss and its prevention. Retrieved March 16, 2009, from MedicineNet Website: [http://www.medicinenet.com/noise\\_induced\\_hearing\\_loss\\_and\\_its\\_prevention/article.html](http://www.medicinenet.com/noise_induced_hearing_loss_and_its_prevention/article.html)
19. Daniilidis, IC. (2004). Evaluation of various therapeutic schemes in the treatment of tinnitus due to acute acoustic trauma. *Kulak Burun Bogaz Ihtis Derg*; 12(5-6): 107-114.

20. Zalin, H. (1971). Noise-induced hearing loss: Session I: Differential Diagnosis. *Section of Occupational Medicine with Section of Otolaryngology: Joint Meeting No.2*; 64: 187-190.
21. The ASHA Leader. Hearing loss rises among U.S. Soldiers in Iraq. *The ASHA Leader* 2006; 11(4):5, 19.
22. Hearing Review, The Insider (2006). Military Vets suffer dramatic increase in tinnitus and other hearing damage. Retrieved December 2, 2008, from Hearing Review Website: [http://www.hearingreview.com/insider/2006-10-12\\_1861.aspx](http://www.hearingreview.com/insider/2006-10-12_1861.aspx)
23. U.S. Army Center for Health Promotion and Preventative Medicine (2003). Just the facts...Tinnitus and noise-induced hearing loss. Retrieved March 16, 2009, from The USACH PPM Website:<http://chppm-www.apgea.army.mil/documents/FACT/51-003-1103.pdf>
24. Ritenour, AE; Wickley, A; Ritenour, JS; Kriete, BR; Brian, R; Blackbourne, LH; Holcomb, JB; Wade. (2008). Tympanic Membrane perforation and hearing loss from blast overpressure in Operation Enduring Freedom and Operation Iraqi Freedom wounded. *The Journal of Trauma Injury, Infection and Critical Care*; 64(2): S174-S178.
25. Narozny, W; Sicko, Z; Kuczkowski, J; Stankiewicz, C; Przewozny, T. (2003). Usefulness of hyperbaric oxygen therapy in patients with sensorineural acute and chronic tinnitus. *International Congress Series*; 1240: 277-286.
26. Kau, RJ; Sendther-Gress, K; Ganzer, U; Arnold, W. (1997). Effectiveness of hyperbaric oxygen therapy in patients with acute and chronic cochlear disorders. *ORL*; 59: 79-83.
27. Schumann, K; Lamm, K; Hettich, M. (1990). Effect and effectiveness of hyperbaric oxygen therapy in chronic hearing disorders. *HNO*; 38(11): 408-411.
28. Bohmer, D. (1997). Treating tinnitus with hyperbaric oxygenation. *International Tinnitus Journal*; 3(2): 137-140.
29. Mathur, NN; Prince, M. (2006). Hyperbaric oxygen. Retrieved March 5, 2009 from eMedicine Website: <http://emedicine.medscape.com/article/853951-overview>.
30. Lamm, K; Lamm, H; Arnold, W. (1998). Effect of hyperbaric oxygen therapy in comparison to conventional or placebo therapy or not treatment in idiopathic sudden hearing loss, acoustic trauma, noise-induced hearing loss and tinnitus: A literature Survey. *Adv Otorhinolaryngology*; 54: 86-99.
31. Porubsky, C; Stiegler, P; Matzi, V; Lipp, C; Kontaxis, A; Klemen, H; Walch, C; Smolle-Juttner, F. (2007). Hyperbaric oxygen in tinnitus: Influence of psychological factors on treatment results? *ORL*; 69: 107-112.
32. Biesinger, E; Heiden, C; Greimel, V; Lendle, T; Hoing, R; Albecker, K. (1998). Strategien in der ambulanten Behandlung des tinnitus. *ORL*; 46: 157-169.
33. Delb, W; Muth, CM; Hoppe, U; Iro, H. (1999). Outcome of hyperbaric oxygen therapy in therapy refractory tinnitus. *HNO*; 47(12): 1038-1045.
34. Lamm, K. (2003). Hyperbaric oxygen therapy for the treatment of acute cochlear disorders and tinnitus. *ORL*; 65: 315-316.
35. Lamm, H. Der einfluss der hyperbaren sauerstofftherapie auf den tinnitus und den horverlust bei akuten und chronischen in nenohrschaden.
36. Stiegler, P; Matzi, V; Lipp, C; Kontaxis, A; Klemen, H; Walch, C; Smolle-Juttner, F. (2006). Hyperbaric oxygen (HBO<sub>2</sub>) in tinnitus: Influence of psychological factors on treatment results? *UHM*; 33(6): 429-437.
37. Dundar, K; Gumus, T; Ay, H; Yetiser, S; Ertugrul, E. (2007). Effectiveness of hyperbaric oxygen on sudden sensorineural hearing loss: Prospective clinical research. *The Journal of Otolaryngology*; 36(1): 32-37.
38. Tan, J; Tange, RA; Dreschler, WA; Kleij, A; Tromp, EC. (1999). Long-term effect of hyperbaric oxygenation treatment on chronic distressing tinnitus. *Scand Audiology*; 28: 91-96.

## ADDITIONAL REFERENCES

Hoffmann, G; Bohmer, D; Desloovere, C. (1995). Hyperbaric oxygenation as a treatment of chronic forms of inner ear hearing loss and tinnitus. In: Li W-R Cramer F. eds. Proceedings of the Eleventh International Congress on Hyperbaric Medicine. Best Publishing; 141-145.

Hoffmann, G; Bohmer, D; Desloovere, C. (1995). Hyperbaric oxygenation as a treatment for sudden deafness and acute tinnitus. In: Li W-R. Cramer F. eds. Proceedings of the Eleventh International Congress on Hyperbaric Medicine. Best Publishing; 146-51.

Schwab, B; Flunkert, C; Heermann, R; Lenarz, T. (1998). HBO in the therapy of cochlear dysfunctions – First results of a randomized study. In: Gennser M. ed EUBS Diving and Hyperbaric Medicine: Controlled manuscripts of XXIV Annual Scientific Meeting of the European Underwater and Baromedical Society. Stockholm: EUBS; 40-42.

Pilgramm, M; Lamm, H; Schumann, K. (1985). Hyperbaric oxygen therapy in sudden deafness. *Laryngologie, Rhinologie, Otologie*; 64(7): 351-354.

Schumann, K; Lamm, K. Hetlich, (1990). Zur Wirksamkeit der hyperbaren Sauerstofftherapie bei alten Horstorungen. Bericht uber 557 Fale aus dem Jahre 1989, HNO; 38: 408-411.



LCDR Thomas Baldwin is the Senior Medical Officer at Special Boat Team Twenty-Two, Stennis Space Center, Mississippi. He is a graduate of the University of Texas Health Science Center at San Antonio Medical School and also holds a Master's Degree in Physical Therapy from the U.S. Army-Baylor University program at Fort Sam Houston.