View metadata, citation and similar papers at core.ac.uk
brought to you by CORE
provided by Helsingin yliopiston digitaalinen arkisto

Department of Otorhinolaryngology – Head and Neck Surgery
University of Helsinki
Helsinki, Finland

INVESTIGATIONS OF NOISE-RELATED TINNITUS

Roderik Mrena

Academic dissertation

To be presented for public examination, with the permission of
the Medical Faculty of the University of Helsinki,
in the Lecture Room of the Department of Otorhinolaryngology,
Haartmaninkatu 4E, Helsinki, on October 7th, 2011, at 12 noon.

Supervised by
Professor Antti Mäkitie
Department of Otorhinolaryngology – Head and Neck Surgery
Helsinki University Central Hospital and University of Helsinki
Helsinki, Finland

Professor Emeritus Jukka Ylikoski
Department of Otorhinolaryngology – Head and Neck Surgery
University of Helsinki
Helsinki, Finland

Reviewed by
Professor Claes Möller
Audiological Research Centre
Örebro University
Örebro, Sweden

Docent Kyösti Laitakari
Department of Otorhinolaryngology – Head and Neck Surgery
Oulu University Hospital and University of Oulu
Oulu, Finland

Discussed with
Professor Göran Laurell
Department of Clinical Sciences
Umeå University
Umeå, Sweden

ISBN 978-952-92-9236-3 (Paperback)
ISBN 978-952-92-9237-0 (PDF)

© Roderik Mrena

Kopijyvä Oy
Jyväskylä, Finland
2011

To my family

CONTENTS

1 List of original publications

This thesis is based on the following original publications, which are referred to in the text by their Roman numerals:

I Mrena R., Savolainen S., Pirvola U. & Ylikoski J. Characteristics of acute acoustical trauma in the Finnish Defence Forces. *Int J Audiol. 2004 Mar;43(3):177-181.*

II Mrena R., Pääkkönen R., Bäck L., Pirvola U. & Ylikoski J. Otologic consequences of blast exposure: a Finnish case study of a shopping mall bomb explosion. *Acta Otolaryngol. 2004 Oct;124(8):946-952.*

III Mrena R., Savolainen S., Kuokkanen J.T. & Ylikoski J. Characteristics of tinnitus induced by acute acoustic trauma: a long-term follow-up. *Audiol Neurootol. 2002 Mar-Apr;7(2):122-130.*

IV Mrena R., Ylikoski M., Mäkitie A., Pirvola U. & Ylikoski J. Occupational noise-induced hearing loss reports and tinnitus in Finland. *Acta Otolaryngol. 2007 Jul;127(7):729-735.*

V Mrena R., Ylikoski J., Kiukaanniemi H., Mäkitie A.A. & Savolainen S. The effect of improved hearing protection regulations in the prevention of military noise-induced hearing loss. *Acta Otolaryngol 2008;128(9):997-1003.*

VI Mrena R., Savolainen S., Kiukaanniemi H., Ylikoski J & Mäkitie A.A. The effect of tightened hearing protection regulations on military noise-induced tinnitus. *Int J Audiol 2009;48(6):394-400.*

Reprinted here with the permission of the publishers.

2 Abbreviations

AAT	Acute acoustic trauma
CMH	Central Military Hospital
dB	Decibel
ENT	Ear, nose and throat
EU	European Union
FDF	Finnish Defence Forces
HC	Hair cell
IHC	Inner hair cell
ISCO-88	International Standard Classification of Occupations (1988)
kHz	Kilohertz
kPa	Kilopascal
NCO	Non-commissioned officer
NIHL	Noise-induced hearing loss
OHC	Outer hair cell
SPL	Sound pressure level
THQ	Tinnitus Handicap Questionnaire
TM	Tympanic membrane
TRT	Tinnitus Retraining Therapy
VAS	Visual analogue scale

3 Abstract

Tinnitus is a frequent consequence of noise trauma. Usually, however, the main focus regarding the consequences of noise trauma is placed on hearing loss, instead of tinnitus.

The objectives of the present study were to assess various aspects of noise-related tinnitus in Finland, such as to determine the main causes of conscript acute acoustic traumas (AAT) in the military, assess tinnitus prevalence after noise trauma, characterize long-term AAT-related tinnitus prevalence and characteristics, assess occupational tinnitus, and evaluate the efficacy of hearing protection regulations in preventing hearing loss and tinnitus. The study comprised several independent noise-exposed groups: conscripts performing their military duty, former conscripts who suffered an AAT over a decade earlier, bomb explosion victims, and retired army personnel. Tinnitus questionnaires were used to assess tinnitus prevalence and characteristics. For occupational tinnitus, occupational noise-induced hearing loss (NIHL) reports to the Finnish Institute of Occupational Health were reviewed.

Tinnitus is a common result of AAT, blast exposure and long-term noise exposure. Despite hearing protection regulations, up to hundreds of AATs occur annually among conscripts in the Finnish Defence Forces (FDF). The most common cause is an accidental shot, accounting for approximately half of the cases. Conscript AATs are mainly due to accidental shots, while the ear is unprotected. Only seldom is an AAT due to negligence. The most common causative weapon of conscript AATs is the assault rifle, accounting for 81% of conscript AATs. After AAT, the majority of tinnitus cases resolve during military service and become asymptomatic. However, in one-fifth of the cases, tinnitus persists, causing problems such as sleeping and concentration difficulties in many.

In Finland, occupational tinnitus often remains unreported in conjunction with NIHL reports. In a survey of occupational NIHL cases, tinnitus was mentioned in only four per cent. However, a subsequent inquiry revealed that almost 90% in fact had tinnitus, indicating that most cases remained undetected and unreported.

The best way to prevent noise-related tinnitus is prevention of noise trauma. In the military, hearing protection guidelines have been revised several times over the years. These regulations have been effective in reducing hearing loss of professional soldiers. There has also been a reduction in cases with tinnitus, but the decrease was not significant. However, with improved hearing protection regulations, a significant reduction in the risk of more serious, disturbing tinnitus was observed.

4 Review of the literature

4.1 Introduction

Acute acoustic trauma (AAT) is a result of short-term exposure to loud noise, with a subsequent temporary or permanent hearing threshold shift and/or tinnitus, with exposure times up to minutes or hours, rather than days or years. The effect of long-term noise exposure, in contrast, is termed noise-induced hearing loss (NIHL). Subjective tinnitus indicates an individual sound experience without an external sound source (Eggermont, 2005). Most people have experienced tinnitus, at least of short duration, in some form, at some point in their life. It has been claimed that tinnitus, in fact, might be a condition existing in almost all normally hearing individuals. In the classic Heller and Bergman experiment (Heller & Bergman, 1953), normally hearing adults without tinnitus were placed in a sound-proof chamber and asked if they heard any sound. Of these subjects, 94% reported hearing sounds, which could be interpreted as tinnitus.

4.2 Historical aspects

Probably the earliest reference to noise-related tinnitus is provided by Paracelsus, who in the 1500's reported that loud noises often cause tinnitus. Francis Bacon in "Sylva Sylvarum", published in 1627, mentions temporary tinnitus after an explosion. Martinus Hartmann, in 1669, relates tinnitus to hereditary qualities and depression. The first known otologic publication, Du Verney's "Traité de l'organ de l'ouie" in 1683, included a section on tinnitus, and he attributed it to hyperexcitability of nerves, among other things. Jean Marie Gaspard Itard in 1821 mentioned that tinnitus was often found in ears previously exposed to loud noise. He also described the effects tinnitus could have on sufferers, and what painful and unpleasant treatments they would endure in the hope of curing their tinnitus (Stephens, 1987).

4.3 Epidemiology

In a randomly selected population, Hannaford et al. found overall tinnitus prevalence to be 17%, increasing with age (Hannaford et al., 2005). In a Swedish randomly selected group of 3,600 adults, without screening for otological disorders, total tinnitus prevalence as described by the terms "often" or "always" was 14.2% among the respondents in all age groups, varying from 5.8% in the 30-39 age group, up to 21.3% in the oldest age group (Axelsson & Ringdahl, 1989). In 590 randomly selected adults, in whom occupational noise exposure was excluded, tinnitus prevalence was 13.2% (Johansson & Arlinger, 2003), and it increased with age.

Tinnitus may be associated with a number of otological conditions and other factors (Heller & Bergman, 1953). These include some medications (Huang & Schacht, 1989; Arora et al., 2009; Dille et al., 2010), otologic diseases such as otitis media (Podoshin et al., 1997) and otosclerosis (Gristwood & Venables, 2003), age-related hearing loss

(Rosenhall & Karlsson, 1991; Podoshin et al., 1997), sudden deafness (Chiossoine-Kerdel et al., 2000; Sakata et al., 2008), Ménière's disease (Holgers & Finizia, 2001; Söderman et al., 2002), vestibular schwannoma (Baguley et al., 2006), trauma to the head and neck regions (Koefoed-Nielsen & Tos, 1982; Folmer & Griest, 2003), masticatory disorders (Kuttila, 2003), AAT (Axelsson & Hamernik, 1987; Chermak & Dengerink, 1987; Temmel et al., 1999) and long-term loud noise exposure (McShane et al., 1988). It is often difficult to isolate the specific cause of tinnitus, since individuals are affected by several potentially tinnitus-causing factors during their life. For instance, to the authors knowledge, it is not known, how much tinnitus is related to loud snoring, which is a frequent problem and sometimes a contributing factor to hearing loss (Sardesai et al., 2003).

The prevalence of tinnitus reports may in some cases be influenced by possible financial aspects, and therefore it may be higher among compensation claimants (Axelsson & Prasher, 2000). Another factor is the manner in which tinnitus is addressed: is it a matter of self-reporting, or are patients specifically asked about tinnitus (Axelsson & Barrenäs, 1992).

4.4 Noise and tinnitus

One of the most common causes of tinnitus is noise exposure (Coles, 1984; Meikle et al., 2004), and there is general agreement that NIHL is strongly associated with tinnitus (Griest & Bishop, 1998). Tinnitus may be related to various types of noise exposure: a single noise impulse, long-term exposure to impulses, continuous noise, or a combination of the above (Loeb & Smith, 1967; Anttonen et al., 1980; Alberti, 1987; Chermak & Dengerink, 1987; Temmel et al., 1999). In the database of the Tinnitus Archive, noise was the primary traumatic cause of tinnitus, with 13.0% of all respondents relating tinnitus to long-term noise exposure, and 10.6% to sudden loud exposure or blast (Meikle et al., 2004).

Due to the differences in individual noise exposure durations, noise level variation, and individual noise susceptibility, it is difficult to establish direct relationships between noise exposure, age, hearing loss and tinnitus. It is a reasonable assumption that factors damaging hearing also contribute to tinnitus (Axelsson & Prasher, 2000).

Descriptions such as “ringing”, “buzzing”, “hissing” or “humming” are commonly associated with tinnitus, in hearing impaired and in normally hearing subjects (Heller & Bergman, 1953; Meikle & Taylor-Walsh, 1984; Meikle et al., 1984; Stouffer & Tyler, 1990; Newman et al., 1994). Noise-related tinnitus is usually also tonal, or of the pure-tone type and continuous (Cahani et al., 1984; Axelsson & Sandh, 1985; Chermak & Dengerink, 1987; Kodama & Kitahara, 1990), and it, too, may be described e.g. as ringing, buzzing or whistling (Alberti, 1987; Chermak & Dengerink, 1987). Noise-related tinnitus is usually high pitched (Man & Naggan, 1981; Alberti, 1987; Nicolas-Puel et al., 2006). By Cahani et al., high-pitched tinnitus, typical of acoustic trauma, may be a result of well-defined cochlear damage, whereas lower pitched tinnitus could be found in processes involving larger portions of the cochlea, such as Ménière's disease (Cahani et al., 1983).

4.5 Tinnitus and hearing loss

Tinnitus is commonly associated with hearing loss (Axelsson & Sandh, 1985; Axelsson & Ringdahl, 1989; Phoon et al., 1993; Temmel et al., 1999). Griest and Bishop found the prevalence of tinnitus to increase with increasing thresholds shifts (Griest & Bishop, 1998). In contrast, there are also some studies that have not associated tinnitus prevalence with hearing loss (Alberti, 1987; McShane et al., 1988). Hearing loss related to AAT and long-term noise exposure is usually located at high frequencies (Axelsson & Sandh, 1985; Axelsson & Hamernik, 1987).

Tinnitus after noise exposure may be present although no measurable hearing loss is detected (Loeb & Smith, 1967; Cahani et al., 1983; Axelsson & Hamernik, 1987; Temmel et al., 1999), and not all patients with hearing loss experience tinnitus. Although tinnitus patients and subjects without tinnitus may have normal hearing when tested by routine pure-tone audiometry, otoacoustic emissions may reveal abnormalities. Normal pure-tone audiometry does not always constitute normal auditory function (McKee & Stephens, 1992).

In continuous long-term noise exposure, NIHL appears commonly before tinnitus (Axelsson & Barrenäs, 1992). In AAT, this is not necessarily the case, and tinnitus may be present without detected hearing loss (Axelsson & Barrenäs, 1992; Temmel et al., 1999). It has also been suggested that hearing loss may be present, but at such high frequencies that it is not detected (Tonndorf, 1981). Some patients with tinnitus may have hearing loss at high frequencies not detectable by conventional audiometry up to 8 kHz, supporting the supplemental value of high-frequency audiometry (Shim et al., 2009), but also contrary views exist (Balatsouras et al., 2005).

The relationship between hearing loss and experiencing tinnitus is not entirely clear, although both are often present simultaneously. Subjective perceived tinnitus levels seem to correspond with hearing loss levels (Hazell, 1981; Man & Naggan, 1981; Axelsson & Sandh, 1985; Mazurek et al., 2010). Mazurek et al reported a weak but significant correlation between hearing loss and higher tinnitus-related distress (Mazurek et al., 2010). In investigating the relationship between occupational NIHL and tinnitus, König et al. found the occurrence of tinnitus to be significantly related to steeper audiogram configurations, rather than hearing levels (König et al., 2006). Dias and Cordeiro found the prevalence and severity of tinnitus to increase with increasing hearing loss (Dias & Cordeiro, 2008).

Hallberg and Carlsson found that although perceived handicap correlated with hearing loss, the perceived handicap was surprisingly not influenced by the possible presence of tinnitus (Hallberg & Carlsson, 1991). Several studies have failed to show a relationship between tinnitus annoyance and the degree of hearing loss (Axelsson & Sandh, 1985; McShane et al., 1988; Hallberg et al., 1993). In fact, Hallberg and Erlandsson found that tinnitus complainers had better - even normal - hearing than noncomplainers (Hallberg & Erlandsson, 1993). The patients with tinnitus also in König and colleagues' study had, in fact, better hearing than those without tinnitus (König et al., 2006). It has been hypothesized that hearing loss may in fact reveal underlying tinnitus (Kaltenbach et al., 2005). The correlations between hearing loss, tinnitus parameters and tinnitus distress are inconclusive.

4.5.1 AAT, tinnitus and hearing loss

After AAT, tinnitus is frequently present (Chermak & Dengerink, 1987). By Atherley and colleagues, temporary tinnitus was induced in 89% of subjects with a 5-minute exposure to 110 dB sound pressure level (SPL) white noise (Atherley et al., 1968). In a study of 52 AAT cases, hearing loss was present immediately after the AAT in 98% and tinnitus in 92% (Axelsson & Hamernik, 1987). After acoustic trauma, Man and Naggan found tinnitus to be present in 79.4% of patients (Man & Naggan, 1981). In soldiers, Melinek et al. found tinnitus to be the most common complaint after AAT, with or without hearing loss. Tinnitus prevalence with or without hearing loss was 61.2% in their study, and the prevalence of tinnitus as a solitary subjective symptom without hearing loss was 26% after AAT (Melinek et al., 1976). In contrast, Temmel et al. found the prevalence of tinnitus without hearing loss after AAT in conscripts to be 6.2% (Temmel et al., 1999).

When exposing soldiers to rifle noise with unprotected ears, Dancer et al. found tinnitus to be induced directly after the exposure, instead of appearing later after exposure (Dancer et al., 1991). Unlike tinnitus appearance, the appearance of threshold shifts was distributed more widely, up to 1 hour after the exposure. In all subjects, tinnitus disappeared within 24 hours of exposure, and the hearing thresholds recovered in 24 hours in all but one case (Dancer et al., 1991). After AAT, tinnitus and hearing loss do not necessarily improve in direct relation (Markou et al., 2001; Moon, 2007), and tinnitus may be a more serious subjective problem than hearing loss (Stouffer & Tyler, 1990; Temmel et al., 1999).

4.5.2 Age, tinnitus and hearing loss

Tinnitus has been found to be related to hearing loss, rather than the age of the patient (Coles, 1984; Axelsson & Ringdahl, 1989; Axelsson & Barrenäs, 1992; Axelsson & Prasher, 2000; Rosenhall, 2003). Neuberger et al. found increased tinnitus rates with increasing hearing loss and age (Neuberger et al., 1992). McShane et al. found no increase in tinnitus prevalence with increasing age, but in contrast to several other studies, also no correspondence was found between hearing loss and tinnitus (McShane et al., 1988). In that study, no increase in tinnitus prevalence was found as the duration of noise exposure increased, in contrast to the observations of Palmer et al. (Palmer et al., 2002). Age does not seem to have a direct correlation with tinnitus, but the higher tinnitus prevalence in higher age groups is most likely due to the simultaneous higher prevalence of hearing loss (Chung et al., 1984). The author has not come across tinnitus studies involving elderly adults without hearing loss.

4.6 Impulse noise

Impulse noise is a term used to describe transient sounds of higher levels than background noise and short duration (Coles et al., 1968; Henderson & Hamernik, 1986; Hamernik & Hsueh, 1991). The characteristics of impulse noise consist of several factors: peak pressure level, the rise time of the impulse, pressure wave duration and number, level distribution and frequency spectrum (Coles et al., 1968; Liang, 1992; Starck et al., 2003).

Impulse noise is generally considered more damaging to the ear than steady state noise (Fausti et al., 1981; Kellerhals, 1981). Hearing loss development is likely to be accelerated in noisy environments containing also impulse noise, compared to only continuous noise (Henderson & Hamernik, 1986). Commonly, the peak amplitude has been the primary consideration of impulse noise damage risk criteria (Henderson & Hamernik, 1986). In steady-state noise, exposure duration is an important factor in addition to exposure level (Smoorenburg, 1992). The hazard from low-frequency impulses is reported to be lower than from higher frequency impulses (Price, 1983; Price, 1986; Price et al., 1989; Axelsson & Prasher, 2000).

In the case of recurrent noise impulses, the resulting damage is likely to depend on the interval between the impulse peaks. With impulses wider apart, around a few seconds or longer, the resulting hearing loss seems to diminish (Perkins et al., 1975; Roberto et al., 1994). A “priming effect” with lower level noise, before greater noise exposure, has been reported to protect the ear from damage (Roberto et al., 1994; Hamernik & Ahroon, 1999).

Tinnitus has been more often linked to impulse noise exposure than continuous noise (Axelsson & Sandh, 1985; Alberti, 1987; Axelsson & Barrenäs, 1992). However, in a large study of industrial workers, there was no association between tinnitus and type of noise (Neuberger et al., 1992).

4.7 Explosions and blast injuries

Blast waves are an extreme form of an (acoustic) pressure wave, resulting from an explosion. The changing society is faced with a terrorism threat, where civilians may also be affected, and a clinician with no military reference may therefore encounter blast injuries (Katz et al., 1989; Frykberg, 2002). Explosion injuries may result also from fireworks and pressure vessels (Mäkitie et al., 1997).

In Finland, according to the statistics of Tukes (Turvatekniikan keskus, Safety Technology Authority), six industrial explosions occurred in Finland in 2002 and four industrial explosion accidents were investigated by Tukes in 2005 (Rusanen & Laanti, 2003; Mattila & Rusanen, 2006). Industrial explosions directly caused by explosives have ranged from none to three annually in 2003-2007 (Heinsalmi & Mattila, 2008). The worse explosion in the recent years was not industrial, but caused by a self-made bomb exploding in a shopping mall in 2002, killing seven people and injuring nearly 200, of which 164 seeked medical care (Ojanen & Vainio, 2003). Over a 20-year period from 1985 to 2004, altogether 61 people died in explosions (Mäkitie & Pihlajamäki, 2006).

Peak overpressures of a blast wave may reach tens of kPa, while impulse noise is usually less than 2 kPa (160 dB SPL). Primary blast injury mainly involves air-containing organs, such as the ear, lungs, and bowel. Secondary injuries are caused by displaced debris from the explosion, and tertiary injuries by displacement of the whole body by the blast energy, with resulting fractures or amputation of limbs (Cernak et al., 1999; Chaloner, 2005). Quaternary injuries, such as burns and psychological effects, may be inflicted (Horrocks, 2001). Most serious injuries of bombing survivors are caused by secondary or tertiary blast effects (Horrocks, 2001; Frykberg, 2002). When a blast front reaches the body at supersonic speeds, the surface of the encountered tissue rapidly accelerates producing a stress wave, which is subsequently transferred into underlying tissues (Wightman & Gladish, 2001).

4.7.1 Blast exposure and damage to the ear

The damage to the ear most commonly encountered after an explosion is sensorineural hearing loss, tinnitus, and perforation of the tympanic membrane (TM) (Kerr & Byrne, 1975). At what pressure the TM ruptures may vary individually, but a value of 160 dB SPL (2 kPa) is proposed on account of experiments on the chinchilla by Henderson and Hamernik (Henderson & Hamernik, 1986). In a review by Richmond et al., TMs were found to rupture at 37-298 kPa (185-203 dB SPL) with slow increase in pressure, and in the case of blast waves, 15-16.5 kPa (approximately 178 dB SPL) (Richmond et al., 1989). In field conditions, however, the shock wave is intensified by the reflections in the ear canal (Richmond et al., 1989), which means that the levels at which TMs rupture are lower in reality. The detrimental effects of an explosion are likely to be more devastating in a confined than open space, because the closed environment enables the pressure wave to increase rapidly and sustain the overpressure longer (Kerr & Byrne, 1975; Leibovici et al., 1996). Rupture of the TM does not seem to protect the hearing (Kerr & Byrne, 1975). An isolated perforation of the TM, in conjunction with blast injury, does not seem to be an indicator of pulmonary injury or poor prognosis (Leibovici et al., 1999).

In animal studies of chinchilla, sheep and pigs exposed to 160 - 209 dB SPL, despite damage susceptibility differences between the species, common microscopic findings directly after blast exposure included the fracture of the reticular lamina, and dislocation of a large portion of the organ of Corti from the basilar membrane. Naturally, also TM and ossicular chain failures were found (Roberto et al., 1989). The features determining the damage to the ear depend on rise time of the impulse, intensity of the overpressure, and duration of the positive phase of the pressure wave (Kerr & Byrne, 1975).

Often, although the ear as an air-containing organ is one of the most common organs to be injured in an explosion, the primary concern of victims of blast injuries is not the trauma to the ear, which may therefore remain unattended, but more serious injuries take precedence (Garth, 1995). Other organs may be damaged, with life-threatening or deadly consequences, such as blast lung, hemopneumothorax and rupture of internal organs (Hirshberg et al., 1999; Horrocks, 2001; Chen et al., 2002; CDC, 2003).

4.7.2 Blast exposure and tinnitus

The literature on tinnitus after blast exposure is rather scarce, and often only hearing loss is mentioned, with no reference to possible tinnitus. This is understandable, since usually there are more serious consequences, often including the loss of lives. In the extensive Tinnitus Archive, 6.6% of all patients attributed their tinnitus to be the result of an explosion (Meikle et al., 2004). In a study of a terrorist bomb explosion in a municipal bus, where 22 people lost their lives and 48 were wounded, 23 patients were hospitalized, and 17 of them were followed for six months in the otolaryngologic outpatient clinic (Cohen et al., 2002). All but one patient had a perforated TM. The most common auditory complaints of these 17 patients were aural fullness and pressure (88%), tinnitus (88%), otalgia (53%), dizziness (41%) and aural discharge (53%). After the six month follow-up period, 40% of the patients initially complaining of tinnitus still had tinnitus. Tinnitus had improved in 13%, and in 47% it had disappeared by the end of the six month follow-up period. In that study, only 17 of the initially 48 wounded blast injury survivors underwent otologic evaluation. In explosion injuries, where severe injuries take precedence, subjectively minor symptoms may be overlooked.

Almost all survivors of a restaurant bombing in Belfast complained of tinnitus after the blast (Kerr & Byrne, 1975). Also, high-frequency hearing loss with subsequent spontaneous recovery was found in most patients. Miller et al. found the most common otological complaints after a large car bomb explosion to be TM perforation, tinnitus, deafness and otalgia (Miller et al., 2002). In that study, the patients underwent initial examination from the first day up to 10 months after the explosion, so the frequency of acute effects is not known. Pahor found the most frequent otologic symptoms in 111 hospitalized bombing victims to be deafness, high-pitched tinnitus, TM perforations, and earache. Otologic problems were found in less than one-third of the patients. Deafness was reported in 27 cases, and tinnitus in 26 cases. Twenty patients had perforated TMs. Hearing loss was mostly in the high-tones. In that study, all but one tinnitus case resolved spontaneously under follow-up (Pahor, 1981). In England, 12 patients suffered ear injuries after a bomb explosion at London Bridge. All of them had tinnitus and hearing loss, three had perforated TMs. One year after the incident, sensorineural hearing loss was present in 42%, and tinnitus was also present. The final outcome of tinnitus was not reported (Walsh et al., 1995).

4.8 Occupational tinnitus and hearing loss

Literature on the effects of occupational noise exposure focuses often on hearing loss, less on tinnitus. Tinnitus often accompanies occupational hearing loss (Neuberger et al., 1992). McShane found tinnitus prevalence to be 49.8% in a study of 3,466 claimants for occupational NIHL (McShane et al., 1988). Barrs et al. found the presence of tinnitus to be 58% in their study of 246 occupational NIHL claimants (Barrs et al., 1994), and Daniell et al. found tinnitus to be present in 64% of accepted occupational NIHL cases (Daniell et al., 2002). In Alberti's study of Workers' Compensation Board of Ontario, Canada, 58% of claimants with exposure to intense occupational noise reported tinnitus. Noise exposure and NIHL were by far the most common causes of tinnitus (Alberti, 1987). Phoon et al. found tinnitus in 23.3% of noise exposed workers (Phoon et al., 1993). The patients in Phoon's study had a lower prevalence of tinnitus than e.g. Alberti or McShane et al., but also the mean age of the patients was lower. When analyzing different age groups with and without occupational noise exposure, the risk for tinnitus roughly doubled in each age group, if there was a history of noise exposure (Coles, 1984). McShane et al. also found tinnitus to be related to noise exposure history (McShane et al., 1988).

The risk of tinnitus increases with the duration of occupational noise exposure (Palmer et al., 2002). However, the relationship between occupational noise exposure and tinnitus seems to be present, if there is also hearing loss, whereas in normally hearing individuals, occupational noise exposure and tinnitus do not seem to be related (Rubak et al., 2008). Rosenhall reported elderly men having continuous tinnitus twice as often after a history of occupational noise exposure, than without occupational noise exposure (Rosenhall, 2003).

In many industrial environments, steady-state background noise may be supplemented with impulse noise, which may increase the risk of AAT (Axelsson & Hamernik, 1987). On the other hand, also a protective priming effect of lower level noise exposure before higher intensity noise exposure has been described (Hamernik & Ahroon, 1999). The onset of occupational tinnitus is often slow (Axelsson & Prasher, 2000). Although slow, chronic development of noise-related effects are more commonly reported in literature, also acute acoustic insults occur, resulting in sudden development of symptoms (Axelsson & Hamernik, 1987). The noise level of 80 dB(A) is considered safe for the majority of individuals, but above 85 dB(A) noise reducing actions in the working environment are required (Lutman, 2000; EU, 2003). According to European Union (EU) regulations, the following noise exposure limits currently apply (EU, 2003):

Lower exposure action value (8 hours) 80 dB(A), and peak value 135 dB(C)
Upper exposure action value (8 hours) 85 dB(A), and peak value 137 dB(C)
Exposure limit value (8 hours) 87 dB(A), and peak value 140 dB(C).

In a study comparing the hearing levels of forest workers, shipyard workers and paper mill workers, differences were found according to the type of noise exposure. Shipyard workers exposed to impulse noise had worse hearing than forest and paper mill workers exposed to steady state noise (Toppila et al., 2000). However, in another study of workers, tinnitus could not be associated with noise type (Neuberger et al., 1992).

The interval between the beginning of occupational noise exposure and appearance of tinnitus may vary considerably. Axelsson and Barrenäs found the interval between the beginning of occupational noise exposure and tinnitus onset to be from 11 to 35 years, depending on the occupation (Axelsson & Barrenäs, 1992). Military professions showed the shortest delay for tinnitus appearance. It has been proposed that since tinnitus sometimes seems to be an early indicator of auditory damage (Dancer et al., 1991), the presence of tinnitus in regular follow-ups of workers could be of use in identifying workers at greater risk for threshold shift (Griest & Bishop, 1998). In contrast, tinnitus has been reported to arise after hearing loss in long-term noise exposure (Axelsson & Prasher, 2000). In industrialized countries, the risks of noise have generally been recognized, but noise-related risks are a problem in developing countries (Rösler, 1994).

The harmful otologic effects of occupational noise may be amplified by industrial chemical exposure such as toluene and styrene, but although the detrimental effects on hearing in animals have been observed, the relationship in humans is not entirely clear (Al-Otaibi, 2000; Hoet & Lison, 2008). Neither is it clear, to what extent chemical exposure increases the probability of tinnitus.

4.8.1 Occupational NIHL in Finland

In Finland, occupational NIHL cases have been decreasing for years. During the 1990's, the annual number of reports decreased from over 1,000 to roughly 800 annual reports (Kauppinen et al., 2000). The annual reported number was below 1,000 for years (Karjalainen et al., 2001; Karjalainen et al., 2002; Karjalainen et al., 2008). Lately, however, the reports have increased again, but figures are not comparable to previous ones, because of changes in reporting methods. This is also due to other causes, such as changes in legislature, which makes insurance companies responsible for the costs of occupational hazards, tightened hearing protection regulations for employees, and campaigns for increasing general knowledge of the effects of noise and NIHL (Karjalainen et al., 2008).

In Finland, disability categories range from 1 to 20, and the categories applicable for otologic disability compensation based on hearing levels are categories 2 to 10. Up till 2010, category 2 required a mean hearing threshold of 20-29 dB in the better ear in frequencies 0.5-1.0-2.0 kHz, but from 2010 onwards, frequencies 0.5-1.0-2.0 and 4.0 kHz have been taken into account. Disability category 10 requires an over 90 dB mean hearing threshold in these frequencies. From 2010 onwards, tinnitus has warranted the disability category to be increased by one. Also, if the hearing in the worse ear in the aforementioned frequencies is at least 35 dB worse than in the better ear, the disability category may be increased by one (STM, 2009).

The majority of occupational NIHL cases are mild. Over half of the reported occupational NIHL cases in Finland have been classified into disability category 1, constituting a better than 20 dB mean hearing threshold in frequencies 0.5-1.0-2.0 kHz (before 2010) in the better hearing ear (Karjalainen et al., 2002). Men constitute the majority of occupational NIHL cases, representing over 90%. In all, occupational NIHL cases have represented 17-25% of all reported occupational diseases in Finland (Karjalainen et al., 2001; Karjalainen et al., 2008). Due to the mostly slow development of NIHL over the years, most presently reported cases originate from the 1990s and 1980s, or even earlier (Kauppinen et al., 2000; Karjalainen et al., 2008). Not much information is available about occupational tinnitus in Finland.

4.8.2 Noise exposure, hearing loss and tinnitus in the military

The military is an environment with significant noise almost entirely of impulse type (Riihikangas et al., 1980), and therefore the military, including the Finnish Defence Forces (FDF), is a challenging workplace (Salmivalli, 1967; Ylikoski & Ylikoski, 1994; Job et al., 1998). Impulse noise exposure of conscripts, non-commissioned officers (NCO) and officers has been abundant, especially before hearing protectors were widely used. The military has traditionally presented a high-risk environment for AAT and NIHL, placing both regular personnel and conscripts at risk. The peak levels of different weapons range from around 130 dB SPL of the pistol, 160 dB SPL of the assault rifle, to approximately 180 dB SPL of the cannon, depending somewhat on ammunition, shooting circumstances and the side of the ear measured (Anttonen et al., 1980; Ylikoski & Pääkkönen, 1986). The frequency spectrum of hand-held weapons is higher than large caliber weapons (Ylikoski & Pääkkönen, 1986). The noise of the gunshot itself produces most of the noise, with movement of the gun mechanisms and shell producing relatively little noise (Pääkkönen, 1988). The greater the gun calibre, the more energy is present in the low frequencies of the noise spectrum (Pääkkönen, 1988). High-pitched sounds are probably more harmful to the ear than low-frequency sounds (Price, 1983; Price, 1986; Price et al., 1989; Axelsson & Prasher, 2000), and even a single shot may cause permanent damage to the ear (Anttonen et al., 1980; Axelsson & Hamernik, 1987).

Noise exposure especially by impulse noise has been substantial both in regular military personnel and in conscripts. Hearing protector regulations have been aimed to address the issue of military noise exposure (Ylikoski et al., 1987; Savolainen & Lehtomäki, 1996; Savolainen & Lehtomäki, 1997). Still, annually an estimated 200-350 conscripts suffer an acoustic trauma in the FDF (Savolainen & Lehtomäki, 2005). Military service still has an impact on the hearing loss and tinnitus incidence of young conscripts (Jokitulppo, 2009).

In a random sample of 699 regular army officers, hearing loss was found in 68% and normal hearing in 32% (Ylikoski & Ylikoski, 1994). Of all the subjects, including the normally hearing, 33.6% had occasional and 8.9% had constant tinnitus. In cases with disabling hearing loss, tinnitus was present in 68%. Tinnitus presence correlated strongly with the use of small caliber weapons, and tinnitus presence correlated negatively with the use of hearing protectors (Ylikoski & Ylikoski, 1994).

In another study of 422 regular army personnel, 33.2% had normal hearing (Salmivalli, 1967). Tinnitus was present in 25-56% depending on hearing loss, and in 16% of normally hearing subjects (Salmivalli, 1967). In the 422 subjects, 93.4% had a slow development of acoustic trauma extending over a period of years, whereas in 6.6% the injury occurred suddenly during a single event, such as a pistol shot, cannon shot or explosion (Salmivalli, 1967). Hearing loss was typically more common in high frequencies.

In Axelsson and Hamernik's study of 52 AAT victims, 23 were related to military service, with AAT causes including single gun shots, rapid firing by machine gun, cannon firing, explosions and fire crackers (Axelsson & Hamernik, 1987).

In a study of 2,200 randomly selected young conscripts exposed to noise, tinnitus was reported by 308 (14%) conscripts (Attias et al., 2002). Of the subjects experiencing tinnitus, 234 (76%) had NIHL, 15% had hearing loss of other etiology and 9% had normal hearing. On the other hand, of the 1,892 subjects with no tinnitus, 1,002 (53%) had NIHL, which seems a rather high figure for NIHL in young conscripts. Of all the 823 conscripts with normal hearing, 28 (3.4%) had tinnitus (Attias et al., 2002). In 5,000 male soldiers experiencing tinnitus, 355 (7%) were considered tinnitus help seekers and accepted treatment (Attias et al., 2002). In the tinnitus help seeking group of soldiers, a steep increase in tinnitus occurrence was found after age 30. Tinnitus occurrence was

6% below 10 years of military service, and then increased markedly. Hearing loss, on the other hand, showed only a near-linear increase with age (Attias et al., 2002).

4.9 Mechanisms of noise trauma

Kellerhals terms otological damage by noise into micro-mechanical and macromechanical (Kellerhals, 1981). The former includes damage inflicted by direct mechanical trauma such as hair cell (HC) damage, alterations in ciliary structure, and microscopic alterations such as cellular vacuolization and swelling of HC nuclei, resulting from metabolic rather than direct mechanical effects (Kellerhals, 1981). The latter stands for gross damage, such as ruptured TMs.

Cochlear damage following noise exposure may be also divided into primary and secondary changes (Bohne & Harding, 1999). Primary changes encompass degeneration of HCs, especially outer hair cells (OHC). Primary events are followed by secondary events, where progressive degeneration of supporting cells, afferent nerve fibers and additional HCs occurs.

Noise exposures of typically around 90 to 140 dB(A) generally damage the ear more metabolically, in a slower manner, rather than suddenly or mechanically. The damage potential depends on the level and duration of noise exposure (Clark, 1992). In contrast, above 140 dB, the damage is acute and to a great extent mechanical: the sound pressure wave produces mechanical forces that instantly damage the structures of the ear (Clark & Bohne, 1999). The former effect is termed NIHL and it develops slowly in contrast to AAT (Clark & Bohne, 1999). External sources capable of producing such high pressure waves often originate from explosives or firearms. Hence, the third type of waveform damage, the blast injury, represents an extreme form of pressure wave energy reaching the ear (Henderson & Hamernik, 1986).

In Spoendlin and Brun’s study of guinea pig cochleas, exposure to wide band noise of 110 to 140 dB, with various exposure times, caused different types of damage, with some of the damage being visible only a long time after the exposure ended (Spoendlin & Brun, 1973). Up to 90 dB, practically no microscopically visible damage was found, 90-130 dB produced primarily damage by metabolic changes, and above 130 dB irreversible structural damage developed (Spoendlin & Brun, 1973). At long exposures of below 120 dB, single OHC degeneration among normal HCs were found. Sensory cells were found to be affected by swelling, cytoplasm vacuolization and mitochondrial degeneration. For these changes, a metabolic disturbance rather than direct mechanical damage was suggested. At sound levels of 120-130 dB, damage attributed to both mechanical and metabolical origin was encountered, such as HC distortion and bending and fusion of ciliae. Damage was found to proceed even after exposure had ended. Exposures of above 130 dB caused structural alterations visible immediately after exposure. A 30 second exposure by 140 dB wide band noise caused the entire destruction of the organ of Corti in a sharply delineated area. At high exposure levels, inner hair cell (IHC) bulging and eventually rupture may occur (Spoendlin, 1971). Nerve fibers to IHCs swell and may rupture, and as a delayed finding, retrograde nerve degeneration ensues (Spoendlin, 1971). At high intensity levels, exposure time was not as important in determining damage as was exposure intensity. Intense noise exposure produces a wide variety of damage, including damage to the vascular supply of the inner ear, changes in the tectorial membrane, sensory hairs and tip links, and leads to functional changes also in the central nervous system (Saunders et al., 1991; Henderson et al., 2006). Generally, OHCs are damaged first and the IHCs later. Animal experiments indicate that smaller threshold changes generally relate to OHC loss, and larger threshold shifts are found with IHC loss (Hamernik et al., 1989). Oxidative stress, necrosis and apoptosis are involved in HC death (Henderson et al., 2006).

Secondary damage may occur even after noise exposure has terminated. When exposing soldiers with unprotected ears to rifle noise, Dancer and colleagues found the appearance of threshold shifts distributed up to 1 hour after the exposure, but tinnitus was induced directly after the exposure, instead of appearing later (Dancer et al., 1991). Tinnitus may be a more sensitive sign of damage to the ear, at least in high-level impulse noise exposure.

4.10 Susceptibility to damage by noise

Hearing loss increases with increasing noise exposure; with noise level, and exposure duration (Henderson & Hamernik, 1986; Lu et al., 2005; Da Costa et al., 2008). However, individual susceptibility to noise varies (Henderson & Hamernik, 1986; Lu et al., 2005). Susceptibility can be defined as receiving different consequences following similar exposure (Lu et al., 2005). Environmental and individual, probably hereditary, factors play a role in the development of hearing loss (Kaksonen et al., 2000). Factors such as leisure-time noise exposure, smoking, cholesterol levels, blood pressure, and use of non-steroidal anti-inflammatory analgetics probably also affect the level of hearing loss, not necessarily as independent factors, but together with other factors (Cruickshanks et al., 1998; Starck et al., 1999; Al-Otaibi, 2000; Kaksonen et al., 2000; Toppila et al., 2000; Toppila et al., 2001; Meyer et al., 2002). Audiometrically measured damage inflicted upon different individuals varies greatly, and there seems to be great variability to noise sensitivity among individuals (Henderson & Hamernik, 1986). Even somewhat surprising correlates, regarding individual sensitivity to damage by noise, such as fair iris color, have been reported (Da Costa et al., 2008). Not all factors affecting susceptibility to hearing loss are known, and even less is known about susceptibility to tinnitus.

4.11 Central involvement in tinnitus

We do not yet fully understand the generation of subjective tinnitus. It seems that tinnitus is a consequence of aberrant neural activity, which is interpreted as sound (Jastreboff, 1990). Several models, theories and explanations for tinnitus and the underlying pathophysiology have been presented over the years (Baguley, 2002; Eggermont, 2005; Kaltenbach et al., 2005). No single explanation for tinnitus has been found, and no single theory probably explains all forms of subjective tinnitus (Bartels et al., 2007). There is also no consensus on the level of the auditory pathway in which the tinnitus signal is actually generated (Norena & Eggermont, 2003). In addition to peripheral damage after noise exposure, subsequent central changes occur (Lockwood et al., 1998), and it seems likely that several central nervous system loci are involved (Bartels et al., 2007).

Traditionally, pathologic processes were thought to originate peripherally, with microscopic changes in HCs and surrounding structures resulting in altered function, with the changes in cochlear mechanics resulting in the perception of abnormal activity in auditory pathways, inducing tinnitus perception (Jastreboff, 1990). The neurophysiological model presented by Jastreboff offered a different approach, suggesting that tinnitus is a complex symptom of many potential causes, and should not be categorized simply into peripheral or central. Also, according to this model, the limbic system, associative areas and the prefrontal cortex of the brain are involved in the individual perception of tinnitus, which contains also an aspect of emotional involvement (Jastreboff, 1990). Tinnitus generation, pattern recognition neural networks and associative processes are probably involved in individual tinnitus perception (Jastreboff, 1990; Bartels et al., 2007; Han et al., 2009). The role of the prefrontal cortex hypothesized by Jastreboff has been later verified by Schlee and colleagues by magnetoencephalic recordings (Schlee et al., 2009). A change in the normal random activity of the peripheral auditory system, resulting from any cause, is detected by subcortical filters, and with complex associations with the limbic system, cortical areas and autonomic nervous system, tinnitus perception and reaction is formed (Jastreboff et al., 1996). If the signal is not assigned to a negative emotional state such as fear, it is classified as insignificant and may subsequently undergo habituation. However, if the signal is interpreted as alarming, with qualities of a threat, and therefore associated with a negative emotional state, detection of the signal is enhanced, and possibly the autonomic nervous system activated as a response to the threat, resulting in the development of tinnitus annoyance (Jastreboff et al., 1996). In this case, a self-strengthening circle of negative reinforcement and increased perception is initiated, and habituation to the signal prevented.

According to Rauschecker and colleagues, a peripheral cochlear lesion causes a void in the central representation of the lesioned frequency area, which is then filled by adjacent frequencies, which in turn become over-represented and amplified compared to other frequencies (Rauschecker et al., 2010). These edge frequencies also lose their inhibitory signal from the deafferented region, and increased activity results. Normally, this hyperreactivity (or unwanted tinnitus signal) is filtered in the thalamic region, and does not reach perception. Rauschecker and colleagues theorize that sound-evoked neural activity is transferred via the thalamus to the auditory cortex for perception, but the same signal is conveyed in parallel via the amygdala to the subcallosal paralimbic area, where the emotional content of the sound is evaluated, and from there connections back to the thalamic region normally result in filtering of repetitive unwanted noise, which does not reach the auditory cortex (Rauschecker et al., 2010). If the paralimbic system is not functioning, the tinnitus signal is not filtered at the thalamic gate and reaches perception, and later reorganization of the auditory cortex results in chronic tinnitus. The involvement of brain areas other than auditory processing areas, such as the limbic system, have been verified by imaging studies (Mirz et al., 2000).

Changes in the tonotopic map of the auditory cortex in tinnitus patients have been demonstrated by magnetoencephalographic recordings, where the cortical map was distorted in tinnitus patients (Mühlnickel et al., 1998). These authors also found a correlation between the subjective strength of tinnitus and tonotopic reorganization. Tinnitus and phantom limb symptoms seem to be similar in that they both involve cortical reorganization (Karl et al., 2004). There seems to be a relation between tinnitus distress and increased information flow from other cortical areas to the temporal cortices (Schlee et al., 2009).

4.12 Tinnitus sufferers and tinnitus suffering

Tinnitus is analogous to pain in that neither can be quantified by objective, external measurements, but instead the experience is individual and subjective. Both in acute tinnitus and in acute pain, peripheral causes may be found, such as damage to the ear by sound, or tissue damage in the case of pain. However, in chronic forms of tinnitus and pain, central mechanisms are involved (Möller, 2000).

Approximately 1-3% of the general population have been estimated to suffer from severe tinnitus (Axelsson & Ringdahl, 1989). House reports 10% experiencing serious disturbance (House, 1981). Also lower figures of 1% (Oliveira et al., 1999) and higher figures of over 20% have been reported (Meric et al., 1998), indicating variability in the selection of patients, and the definition of disabling tinnitus. In a randomly selected population, where 17% of respondents reported having tinnitus, 7.3% of tinnitus sufferers reported severe tinnitus annoyance (Hannaford et al., 2005). In that study, 20.9% of individuals experiencing tinnitus reported slight, 7.0% moderate and 2.2% a severe effect of tinnitus on the ability to lead a normal life (Hannaford et al., 2005). In Alberti's study, tinnitus was a major problem for 19% of subjects experiencing tinnitus, and a minor one for 39% (Alberti, 1987). By McShane and colleagues, of the people experiencing tinnitus, 29.2% considered it a major problem, 61.5% a minor problem and 9.3% were not at all bothered by their tinnitus (McShane et al., 1988). Tinnitus interfered with daily activities in 30% of their patients. In a study by Phoon and colleagues, 13.9% had consulted a doctor because of tinnitus (Phoon et al., 1993). Regardless of the percentages, it is safe to say that fortunately, in most cases, tinnitus is not seriously annoying.

4.12.1 Psychological patterns

Gerber et al. found no psychopathological pattern by which to characterize tinnitus patients, for instance no predominance of neurotic or somatisizing personalities were found, nor any evidence of increased psychosomatic symptomatology (Gerber et al., 1985). The authors did not find any distinguishing psychopathological features in the personality of those bothered by tinnitus, in comparison to those who were not. On the other hand, in a study of 150 patients with severe tinnitus, House found three psychological categories characterizing these subjects: depressive reactivity, hysterical conversion, and seriously disturbed patients with conversion reactions with schizoid features and character disturbances (House, 1981). McKee and Stephens also found neurotic personality traits in tinnitus patients (McKee & Stephens, 1992). Meric et al. found the psychological profile of half of tinnitus sufferers to be normal, with predominance of hysterical and weak depressive characteristics in the rest (Meric et al., 1998). Bartels and colleagues found that tinnitus patients with a distressed type personality were more likely to experience self-reported tinnitus-related distress (Bartels et al., 2010). They also found that tinnitus patients were more likely to have neurotic and negative general outlook tendencies, and the distressed type personality might be in fact associated with having tinnitus, and the personality type might also contribute to experienced tinnitus severity (Bartels et al., 2010).

4.12.2 Tinnitus problems

Sleeping disorders and concentration difficulties are common problems associated with tinnitus (Man & Naggan, 1981; Axelsson & Sandh, 1985; Jakes et al., 1985; Stouffer & Tyler, 1990). Meikle and Taylor-Walsh found 53% of tinnitus patients reporting sleeping difficulties due to tinnitus (Meikle & Taylor-Walsh, 1984). In a study of 2,442 industrial workers, over half of the patients were experiencing sleep disturbances because of tinnitus (Alberti, 1987). Alster et al. found that tinnitus sufferers in general complained of sleeping disturbances, and tinnitus annoyance correlated with sleep disturbances (Alster et al., 1993). Subjective tinnitus severity has been found to correspond with sleep disturbances also by other authors (Meikle & Taylor-Walsh, 1984; Meikle et al., 1984; Axelsson & Ringdahl, 1989). Hallberg and Erlandsson found a strong relation between tinnitus complaints and the presence of concentration difficulties, irritability and sleep disorders (Hallberg & Erlandsson, 1993). Difficulties in concentration are a major factor in predicting the effects of tinnitus on life satisfaction (Erlandsson & Hallberg, 2000).

Other discomforts experienced by tinnitus sufferers are difficulties in speech discrimination, difficulties in understanding speech, and even depression (Tyler & Baker, 1983; Axelsson & Sandh, 1985). Folmer et al. found an association between depression and subjective tinnitus severity (Folmer et al., 1999). A study comprising 80 military personnel with chronic tinnitus showed 37% of tinnitus sufferers to experience mild or moderate depressive symptomatology (Alster et al., 1993).

Hallberg and Erlandsson found tonal tinnitus less disturbing than other types of tinnitus in a study of tinnitus complainers and non-complainers (Hallberg & Erlandsson, 1993). The type of the tinnitus sound did not correlate with severity by Meikle and colleagues (Meikle et al., 1984). Man and Naggan found a significant relationship between measured tinnitus levels and subjective severity and presence. (Man & Naggan, 1981). They also reported an association, although statistically insignificant, between matched tinnitus levels, sleep disturbances, and concentration difficulties (Man & Naggan, 1981). Jakes et al. found a low but existing correlation between tinnitus loudness matches and tinnitus loudness self-reports (Jakes et al., 1986). However, they also found that audiometric measures of tinnitus did not relate to tinnitus complaints (Jakes et al., 1985). Meikle and Taylor-Walsh did not find a correlation between tinnitus loudness matching and severity, but there was a correlation between severity and sleeping disturbances (Meikle & Taylor-Walsh, 1984). Newman et al. found subjective tinnitus loudness to correlate with annoyance (Newman et al., 1994).

No consistent correlation seems to be evident between subjective loudness and perceived tinnitus severity or handicap, meaning that other factors than tinnitus loudness probably contribute to tinnitus annoyance (Folmer et al., 1999).

In the matter of suffering caused by tinnitus, the terms impairment, disability and handicap are used. These terms may generally be defined as follows (Tyler, 1993; Barbotte et al., 2001): *Impairment* is any temporary or permanent loss, or abnormality, of a body structure or function, whether physiological or psychological. The actual perception of tinnitus may be considered impairment. *Disability* is the consequence of impairment, i.e. a restriction or inability to perform an activity in the manner, or within the range, considered normal for a human being, mostly resulting from impairment. Thus, disability is the gap between what one wants or needs to do, and what one can do. Disability may be the effect of tinnitus on concentration or sleep, for instance. *Handicap* is the result of an impairment or disability that limits or prevents the fulfilment of one or several roles regarded as normal, depending on age, sex and social and cultural factors, i.e. the way in which a disability affects life. Tinnitus handicap would be, for instance, the feeling of anxiety, avoiding quiet situations, or experienced annoyance. Distinguishing between disability and handicap is not always clear. The handicap experience caused by tinnitus is complex. Non-auditory factors, such as environmental circumstances, might be involved in determining tinnitus suffering (Erlandsson et al., 1992; Hallberg et al., 1993).

4.13 Tinnitus assessment

The sensation level of tinnitus has been often found to be in the magnitude of less than 10 dB above the hearing level (Man & Naggan, 1981; Meikle & Taylor-Walsh, 1984). Although tinnitus measurements have been shown to be relatively reliable in most patients (Nageris et al., 2010), no systematic relation seems to exist between subjective tinnitus severity and audiometrically determined tinnitus level. Several studies have failed to show a systematic, significant correlation between severity ratings and measured tinnitus loudness (Meikle & Taylor-Walsh, 1984; Meikle et al., 1984; Axelsson & Sandh, 1985; Jakes et al., 1985). Man and Naggan found a correlation between matched tinnitus levels and sleeping and concentration difficulties, but the results were statistically insignificant (Man & Naggan, 1981). It has been postulated that the frequently observed lack of correlation between tinnitus problems and tinnitus measurements might be due to a methodological problem in tinnitus matching (Goodwin & Johnson, 1980; Risey et al., 1989). There is no consensus on the proper tinnitus matching protocol, and tinnitus matching cannot be used as an indicator of tinnitus severity.

Attempts have been made to address the apparent discrepancy between tinnitus loudness measures and subjective tinnitus loudness, but the relationship remains unclear (Henry et al., 1999). Because of the complex tinnitus mechanisms and various sounds heard by patients, it is difficult to match an external sound to a noise in the head, and find a simple relationship with audiological criteria (Hazell, 1981). Tinnitus, although characterized by such qualities as pitch, loudness and localization, apparently does not behave similarly as external sounds (Henry & Meikle, 2000).

Since it is not possible to measure tinnitus suffering with objective audiological criteria, a number of questionnaires have been devised by several authors to assess the handicap caused by tinnitus, and impact of tinnitus on the quality of life. These questionnaires include the Tinnitus Effects Questionnaire (Hallam et al., 1988; Hiller & Goebel, 1992), Tinnitus Handicap Questionnaire (Kuk et al., 1990), Tinnitus Reaction Questionnaire (Wilson et al., 1991), Subjective Tinnitus Severity Scale (Halford & Anderson, 1991), Tinnitus Handicap/Support Scale (Erlandsson et al., 1992), Tinnitus Cognitions Questionnaire (Wilson & Henry, 1998), and Tinnitus Handicap Inventory (Newman et al., 1996; Newman et al., 1998).

Tinnitus questionnaires are useful as a tool in determining tinnitus severity and in treatment follow-up evaluation (Newman et al., 1998; Folmer, 2002; Tyler et al., 2008; Landgrebe et al., 2010). Important qualities of questionnaires are reliability, test-retest reliability, internal consistency and validity (Tyler, 1993).

The 27-item Tinnitus Handicap Questionnaire (THQ) was developed to measure the patients' perceived degree of handicap due to tinnitus (Kuk et al., 1990). It describes the patient's views on physical health, emotional status and social consequences of tinnitus, as well as hearing difficulty. Each of the 27 items is answered with a numeric value 0-100. This questionnaire was later re-evaluated by Henry and Wilson (Henry & Wilson, 1998). In their assessment, the THQ was found to possess high internal consistency, and a good test-retest correlation. Low correlation was found between THQ and loudness matches of tinnitus, supporting the notion that audiological findings are not in direct relation to psychological measures (Henry & Wilson, 1998). There is no consensus on which questionnaire is best to assess overall tinnitus problems.

4.14 Treatment and prevention

Tinnitus treatment may be divided into two categories. On one hand, the objective may be to get rid of the tinnitus sound experience, or on the other, to eradicate or diminish the adverse reaction to tinnitus, making it less disturbing.

Various drugs have been utilized in the hope of finding a cure for tinnitus. Steroids have been reported to be useful (Sakata et al., 1997), and melatonin has provided relief for some patients (Rosenberg et al., 1998). Since there is an association between depression and tinnitus severity, regardless of the causative relationship or lack thereof, some tinnitus patients might benefit from antidepressant therapy (Folmer et al., 1999). Although some patients benefit, there is insufficient evidence to conclude that antidepressive medication would improve tinnitus (Baldo et al., 2006). No statistically significant effects were shown by trimetazine, prednisolone or complex vitamin B's (Markou et al., 2001). Tschopp and Probst evaluated nine treatments with no clear effect from them (Tschopp & Probst, 1989). Antioxidants have been proposed as a protective agent against NIHL (Henderson et al., 1999; Le Prell et al., 2007). In patients with NIHL and tinnitus, a low-cholesterol diet and antilipid therapy have produced favorable results (Sutbas et al., 2007). A universally effective tinnitus drug is still waiting to be found (Patterson & Balough, 2006; Shulman & Goldstein, 2006).

Transcranial magnetic stimulation has showed promising potential in relieving tinnitus (Chen et al., 1997; Eichhammer et al., 2003; Langguth et al., 2003; Plewnia et al., 2003; De Ridder et al., 2004; Londero et al., 2006).

Hyperbaric oxygen therapy has been reported to improve recovery from NIHL and tinnitus (Vavrina & Muller, 1995; Kuokkanen et al., 1997; Lamm et al., 1998; Kuokkanen et al., 2000). It is not yet uniformly accepted as standard AAT treatment, which is in large part due to the lack of sufficient prospective efficacy studies, which will also in the future be difficult to obtain, due to ethical concerns.

Some authors present a comprehensive tinnitus management method for clinicians, adapting components of treatment protocols such as Tinnitus Retraining Therapy (TRT) and sound therapy (Henry et al., 2005; Tyler et al., 2008). In TRT, the basic principle is the notion that tinnitus itself is not the main problem, but instead the problem is the adverse reaction to tinnitus. The key role for habituation of tinnitus is to get rid of the emotional state associated with the condition (Jastreboff et al., 1996). Successful habituation occurs usually within 12-18 months (Jastreboff et al., 1996), at which point it seems more effective than tinnitus masking (Henry et al., 2006). The same conclusion was reached also by Phillips and McFerran (Phillips & McFerran, 2010). According to the Cochrane Collaboration, masking therapy failed to show conclusive evidence of efficacy (Hobson et al., 2010).

Therapeutic sound in the treatment of tinnitus has been traditionally used as soothing, reducing contrast between tinnitus and background noise, or diverting attention from tinnitus (Henry et al., 2008). In Neuromonics Tinnitus Treatment, education and individually modified acoustic stimulation customized to the patients' hearing loss are used to achieve relief from tinnitus, improved relaxation and sleep, and later reduced tinnitus awareness (Davis et al., 2008; Hanley & Davis, 2008).

Contrary to treatment regimens that aim to facilitate tinnitus habituation, recent investigations have focused on modifying the tinnitus sound perception. Okamoto and colleagues reported decreasing tinnitus loudness by using customized music, which was spectrally filtered to contain no energy within one octave of the patient's matched tinnitus frequency. Their results after 12 months showed significantly reduced tinnitus loudness and decreased cortical activity, measured by magnetoencephalography, in areas corresponding to the tinnitus frequency (Okamoto et al., 2011). Vagus nerve stimulation and its potential to release neuromodulators, capable of promoting plastic changes in the nervous system, has been utilized with tone pairing by Engineer and colleagues, to alter neural and behavioural correlates of tinnitus in noise-exposed rats. The results indicated successfully abolished tinnitus (Engineer et al., 2011).

4.14.1 Hearing protection in the FDF

Because noise-related trauma is preventable, emphasis should be placed on protective measures. The guidelines for hearing protection in the military have been revised several times over the years. In 1968, the use of hearing protection was first ordered in shooting with hard ammunition. However, protection was generally not used until the 1970s, until muff-type protectors became readily available (Ylikoski & Tuominen, 1988). In 1979, muff-type protectors were ordered to be used in the shooting gallery, and plugs in open-air shooting galleries or combat training (Puolustusvoimat, 1979). In 1985, hearing protection was ordered to be used also when shooting blanks (Puolustusvoimat, 1985). In 1989 it was stated that the officer in charge of activities involving noise exposure must order all participants to use protectors, and make sure that suitable protectors are available and that training on protector usage is provided (Puolustusvoimat, 1989). However, each individual is responsible for using personal protectors.

In 1978, Riihikangas et al. found that 84.3% of conscripts did not use protectors in battle exercises, and in that study, 14.8% of conscripts developed hearing loss during military service (Riihikangas et al., 1980).

Earmuffs have been shown to be most effective against impulse noise of small caliber weapons, such as pistol shots, where attenuation is around 30 dB, and least effective for cannon shots, where attenuation may be only around 4 dB (Ylikoski et al., 1987). Toivonen et al. found earplug attenuation to be 18-20 dB, but point out that correct insertion of the plugs is a problem for some persons (Toivonen et al., 2002). Indeed, actual attenuation characteristics depend on individual characteristics, such as the anatomy of the head, possible bows of eye glasses under the cushion of the protectors, or hair, all of which may allow leakage and therefore worsen the attenuation of the protectors. The combination of correctly placed earplugs and muffs is the best choice.

5 Aims of the present study

The objective of this study was to examine aspects of tinnitus related to noise exposure. The more specific aims were to:

- determine conscript AAT causes, and assess tinnitus due to AAT (I)
- assess tinnitus related to blast exposure (II)
- assess long-term tinnitus related to previous AAT (III)
- investigate reporting of occupational tinnitus in Finland, and assess risk professions (IV)
- investigate the effectiveness of hearing protection regulations in reducing hearing loss (V) and tinnitus (VI) in military personnel.

6 Study designs and patients

A brief study group overview is presented in **Table 1**.

Table 1. *Outline of the six studies, with the number of subjects, and a brief description of the study group.*

Study	Subjects (n)	Description
I	119	Conscripts with AAT
II	29	Bomb explosion victims
III	101	AAT suffered 10-15 years earlier
IV	858/366	Occupational NIHL reports (858) and tinnitus questionnaires (366)
V, VI	252	Professional soldiers ending active duty

6.1 Study I

Despite strict safety regulations during firearm shooting, hundreds of conscripts still suffer an AAT in the FDF every year. The aim was to assess, why so many conscripts suffer AATs, and what the consequences of these AATs were. A total of 163 conscripts were treated because of AAT in the Central Military Hospital (CMH), Helsinki, Finland from January 1st to December 31st, 2000. After the exclusions described in Study I, 119 patients with 120 AATs were included in the study. Upon arrival to the CMH, each patient was given a thorough otological examination including pure-tone audiometry, and tinnitus matching to the contralateral ear. The examination and audiometry were performed in almost all cases within two days of the AAT, and in all cases within one week. A structured AAT form was used to register exposure data including the causative weapon, and hearing protection used at the time of gunfire exposure. In addition, a tinnitus questionnaire was administered (**Appendix 1**). The patients were then observed in the hospital and treated either with normobaric oxygen, hyperbaric oxygen or both, according to the severity of symptoms, audiological findings and judgement by an ear, nose and throat (ENT) specialist. Tinnitus was evaluated using the questionnaire at the initial visit and at the last follow-up visit, if the patient still had tinnitus upon discharge from the military service, one to 319 days after the AAT. The medical records of the participants were retrospectively reviewed, and questionnaire responses analyzed.

6.2 Study II

The objective was to investigate acute and subacute otologic consequences of a blast exposure. The opportunity presented itself in the unfortunate bomb explosion in a shopping mall, which occurred on October 11th, 2002, in the second largest shopping mall of Finland, killing seven people and injuring more than 160. Of injured individuals, 44 sought medical help because of ear-related symptoms. Twenty-nine of them were examined at the Helsinki University Hospital ENT department within one month of the explosion. At the first visit, the patients were given a thorough otological evaluation by an ENT specialist. A standard pure tone audiogram was obtained, and a short AAT form (**Appendix 2**) was filled to assess exposure data and the patients' otologic symptoms.

6.3 Study III

The objective was to investigate the prevalence, characteristics and long-term effects of tinnitus caused by AAT. The initial study group consisted of 418 former military conscripts, who had been treated between 1984 and 1989 at the CMH because of AAT, from exposure to impulse noise caused by firearms. All 122 patients reporting tinnitus still at discharge from military service were contacted by telephone, and 101 were reached, with the intention of investigating whether tinnitus was still present. Thereafter, a structured tinnitus questionnaire (**Appendix 3**), and the THQ (Kuk et al., 1990), were sent to 66 voluntary consenting subjects with tinnitus. Also, their medical records were reviewed. The characteristics of long-term tinnitus were analyzed.

6.4 Study IV

Occupational tinnitus was studied by retrospectively reviewing records of occupational NIHL reports in Finland in the year 2000, which was the most recent data available when initiating the study. The objectives of this study were to obtain information about occupational tinnitus in Finland, of which very scarce information is available. The study comprised all 858 occupational NIHL reports made to the register kept by the Finnish Institute of Occupational Health, Helsinki, Finland, in the year 2000. The severity of occupational NIHL cases reported in Finland were investigated, risk occupational fields identified according to the International Standard Classification of Occupations (ISCO-88), and prevalence of tinnitus among the reported NIHL cases analyzed. Audiograms were not available, but instead hearing disability categories were. A tinnitus questionnaire (**Appendix 4**) was sent to 366 patients whose disability category and contact information could be obtained.

6.5 Studies V and VI

Since there is no uniformly accepted treatment for AAT or NIHL, the effectiveness of preventive measures, i.e. hearing protection regulations, was assessed. The study group consisted of 252 officers and NCOs, discharged from active duty and examined at that point at the CMH ENT Department. The medical records and audiograms of two groups, those examined from 1984 to1986 (Period I) and those examined from 2003 to 2005 (Period II), were reviewed. Hearing protection regulations had been revised between Period I and Period II. Changes in hearing between the two time periods, during which regulations were revised, were investigated.

Study VI consisted of the same patients as Study V. The effect of hearing protection regulations on tinnitus between Period I and II was investigated. A tinnitus questionnaire was used for tinnitus assessment during Period II (**Appendix 1**), but it was not yet in use during Period I.

7 Methods

Studies I and III-VI were retrospective, Study II was prospective. Patient selection and exclusion criteria, and study protocols are discussed in detail in the original papers (I-VI). For assessing both short term and long-term consequences of AAT and tinnitus, studies I, III, V and VI were performed in the CMH because of several reasons. First, the age distribution of conscripts is fairly uniform, and the young age of conscripts eliminates the role of presbyacusis. Second, nearly all conscripts and all regular army personnel were men, making it unnecessary to differentiate analysis according to sex. Third, noise exposure among military personnel is substantial, and consists almost entirely of impulse noise by firearms, making study groups rather homogenous regarding noise exposure.

7.1 Tinnitus and noise exposure assessment

All questionnaires were in Finnish. For Study I, an AAT form, which was routinely used in the CMH, was utilized. The form contained questions about circumstances leading to AAT, causative weapons, hearing protection and number of shots fired. In addition, a tinnitus questionnaire in routine use at the CMH was used to assess main characteristics of tinnitus (**Appendix 1**). The same questionnaire was used in Study VI, since it was the questionnaire routinely used at the CMH for tinnitus assessment.

In Study II, a short questionnaire about otologic symptoms was used (**Appendix 2**).

In Study III, a more detailed questionnaire was sent to the subjects (**Appendix 3**). In addition, a Finnish translation of the THQ (Kuk et al., 1990) was used. This was considered suitable for long-term tinnitus effects analysis, but not for short term tinnitus assessment, such as in Studies I and II. In the acute and subacute period after AAT and onset of tinnitus (I, II), this type of questionnaire would probably be too complicated and in-depth, and might even lead to possible undesirable focusing on tinnitus by the patient, which is why shorter questionnaires were used.

In the study of occupational tinnitus (IV), a short and quick to complete tinnitus questionnaire was constructed (**Appendix 4**). The purpose was to facilitate a high response rate, since the main focus of the study was to obtain information on occupational tinnitus reporting in Finland in general.

7.2 Audiometry

Audiometry was performed by qualified technicians in a sound-proof booth with a calibrated clinical audiometer. In Study I and for Period II of Studies V and VI, the Interacoustic Clinical Audiometer AC40 was used. In studies V and VI, Period I, the Grason-Stadler 10 was used. In Study II, a Madsen Midimate 602 was used. In study IV, hearing was determined by the examining doctor, so the equipment in each case is not known. The level for normal hearing was determined the same as the normal screening level 20 dB, at frequencies 0.125, 0.5, 1.0, 2.0, 3.0, 4.0, 6.0 and 8.0 kHz. In Study III, an attempt was made to obtain new audiograms to correspond with the tinnitus questionnaires, despite the retrospective study design. Due to the scattered residential situation of the subjects throughout Finland, audiograms of only a few subjects were obtained, as the others were, due to lack of financial support, reluctant to arrange for their own travel expenses to participate in the study.

7.3 Statistical analysis

Statistical analyses were performed using a personal computer with SPSS 10.0 for Windows (SPSS Inc.) and SAS Statistical software v.9 for Microsoft Windows (SAS Institute Inc.). Subjective tinnitus parameter correlations were analyzed with Spearman's correlation coefficient (I, III). Audiogram comparisons between groups were performed using the Mann-Whitney U-test (V). Differences in tinnitus reports were analyzed with the chi-square test (VI). A Cox regression analysis was performed to analyze the risk of various kinds of tinnitus by assigning tinnitus values to "no tinnitus", "intermittent or mild tinnitus" and "constant or disturbing tinnitus" (VI). For statistical significance, $p<0.05$ or lower was required.

8 Results

8.1 AAT causes in conscripts (I)

The assault rifle was the most common causative weapon accounting for 81% of conscript AATs (I). The machine gun accounted for 8% and the cannon and explosives for less than 3% each. In 58%, noise exposure consisted of only one shot and 96% were exposed to 10 shots or less during the AAT. In 68%, the weapon was fired by someone other than the patient himself. A rather high number of young conscripts had hearing loss already upon entering military service (22%), and were excluded from the AAT study (I).

Hearing protection was not used by 88% of conscripts suffering an AAT during their military service (I). Adequate protection was reported by six individuals (5%), in which three AATs were caused by an assault rifle, and the rest by an explosive or cannon (unpublished). In the rest of the conscripts, the used protectors were either broken or shifted out of place. The reasons for not wearing any hearing protection are presented in Study I, Table 2, the most frequent cause being “accidental shot” (40%). There probably is some overlap, at least between the reasons “accidental shot” and “situation undemanding” (18%), raising the likely proportion of accidental shots to well over 50%.

8.2 Tinnitus and other otologic findings after AAT and blast exposure (I, II)

Tinnitus and hearing loss were dominating symptoms both after AAT and after blast exposure. Tinnitus prevalence after AAT, with or without hearing loss, in conscripts was 97%. Audiological findings of conscripts are presented in **Table 2**. After blast injury, the dominant symptom was also tinnitus, which was present in 66% (II).

Table 2. *Findings after conscript AATs at the initial visit, and at the last follow-up visit (at the time of discharge from military service, 1-319 days after the AAT) at the CMH.*

Finding	Initial examination		Last follow-up	
	n	%	n	%
Only hearing loss	3	2.6	16	13.5
Only tinnitus	60	51.7	27	22.7
Tinnitus and hearing loss	53	45.7	11	9.2
Symptoms not present	0	0.0	65	54.6
Missing data	4	-	1	-
Total	120	100.0	120	100.0

Hearing loss with or without tinnitus was present altogether in 48% after AAT (I) and in 55% after blast exposure (II). After AAT, tinnitus without hearing loss detected by routine audiometry was present in half the conscripts. In contrast, only hearing loss without tinnitus was present in three per cent (I). None of the subjects either before AAT or blast exposure had tinnitus prior to the noise trauma, but hearing loss in association with blast exposure was difficult to determine, since also elderly people were involved, and at least three patients with blast injury were aware of pre-existing noise trauma (II).

Of conscripts exposed to a single shot, tinnitus was initially present in 96% and hearing loss in 52% (I). At discharge, 23% had tinnitus, 23% had hearing loss, and 39% had either symptom still at discharge from the military service, indicating a long-lasting effect after exposure to only a single shot.

Although tinnitus was present in 97% of conscripts after AAT, the majority had resolved by the end of the military service, at which time tinnitus was still present in 32%, and 68% had resolved. Hearing loss was present in 48% after AAT and 23% persisted at the end of service. Thus, at the last visit before discharge, tinnitus, hearing loss or both were present in 45%, and 55% were cured. Most of the conscripts received hyperbaric oxygen treatment, so it is not known, what the spontaneous recovery rate of symptoms was, and how many were cured due to treatment.

TM perforations were found in one case after AAT and in 27.6% after blast exposure. Seven of the eight patients with a ruptured TM after blast injury were situated less than 10 meters from the explosion site. No significant correlation was observed between the degree of hearing loss and distance from the explosion site. The otologic symptoms after blast exposure are presented in Study II, Figure 2. Other than otologic injuries, some of them life-threatening, were sustained by 34.5% of these patients. Otologic symptoms showed a spontaneous healing tendency.

8.3 Long-term tinnitus after previous AAT (III)

Of 418 conscripts with AATs having occurred in the 1980s, all had tinnitus initially, and 122 (29%) had tinnitus at discharge from the military service. The rest (71%) did not have tinnitus at discharge. When contacted 10-15 years later, 101 of these 122 ex-conscripts were reached, and 66 (65%) of them still had tinnitus, 35% did not. Thus, 19% (29% x 65%) of all AAT induced tinnitus became permanent, under follow-up of over a decade (III). The mean THQ score was 24.2 on a relative scale of 0-100. Mean annoyance on a visual analogue scale (VAS) in the long-term was 38.7 (VAS 0-100), but 10% of the tinnitus patients experienced annoyance of over 70%, indicating severe permanent tinnitus. Thus, a firearm induced AAT during military service results in severe, persistent tinnitus in two per cent of all young conscripts with AAT.

8.4 Short and long-term tinnitus characteristics and related problems (I-IV, VI)

The subjective tinnitus annoyance distribution is presented in **Figure 1a** (short term tinnitus) and **1b** (long-term tinnitus). For **Figure 1a**, subjects with available tinnitus annoyance information collected within one month after AAT were included, supplementing the results presented earlier in Study I. Including AAT patients for a one-month entry period was considered appropriate here, for comparison with blast injuries, where the data collection period was, equally, one month after the incident.

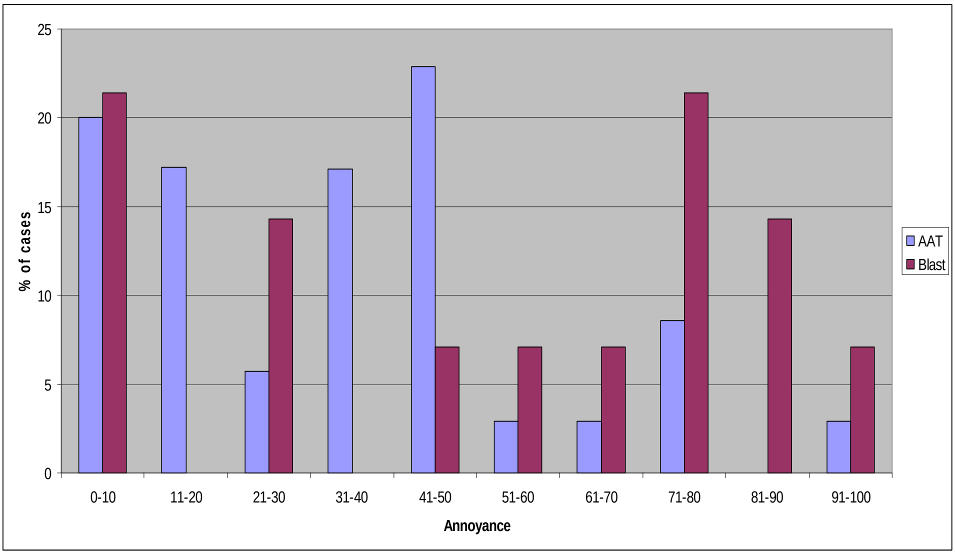

Figure 1a. *Subjective tinnitus annoyance VAS distributions in acute tinnitus after noise exposure: after AAT (Study I, n=35) and blast injury (Study II, n=14). Regarding blast injury, the VAS scale of 0-10 was transferred to 0-100, to correspond with the other studies.*

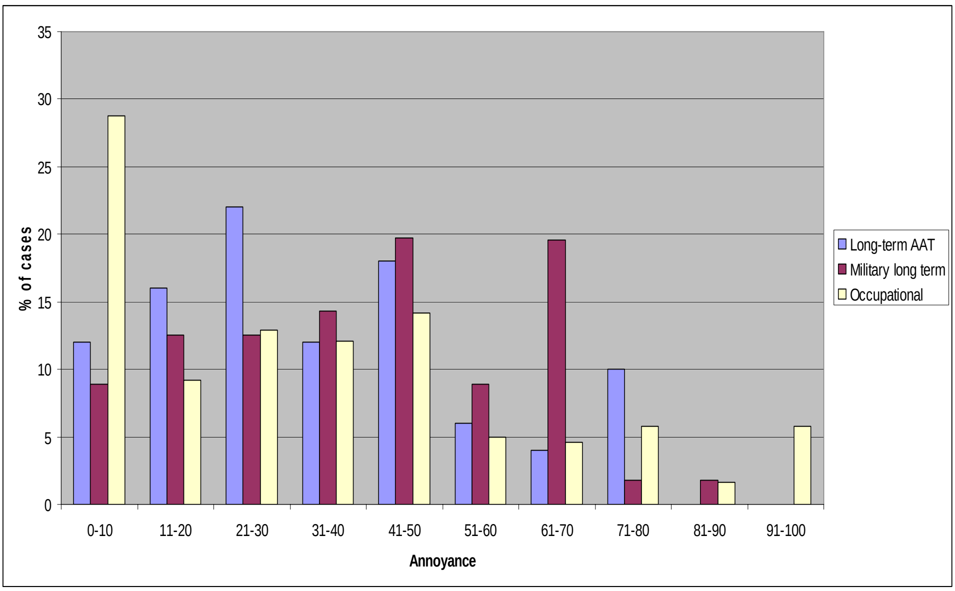

Figure 1b. *Subjective tinnitus annoyance VAS distribution in long-term tinnitus: long-term tinnitus after previous AAT (Study III, n=50), long-term military noise exposure tinnitus (Study VI, n=56), and occupational tinnitus (Study IV, n=240).*

After AAT, at the last follow-up visit to the CMH at discharge from military service, 13% of conscripts reported to be bothered by tinnitus constantly (I). After AAT, tinnitus was described as a high pitched whining, ringing or whistling sound in all cases (I). In the long-term, the descriptions were: whizzing 48%, ringing 27%, whistling 19% and hissing 6%, indicating a subjectively high-pitched sound in all cases (III). This was confirmed by the mean subjective pitch of 81.2 (VAS 0-100).

The mean VAS responses regarding subjective tinnitus parameters in each study are presented in **Table 3**.

Table 3. *The distribution of mean tinnitus parameters in the studies. The VAS ranged from 0 to 100, except in Study II, where a 0-10 scale was used. The value in that study has been transformed to correspond with the others. For Study I, only 16 cases with tinnitus forms and audiograms obtained at the day of the AAT were included. All decimal values have been rounded.*

Assessment	Study	Replies (n)	Loudness	Annoyance	Awareness	Disturbance (% of time)
AAT, immediately	I	16	33	39	57	50
AAT, last follow-up	I	16	35	37	39	33
Blast injury	II	14	-	56	-	-
AAT, long-term	III	50	50	39	76	24
Occupational, long-term	IV	240	40	42	67	57
NIHL, military long-term	VI	56	45	45	59	38

Shortly after AAT, immediate problems reported by the conscripts were concentration difficulties (50%), sleeping disorders (44%) and irritability (19%) (I). However, since only 16/116 (14%) completed the questionnaire immediately on the day of the AAT, the results are indicative. The results for long-term tinnitus (III and VI) are presented in **Table 4.**

Table 4. *Subjective effects of long-term tinnitus. The results of Studies III (Appendix 3, questions 50-58) and VI (Appendix 1, question 14) are presented. The positive/total response rates are presented. The response rates for these questionnaires were 52/60 (87%) in Study III and 56/86 (65%) in Study VI. All respondents in Study III and almost all in Study VI answered these questions.*

Study	Difficulties in falling asleep	Irritation/ Annoyance	Concentration problems	Depression
III	31/52 (60%)	26/52 (50%)	23/52 (44%)	12/52 (23%)
VI	29/55 (53%)	17/53 (32%)	18/55 (33%)	2/54 (4%)

In Study III, no change in tinnitus severity over the years was reported by 61%, improvement was experienced by 28% and worsening by 12%. Constant audibility of tinnitus was reported by 60%, and 25% experienced tinnitus annoyance every day. Eleven per cent suffered from tinnitus always when awake. Factors making tinnitus worse were quiet surroundings (71%), noises (48%), and tiredness (42%). Attenuating factors were sleep (19%) and relaxation (14%), and some other single factors were also mentioned.

Long-term tinnitus THQ relative scores averaged at 24.2, with most of the values distributed along the lower end of the scale (III, Figure 5). Sleeping difficulties correlated with the THQ score ($r=0.6$, $p<0.01$). Difficulties in falling asleep correlated with tinnitus annoyance ($r=0.49$, $p=<0.001$). One-third (35%) had visited a doctor after military service because of tinnitus.

8.5 Tinnitus localization (I-III, VI)

The localization of tinnitus and hearing loss symptoms is presented in **Table 5**.

Table 5. *The localization of symptoms in the Studies.*

Study	Symptom	Symptom localization	
		Left	Right
I	hearing loss	38%	29%
I	tinnitus	39%	30%
II	hearing loss/tinnitus	21%	10%
III	tinnitus	42%	19%
VI	tinnitus	46%	30%

8.6 Hearing loss (I, II, V)

Hearing loss related to AAT (I), blast injury (II) and long-term noise exposure (V) was found typically in the high frequencies: in AAT 4-5 kHz (I), blast injury 6-8 kHz (II) and long-term noise exposure 4-8 kHz (V). For the other studies, recent audiograms were not available.

8.7 Hearing loss versus tinnitus (III)

In Study III, although 87% (45/52) had hearing loss already at the last audiometry during military service, 33% considered tinnitus to be a worse problem than hearing loss, 35% considered them equal and 35% considered hearing loss more problematic than tinnitus (III).

8.8 Occupational NIHL and tinnitus (IV)

In 2000, 858 occupational NIHL reports were made to the Register of Occupational Health. Information about them was available in all but one case. Considering the total number of 2,228,557 workers in Finland that year, the average rate of NIHL reports was 3.8/10,000 workers. In Armed forces, 47.4 occupational NIHL cases were reported per 10,000 workers, making that occupational field most prone to NIHL. The frequency of reported NIHL cases in each occupational field, according to ISCO-88, is presented in Study IV, Table 1. The mean age of all occupational NIHL claimants was 51.6 years and 93.7% of patients were men. Mean noise exposure was 24.8 years, and for Armed forces

19 years (unpublished). In 63%, hearing loss was mild, warranting no disability compensation (disability category 1).

Tinnitus was the primary diagnosis in only three of the 857 cases (0.4 %), and tinnitus was mentioned altogether in 34 (4.0%) of all the reports. However, according to the questionnaire sent to 366 and returned by 307 (83.9%) subjects, whose contact information and hearing disability category could be determined, tinnitus had been present at the time the report was made in 88.7%. When the occupational NIHL report was made, 45.8% were asked whether or not they had tinnitus. Mean subjective tinnitus loudness was 39.7, annoyance 42.4, and awareness 67% (VAS 0-100). Furthermore, 91.6% of the respondents were interested in further examinations or treatment because of their tinnitus. In contrast, only 13% reported tinnitus to be a more significant problem than hearing loss, 45.9% reported them as equal problems and 38.2% considered hearing loss worse than tinnitus. Neither was a problem for 2.9%.

In the small number of tinnitus reports (34/857 cases), the economic activity most frequently represented was Public administration and defence (17/34), with the other tinnitus reports representing miscellaneously other economic activities.

Unfortunately, it was not possible to calculate the mean noise exposure time preceding the appearance of occupational tinnitus.

8.9 Hearing protection regulations in preventing hearing loss and tinnitus (V, VI)

There are strict hearing protection guidelines in the FDF, which have been revised several times over the years. The effectiveness of hearing protector guidelines was analyzed by comparing 252 officers and NCOs ending their active career after 3-36 years of noise exposure in the military during two separate time periods (Period I; 1984-1986 and Period II; 2003-2005), between which hearing protection guidelines were revised. Audiograms of regular army personnel showed most hearing loss to be at high frequencies (Study V, Figures I and II). Audiograms of NCOs showed significant improvement between Period I and Period II. The number of frequencies deviating from the expected normal thresholds also decreased in officers and NCOs from Period I to Period II. Improved hearing between Period I and Period II indicates that hearing protection regulations are useful in preventing hearing loss in the military.

Hearing protection regulations are useful in preventing serious tinnitus. The results for total tinnitus improvement in the same study groups, where hearing loss was found to improve, were not as clear (VI). Although the prevalence of tinnitus diminished both in officers and in NCOs from Period I to Period II, the difference was statistically insignificant. However, when tinnitus was classified into subgroups “no tinnitus”, “intermittent or mild tinnitus” and “constant or disturbing tinnitus”, the hazard ratio for constant or disturbing tinnitus was found to be significantly lower in Period II (Cox regression analysis, HR 0.60, 95% CI 0.37-0.96, p=0.032), indicating that tighter hearing protection regulations help diminish severe tinnitus.

Thus, Study V indicated a significant improvement in hearing with improved hearing protection regulations, but Study VI showed that although also tinnitus prevalence diminished, the improvement was not statistically significant, contrary to hearing loss improvement.

9 Discussion

This thesis confirmed that tinnitus and hearing loss are significant and frequent consequences of AAT, blast exposure, long-term military noise exposure, and occupational noise exposure. The importance of hearing protection regulations in the military was demonstrated both in protecting hearing and preventing severe tinnitus. Occupational tinnitus in Finland is underreported. As the research on tinnitus treatments progresses, it becomes even more important to identify tinnitus sufferers, so that the condition can be appropriately addressed.

9.1 Conscript AATs and hearing loss

Combat practice often involves sudden surprising situations, and accidental shots seem to happen frequently to conscripts (I). In an Austrian study, no protection was worn by 80% of conscripts who suffered an AAT, and an accidental shot was the most frequent reason for AAT (Temmel et al., 1999). In Study I, 88% of conscripts were not using hearing protectors, and a similar earlier finding was reported by Savolainen and Lehtomäki, who found 86% of conscripts not wearing hearing protection at the time of the AAT (Savolainen & Lehtomäki, 1996; Savolainen & Lehtomäki, 1997). In Study I, 5% reported wearing protectors. The correct application of hearing protectors may cause difficulties for some individuals, and the protectors themselves may be broken, or they may provide insufficient protection due to external conditions, such as cold weather, which may cause ear plugs to expand slowly (Savolainen & Lehtomäki, 1996). The reasons for not wearing protectors were previously similar to Study I: accidental shot, hasty situation etc. (Savolainen & Lehtomäki, 1996), indicating reasons other than negligence in the majority of cases. The fact is that accidents do happen, no matter how many guidelines are provided. Still, perhaps some conscript AATs could be prevented by improving training, since responses such as “forgot” and “not ordered” accounted for over 20% of the reasons why protectors were not used (I, Table 2). In Savolainen and Lehtomäki’s study, a single shot caused the AAT in 41% of cases (Savolainen & Lehtomäki, 1997), while the corresponding number in Study I was 58%, indicating that a relatively smaller number of subjects were exposed to several shots without adequate protection in the more recent study, which could be due to improved training and awareness of noise risks.

The assault rifle caused 81% of conscript AATs in 2000 (I), with almost no change since the 1980s, when it was responsible for 79% of AATs (Savolainen & Lehtomäki, 1997). In the FDF, in 2003, the assault rifle was still the most common causative weapon (83%) in AATs of conscripts (Savolainen & Lehtomäki, 2005).

An interesting observation was the rather high number of young conscripts with hearing loss already upon entering military service (22%), which were excluded from the AAT study (I). In the 1980’s, Riihikangas et al. found 18% of conscripts to have impaired hearing upon entering service using the 20 dB screening level (Riihikangas et al., 1980). Between 1983 and 1985, 15% of conscripts had a hearing impairment upon entering service (Ylikoski, 1986). In a more recent study, the percentage of conscripts with a hearing impairment upon entering service was 19% (Jokitulppo et al., 2006; Savolainen et al., 2008), which is still higher than in the 1980's. Tinnitus was experienced often or always by 8% of conscripts at entering military service (Savolainen et al., 2008), earlier figures are not available. Leisure-time noise exposure is probably increasing (Smith et al., 2000), and affects conscripts' hearing.

9.2 Tinnitus prevalence

Tinnitus was a common finding after AAT (97%) (I). This was in line with previous findings of Axelsson and Hamernik (92%) (Axelsson & Hamernik, 1987), and Temmel et al. (84%) (Temmel et al., 1999). Tinnitus was reported less frequently after blast exposure (66%) (II). One probable explanation is the often concomitant serious injury to other organs, and psychological trauma. Ringing of the ears and hearing loss are possibly regarded as normal phenomena after such a dramatic event, and other problems related to the incident take precedence.

Some other studies have focused more on other otologic consequences of bomb explosions than tinnitus or hearing loss, such as TM perforations, which seems understandable considering how serious incidents these explosions often are, possibly involving great numbers of lost lives and even hundreds of injured people (Miller et al., 2002). Tinnitus prevalence has been, however, reported to be rather low in some other studies, even as low as 23% (Pahor, 1981). This does actually seem relatively low, since almost as many (18%) had a TM perforation in that study. In contrast, Kerr and Byrne reported almost all victims suffering from tinnitus (Kerr & Byrne, 1975). In a study of a terrorist bomb explosion in a municipal bus, where 22 people lost their lives and 48 were wounded, all but one patient had a perforated TM, and tinnitus was present in 88% (Cohen et al., 2002). However, in that study, only 17 of the initially 48 wounded blast injury survivors underwent otologic evaluation. Sometimes, patients may not even be aware of having hearing loss or tinnitus (Kerr & Byrne, 1975). Therefore, all victims of blast injuries should undergo otologic evaluation, despite symptoms (Garth, 1995).

In the study of the long-term consequences of AAT (III), tinnitus was present in all 418 conscripts after AAT in the 1980's, in 122 (29%) at discharge from military service and in 19% 10-15 years later. Thus, tinnitus recovery within months after AAT was found in 71%, with a similar trend noted in the AAT study (I). However, it is not known to what extent the recovery was spontaneous, and to what extent due to treatment, which in Study I was in most cases either by hyperbaric or normobaric oxygen. Nevertheless, recovery diminished thereafter, so that of the remaining tinnitus cases, only a further 35% recovered later, and 65% persisted (III). In this age group, 30-40 years, according to epidemiologic studies, the prevalence of tinnitus in a population is usually below 10% (Ries, 1994; Adams & Marano, 1995; Benson & Marano, 1998; Nondahl et al., 2002; Tambs et al., 2003). Over the years, no change in tinnitus annoyance was reported by 61%, relief was experienced by 28% and worsening by 12% (III). In accordance, Stouffer and Tyler found 66% experiencing no change in tinnitus severity since onset of tinnitus, but in contrast to the findings presented here, they found improvement in only 5% and worsening in 28% (Stouffer & Tyler, 1990). A possible and probable explanation is patient selection, since their group consisted of audiology-otology patients of hospitals, where patients are bound to be tinnitus sufferers more likely than in Study III of former conscripts, who were not actively seeking help for otological problems.

The thesis demonstrated consistently that after AAT, in one-third of tinnitus cases, the symptom persists throughout military service (I, 32%; III, 29%), and two-thirds are cured during military service. Later, resolving of tinnitus is less likely, and only one-third (III, 35%) disappears during the next 10-15 years after the AAT. Thus, of all AAT-induced tinnitus, 19% becomes permanent.

Of all conscript AATs, about two percent result in severe tinnitus (III), which is in accordance with the general prevalence of severe tinnitus (Axelsson & Ringdahl, 1989). This represents 10% of persons experiencing persistent tinnitus after AAT (III).

9.3 Tinnitus and hearing loss

Hearing loss often coexists with tinnitus after AAT (I, III) and blast exposure (II), but is also related to long term noise exposure (IV, V, VI). Hearing loss dominated in the high frequencies, which is consistent with previous findings (Axelsson & Sandh, 1985; Axelsson & Hamernik, 1987). Tinnitus does not necessarily improve in direct relation to hearing loss improvement (Markou et al., 2001; Moon, 2007). Studies V and VI showed that significantly improved hearing does not necessarily mean significantly improved tinnitus in the same subjects. Tinnitus might be an earlier indicator of acoustic trauma (Dancer et al., 1991), which appears in some patients before hearing loss is detected, and persist even after hearing loss has recovered.

For some patients, tinnitus may be a worse problem than hearing loss, but at least in some cases the level of hearing loss is bound to influence this. Nevertheless, Hallberg and Erlandsson found that tinnitus complainers had better - even normal - hearing than noncomplainers (Hallberg & Erlandsson, 1993), indicating that tinnitus may be a worse subjective problem than hearing loss. This view is supported also by other authors (Axelsson & Sandh, 1985; Stouffer & Tyler, 1990), and was supported also by Study III.

Significant correlations in officers were found between hearing levels in the mid and high frequencies and tinnitus loudness and annoyance (Study VI, Table 5). This could, however, also indicate not necessarily an effect of tinnitus, but in fact an effect of hearing loss, although subjectively attributed to tinnitus. Hallberg and Carlsson found that although hearing loss correlated with perceived handicap, the perceived handicap was surprisingly not influenced by the possible presence of tinnitus (Hallberg & Carlsson, 1991). In Study VI, the questionnaire in the retrospective study setting, unfortunately, did not contain a question about which was more disturbing subjectively; hearing loss or tinnitus.

9.4 Occupational tinnitus

Occupational tinnitus was extremely underreported (4%) in comparison with the true prevalence of almost 90% (IV). In almost half the cases (45.8%) the participants had been asked, whether or not they had tinnitus, when the occupational NIHL report was made, indicating that clinicians are aware of tinnitus related to NIHL, but often do not report it. The low prevalence of tinnitus reports is likely to be a result of Finnish legislature, which up till recently has not regarded tinnitus as compensable, regardless of concomitant hearing loss. Previous studies have found occupational tinnitus rates to be higher. McShane et al. found half, and Alberti 58% of occupational NIHL claimants reporting tinnitus (Alberti, 1987; McShane et al., 1988). There is no uniform practice for reporting occupational NIHL. Some clinicians may report only cases that are entitled to compensation (disability category 2 or higher), and others may report milder cases, since the majority of the occupational NIHL cases were mild (category 1). Patients with normal hearing but with tinnitus are generally not reported. Unpublished data from 2001 to 2008 shows that the scarce prevalence of tinnitus diagnoses in occupational NIHL reports has not changed.

9.5 Problems related to tinnitus

Sleeping difficulties, concentration difficulties, irritability, and even depression were among the problems found to be attributed to tinnitus (III, VI). Similar findings have been reported in other studies (Man & Naggan, 1981; Meikle & Taylor-Walsh, 1984; Meikle et al., 1984; Axelsson & Ringdahl, 1989; Hallberg & Erlandsson, 1993). Sleeping difficulties correlated with the THQ score ($r=0.6$, $p<0.01$) (III), and also previous studies have demonstrated a relation between perceived tinnitus severity and sleeping difficulties (Meikle & Taylor-Walsh, 1984; Meikle et al., 1984; Alster et al., 1993).

The subjective parameters of tinnitus presented in **Table 3** do not seem to follow any particular time-related, systematic trend, except that annoyance seems to vary quite little over time. It should be noted that in Study I, only 16 of 116 (14%) tinnitus patients completed the questionnaire on the same day of the AAT, at the same time as the audiogram was obtained, making the results narrow-scoped. Perceived tinnitus loudness seems to even show an increasing tendency, comparing long-term with short-term tinnitus. There are several possible explanations for this. First, as tinnitus has a tendency to resolve after AAT, it is possible that the remaining cases are more severe to begin with. This could result in the persistence of the already initially more severe cases, which could result in a seemingly increasing loudness, as the milder cases resolve over time. The slight but visible increase in annoyance might support this theory. Second, the questionnaires themselves might draw attention to tinnitus, leading to aggravation of responses, possibly in the hope for financial benefit. This is especially visible in the case of tinnitus awareness in two studies (III and IV), where a long time had passed since anyone showed interest in the patients' tinnitus, possibly arising hopes of compensation or other benefit. This was especially clear in Study IV of occupational tinnitus, where a compensation claim was already filed because on NIHL. Literature confirms that the possibility of benefit might influence results (McShane et al., 1988; Dejonckere & Lebacq, 2005), although downright malingering is rather rare (Barrs et al., 1994). Third, the inquiry of tinnitus itself may have an influence on awareness, especially in Studies III, IV and VI. This inquiry influence is less likely in the other studies, because directly after AAT, the patients have not had time to ponder their tinnitus for long. These presented theories are in accordance with the reported disturbance of tinnitus (% of time) in the various studies, which generally seems to decreases or stagnate with time, except with occupational NIHL claimants (IV), where a higher disturbance rating is seen, in comparison to the other studies.

9.6 Asymmetry

The left ear seems to be more susceptible to damage by noise. None of the studies (I-VI) showed right side dominance of either tinnitus or hearing loss. Instead, tinnitus was found to dominate on the left side in Studies I, III, IV (unpublished observation) and VI, and hearing loss dominated in the left ear in Studies I, II, and V. Tinnitus dominance on the left side seems to be a rather frequent finding (Hazell, 1981; Meikle & Taylor-Walsh, 1984; Alberti, 1987; McShane et al., 1988; Axelsson & Ringdahl, 1989; Stouffer & Tyler, 1990; Axelsson & Barrenäs, 1992; Meikle et al., 2004). In contrast, Cahani et al. found most of the subjects with a greater hearing loss on the right side reporting right-sided tinnitus, while the majority of subjects with a greater hearing loss on the left reported tinnitus identically in both ears or louder on the right (Cahani et al., 1984). The authors suggest central processes rather than peripheral reasons to be the explanation for this unexpected finding (Cahani et al., 1984). Then again, also in that study, the subjects with normal hearing had tinnitus more frequently on the left (Cahani et al., 1984). The left ear seems to be more vulnerable in general (Khalfa et al., 1997), and it seems reasonable to expect tinnitus to be present in the ear with worse hearing (Cahani et al., 1984; McShane et al., 1988).

There seems to be a degree of asymmetry in the auditory system. In a study of a randomly selected population, the left ear was found to have worse hearing at high frequencies, regardless of handedness (Pirilä et al., 1991). In subjects exposed to gunfire noise, the left ear has been typically found to experience greater damage to hearing (Riihikangas et al., 1980; Cox & Ford, 1995). This has been often attributed to the shooting position of the dominating right-handed subjects, in which the sound is directed more to the left ear, due to the shooting position and shadowing effect of the head. However, there are several reports in literature concerning the possible differences of the right and left ear with different susceptibility to damage by noise. In a study of 4,277 army personnel, the left ear had significantly more pronounced hearing loss than the right ear, and left ears were more affected than right ears regardless of handedness (Nageris et al., 2007). In a study of 644 young military officers, Job et al. concluded that asymmetry of hearing loss between ears in military shooters indicates an intrinsic difference between ears, regardless of shooting posture (Job et al., 1998). The authors hypothesize that the left cochleas do not have the same characteristics as right cochleas, and that the left cochlea might be a more acute and sensitive sensor, and therefore more fragile. A good hearing threshold level in the right ear seems to be better protected from noise-induced temporary threshold shift than a good hearing level of the left ear (Pirilä, 1991). In that study, the average temporary threshold shift was higher in the left than in the right ear, the difference between ears being statistically indicative (Pirilä, 1991). In these experiments, broad-band noise was administered to the normally hearing subjects for a maximum of 8 hours daily and the 4 kHz frequency was tested for hearing. Pirilä and colleagues also found the hearing in the left ear to be worse in subjects exposed to occupational noise (Pirilä et al., 1991).

The reason for the apparent left-right asymmetry is not clear, and also symmetrical NIHL and tinnitus findings have been reported (Mazurek et al., 2010). Interestingly however, in that study, subjective tinnitus distress was significantly higher in patients with left-sided tinnitus, rather than right-sided (Mazurek et al., 2010). In the current studies I-VI, information on the handedness of the subjects was not available.

9.7 Hearing protection

Daniell et al. present the need for future studies to assess whether the strategy of personal hearing protectors and hearing conservation programs is an effective way of preventing the development or progression of noise-related hearing loss in workers with substantial noise exposure (Daniell et al., 2002). Studies V and VI indicate that hearing protection regulations are useful both in preventing hearing loss and severe tinnitus in military personnel. In a large study including the audiograms of over 20,000 workers, Davies and colleagues found a markedly decreased risk of hearing loss, if hearing protectors were used and hearing conservation programs attended (Davies et al., 2008). However, protector regulations are useless, if hearing protection is not used also in recreational activities, as often seems to be the case (Nondahl et al., 2006).

9.8 Compensation aspects

Many people with tinnitus cannot or will not accept their condition and thereafter seek compensation (Hart, 1999). There is an increasing tendency for occupational hearing loss claims (Touma, 1992; Daniell et al., 2002). In the United States, the Veterans Administration has reported a substantial rise in tinnitus-related disabilities during the last decade. In 2000, 144,000 tinnitus-related disabilities were reported, with $150 million paid in compensations. In 2005, 340,000 tinnitus-related disabilities were reported, with compensations amounting to $418 million (AudiologyOnline, 2006). It is estimated that in 2011, over 800,000 veterans will receive compensation amounting to $1.1 billion (ATA, 2010).

We have to date no objective way of measuring tinnitus for compensation claims or otherwise. Uniform guidelines and practices regarding hearing loss and tinnitus compensation are lacking and vary in different countries (Jayne, 1993; Arslan & Orzan, 1998; Al-Otaibi, 2000). Also, the patient’s complaint does not relate to the variables of tinnitus but to the annoyance resulting from tinnitus (Hart & Rubin, 1996).

Several considerations have to be made regarding tinnitus compensation (Vernon, 1995). First, it has to be determined, whether or not the individual actually experiences tinnitus. Second, it should be determined, whether the tinnitus claim is related to, or caused by, the causative agent addressed in the claim, and not some other factor. Third, the level of disturbance or impact on the quality of life due to tinnitus should be determined. No uniform protocol for the processes above currently exists. With our current knowledge and means of investigation, the conclusion reached by evaluating the above aspects, is a probability evaluation at best. NIHL may be impossible to distinguish from presbyacusis, and the situation is even more complicated if these conditions coexist (Daniell et al., 2002).

One aspect of compensation claims is the potential risk for aggravation of symptoms (Hart & Rubin, 1996). There might be an interest to exaggerate symptoms in the hope of financial benefit (McShane et al., 1988; Dejonckere & Lebacq, 2005) although in a study of 246 workers of occupational NIHL, Barrs et al. found malingering to be quite uncommon (9%) (Barrs et al., 1994).

NIHL is generally described as audiological findings and symptoms related to a probability of noise-related etiology (Arslan & Orzan, 1998). Also, it is a bit narrow-scoped to speak of NIHL, since tinnitus is often an important or even only part of subjective otologic damage. Tinnitus may be a worse problem than hearing loss, and therefore it should be considered in compensation aspects (Axelsson & Barrenäs, 1992). Since 2010, tinnitus has been considered in occupational NIHL claims also in Finland, making it possible to increase the disability category by one, in the presence of serious tinnitus (STM, 2009).

When considering compensation for hearing loss or tinnitus related to work, also leisure-time noise exposure should be considered, such as firearm use during hunting or target practice, listening to loud music, woodworking, and use of snowmobiles (Clark, 1992; May, 2000). The possible contributing role of lengthy noise exposure by snoring is difficult to assess (Sardesai et al., 2003), and it is not known to what extent it might be related to tinnitus.

9.9 Study limitations

Retrospective study designs have limitations, one of which is the lack of systematic gathering of e.g. questionnaires. In Study I, only a small number of tinnitus questionnaires were included in the analysis for AAT symptoms. This was because although almost all audiograms were obtained within two days of the AAT, and all within one week, the majority of patients had not filled the tinnitus questionnaire, and some were completed over one week after the AAT, but not included in the original analysis because of the spontaneous resolving tendency of AAT symptoms. However, for **Figure 1a**, a new analysis of Study I annoyance data was performed, including all questionnaires completed within one month after the AAT, to obtain comparable results between AAT and blast exposure, because the collection period for blast exposure data was also one month. On the other hand, in Study II, since the patients were examined up to one month after the blast exposure, the results may be somewhat prone to recollection bias.

In Study III, no recent audiograms were available, and the last audiograms were obtained at the end of the military service more than a decade earlier. An attempt was made to obtain new audiograms, but due to the scattered national residential situation of the subjects, only a few audiograms were obtained, and thus disregarded. Nevertheless, this is not a problem regarding the results, since hearing does not improve over time, making the subjective evaluation of tinnitus vs. hearing loss problems plausible. Furthermore, in Study III, a validated Finnish version of the THQ was not available, and thus a direct Finnish translation was used. In Studies IV-VI, no reliable knowledge was obtainable on actual hearing protector usage. If protectors were not regularly used, which is likely, since in Studies V and VI hearing loss was rather pronounced, the results regarding the conserving effect of hearing protectors could have been even stronger.

The material of the Occupational Tinnitus study (IV) is dated, as the data from year 2000 was the most recent available, when the study was initiated. However, unpublished data show that the proportion of occupational tinnitus reports even further diminished from 2001 to 2008, with only a few annual reported occupational tinnitus cases, making the results still applicable.

In Study VI, no tinnitus questionnaire was in use for Period I, only for Period II, so it was not possible to compare the effect of hearing protectors on tinnitus characteristics between study periods.

10 Conclusions

- The usual reason for AAT in conscripts is an accidental shot by an assault rifle (81%). In nine of ten conscript AATs (88%), no hearing protection was used at the time of the incident (I).

- Tinnitus is a frequent symptom related to AAT (I), blast exposure (II), and long-term noise exposure (IV, VI), especially in the military. After AAT, 97% of conscripts had tinnitus (I). After blast injury, tinnitus was present in 66% (II). In long-term military noise exposure, tinnitus was present in 69% (VI).

- Two-thirds of tinnitus disappears during the first year after AAT (I, 68%) (III, 71%), but later the resolution of tinnitus is less likely; two-thirds (65%) of the cases not resolving during military service become permanent (III). After AAT induced by firearm exposure, one-fifth (19%) of tinnitus cases become permanent (III). Ten per cent of AAT-induced tinnitus is severe (III).

- Occupational tinnitus is greatly underreported and underdiagnosed (IV). In reports, only four per cent of tinnitus cases are acknowledged, although in fact nine of ten occupational NIHL claimants have tinnitus. Military professions pose the greatest risk for occupational NIHL.

- Hearing protection regulations are useful in preventing hearing loss in the military, and they are also useful in preventing severe tinnitus (V, VI).

- Tinnitus might not improve in direct relation to improved hearing (V, VI).

11 Acknowledgements

[Acknowledgements are only available in printed form.]

12 Appendices

12.1 Appendix 1

The Tinnitus Questionnaire used in Study I and Study VI during Period II (2003-2005).

1. How long have you had tinnitus? _______________
2. Date of beginning of tinnitus: __________________
3. Did your tinnitus start: 1) without any apparent reason 2) during acoustic trauma 3) during ear infection 4) during other stress 5) other reason _____________________
4. Does the loudness of your tinnitus vary? yes / no
5. If it varies, for how long have you had louder tinnitus? ________________
6. Where do you localize your tinnitus? 1) right ear 2) left ear 3) both ears equally 4) both ears, more on the right 5) both ears, more on the left 6) other: ______________
7. How many different tinnitus sounds do you hear? ____
8. The tinnitus sound is (if you hear more than one sound, the most annoying sound): 1) ringing 2) whistling 3) whining 4) hissing 5) humming 6) whooshing 7) buzzing 8) other sound, specify: ________
9. How loud is your tinnitus?
0-----10------20------30------40------50-------60------70------80------90------100
(extremely quiet) (loudest imaginable sound)
10. How annoying/disturbing is your tinnitus?
0-----10------20------30------40------50-------60------70------80------90------100
(not annoying) (extremely annoying)
11. While awake, how often are you aware of your tinnitus?
0-----10------20------30------40------50-------60------70------80------90------100
(never) (all the time)
12. How often does tinnitus bother you?
0-----10------20------30------40------50-------60------70------80------90------100
(never) (all the time)
13. When is your tinnitus most disturbing (choose one alternative)?
1) after waking up 2) in the morning 3) in the afternoon 4) when going to sleep 5) at night 6) constantly 24 hours a day 7) all the time when awake
14. Tinnitus causes … (please circle):

1. irritation	yes	no
2. excitement/anxiousness	yes	no
3. depression	yes	no
4. difficulties in concentration	yes	no
5. difficulties in falling asleep	yes	no
6. awakenings at night	yes	no
7. deterioration in the quality of sleep	yes	no

8. other, please specify ____
15. Does tinnitus affect your ability to work?
1) not at all 2) has a slight effect 3) has a moderate effect 4) significant effect

12.2 Appendix 2

The questionnaire used to assess symptoms after a bomb explosion (Study II).

Acute acoustic trauma form

Name Date of birth
Profession:
Acoustic trauma date:___________; cause:_____________________________
Describe:____
Ear plugs at AAT: +/-:____

After AAT ears blocked +/- for____hrs/days
Tinnitus Rt/Lt started:_________ Now same/more/less
Most severe symptom: 1. hearing loss 2. tinnitus 3. hyperacusis
4.other:____

Earlier:
noise exposure +/-:____
ototoxic drugs +/-:_____________ ear infections:______________

Tinnitus frequency:____________Hz Loudness:__________dB
% of time when annoying
% of time when aware
severity: 0 1 2 3 4 5 6 7 8 9 10 changes in severity yes/no
annoyance: 0 1 2 3 4 5 6 7 8 9 10

Sound intolerance/ Hyperacusis Rt/Lt
Description of troublesome sound:
severity: 0 1 2 3 4 5 6 7 8 9 10 changes in severity: yes/no
annoyance: 0 1 2 3 4 5 6 7 8 9 10

ear plugs worn: % of time

when:____

Balance problem nil yes: constant attacks positional mild severe

ENG norm/pathol

12.3 Appendix 3

The Tinnitus Questionnaire used in Study III to assess long-term tinnitus. In addition, the Tinnitus Handicap Questionnaire (Kuk et al., 1990) was used.

1) Did you have tinnitus before the acoustic trauma? 1. no 2. yes

2) Where is your tinnitus located? (Circle ONE of the following)

1. left ear	6. in the head, no exact location
2. right ear	7. in the head, more on the right side
3. both ears equally	8. in the head, more on the left side
4. both ears, but worse in the left ear	9. outside of head
5. both ears, but worse in the right ear	10. middle of head

If you hear more than one sound or a different sound in each ear, answer the following questions with regard to the *most annoying* sound.

Use the scale below as a model in answering the following questions, in which you are requested to use a number between 0 and 100:

0------10------20------30------40------50-------60------70------80------90------100

3) Describe the most prominent PITCH of your tinnitus by using a number.
(0=very low, as a low pitched fog horn, 100=very high, as a high pitched whistle)
______ (Write a SINGLE number between 0 and 100 here)

4) Describe the LOUDNESS of your tinnitus by a using number.
(0=very weak tinnitus, 100=very loud tinnitus)
______ (Write a SINGLE number between 0 and 100 here)

5) How ANNOYING do you find your tinnitus?
(0=not annoying, 100=extremely annoying)
______ (Write a number between 0 and 100 here)

6) How often does your tinnitus interfere with your getting to sleep?
(0=does not interfere, 100=interferes every night)
______ (Write a number between 0 and 100 here)

7) To what degree are you DEPRESSED by your tinnitus?
(0=not at all depressed, 100=very depressed)
______ (Write a number between 0 and 100 here)

8) Do you have trouble concentrating because of your tinnitus?
(0=does not affect concentration, 100=always affects concentration)
______ (Write a number between 0 and 100 here)

9) Does tinnitus affect your ability to understand speech?
(0=not at all, 100=tinnitus extremely affects my ability to understand speech)
______ (Write a number between 0 and 100 here)

10) Does tinnitus affect your ability to work?
(0=not at all, 100=tinnitus extremely affects my ability to work)
______ (Write a number between 0 and 100 here)

11) Does your tinnitus ever change in PITCH?
1. no 2. yes, rapidly 3. yes, gradually

12) How often does the PITCH of your tinnitus change?
1. does not change 2. daily 3. weekly 4. monthly

13) Does your tinnitus ever change in LOUDNESS?
1. no 2. yes, rapidly 3. yes, gradually

14) How often does the LOUDNESS of your tinnitus change?
1. does not change 2. daily 3. weekly 4. monthly

15) Which of the following best describes your tinnitus sound? (Circle ONE option only)

1. humming	5. wheezing	
2. hissing	6. whistling	9. other, please specify: ____
3. whooshing	7. ringing	
4. buzzing	8. ticking	

16) Does the tinnitus ever change to a completely different sound?
1. no 2. yes, rapidly 3. yes, gradually

17) During the time you are awake, what percentage of the time is your tinnitus present?
(E.g. 100% = all the time, eg. 25% = tinnitus is present 1/4 of the time)
______ % (Write a number between 0 and 100 here)

18) On the average, how many days per month are you bothered by your tinnitus?
________ days per month (max 30 days per month)

When does tinnitus most bother you? (Circle ONE option on each lime)

	(1=no	**2=yes)**
19) when when waking up	1	2
20) before noon	1	2
21) in the afternoon	1	2
22) in the evening	1	2
23) when going to sleep	1	2
24) at night	1	2
25) always when awake	1	2
26) 24 hours a day	1	2

Since your tinnitus began, is it now... (Circle ONE option from each line):

27) 1. higher 2. lower 3. same IN PITCH
28) 1. louder 2. softer 3. same IN LOUDNESS
29) 1. higher 2. lower 3. same IN SEVERITY

30) When your tinnitus first began, how many sounds was it composed of? _____ number of sounds

31) How many sounds is your tinnitus composed of now?
______ number of sounds

32) How many years have you had tinnitus?
______ years

33) How many years has your tinnitus really bothered you?
______ years

How do the following affect your tinnitus? (Circle ONE option on each line)
(1=makes it worse 2=makes it better 3=does not affect)

34) alcohol	1	2	3
35) quiet surroundings	1	2	3
36) noise	1	2	3
37) excitement	1	2	3
38) coffee/tea	1	2	3
39) concentration	1	2	3
40) rest/relaxation	1	2	3
41) sleeping	1	2	3
42) physical effort	1	2	3
43) being tired	1	2	3

44) Does something, except the above mentioned, make your tinnitus WORSE?
1. no 2. yes, please specify _____________________

45) Does something, except the above mentioned, make your tinnitus BETTER?
1. no 2. yes, please specify _____________________

46) Does any MEDICATION make your tinnitus BETTER?
1. no 2. yes, please specify _____________________

47) Does any MEDICATION make your tinnitus WORSE?
1. no 2. yes, please specify _____________________

48) Does any FOOD make your tinnitus BETTER?
1. no 2. yes, please specify _____________________

49) Does any FOOD make your tinnitus WORSE?
1. no 2. yes, please specify _____________________

Does your tinnitus cause.... (Circle ONE option on each line)

	1=no	2=yes
50) irritation	1	2
51) excitement	1	2
52) depression	1	2
53) difficulty in concentrating	1	2
54) difficulty in falling asleep	1	2
55) waking up during the night	1	2
56) waking early in the morning	1	2
57) weakens quality of sleep	1	2
58)other, please specify _____________		

59) Which of the above (numbers 50-58) is the worst problem? Number _______ .

60) Which is a worse problem: hearing impairment or tinnitus? (Choose ONE of the following)

1. tinnitus is much worse than the hearing impairment
2. tinnitus is a little worse than the hearing impairment
3. tinnitus is as bad as the hearing impairment
4. the hearing impairment is a little worse than tinnitus
5. the hearing impairment is much worse than tinnitus

61) I am concerned, that my tinnitus is a symptom of a much worse disease.

1. no 2. yes

62) I am concerned, that I might go deaf because of tinnitus.

1. no 2. yes

63) Were you taking any medication just BEFORE your tinnitus began?

1. no 2. yes, please specify _________________

64) Are you taking any medication NOW?

1. no 2. yes, please specify _____________________

65) Are you using a hearing aid?

1. no 2. yes

66) If you use a hearing aid, to what extent does it help you, so that the tinnitus becomes softer or bothers you less when you use the hearing aid? (Circle ONE option) 1. helps much 2. helps a little 3. does not help 4. makes the tinnitus worse

67) If the hearing aid affects your tinnitus, how does the tinnitus change when you remove the device? (Circle ONE option)

1. tinnitus is inaudible for a while, how many minutes ? ______
2. tinnitus is inaudible for a while after removing the hearing aid, but it returns soon
3. tinnitus remains unchanged
4. tinnitus gets worse

12.4 Appendix 4

The tinnitus questionnaire sent to 366 of the 378 participants with a recorded hearing disability category and obtainable contact information, used in the study of occupational tinnitus (Study IV).

1) Do you experience tinnitus (ringing, humming or some other type of sound in the ears) lasting for over 5 minutes, without preceding alcohol consumption or sound exposure? (yes/no)

2) Did you have tinnitus in the year 2000, when your noise-induced hearing loss was reported? (yes/no)

3) Did the reporting physician ask, whether or not you have tinnitus? (yes/no)

4) Is your tinnitus …… 1. intermittent 2. continuous

5) How would you describe the loudness of your tinnitus on a scale from 0 to 100? (0=extremely quiet, 100=extremely loud, as a car horn): _____

6) How would you describe the annoyance tinnitus causes, on a scale from 0 to 100? (0=does not annoy me at all, 100=annoys me extremely)**: ______**

7) Tinnitus can be heard ______% of the time (0=I can never hear it, 100=I can hear it all the time).

8) Tinnitus bothers me ______% of the time I can hear it
(0=tinnitus never bothers me, 100= tinnitus bothers me always when I can hear it).

9) Where is your tinnitus sound localized?
1. left ear 2. right ear 3. both ears equally, or in the center of the head 4. both ears, but more on the left 5. both ears, but more on the right

10) In your opinion, is your hearing normal? (yes/no)

11) The worst problem is … (please circle one choice)
1. tinnitus 2. hearing loss 3. tinnitus and hearing loss equally 4. neither

12) Did your tinnitus develop suddenly or slowly?
1. suddenly 2. slowly

13) In your opinion, is your tinnitus related to the noise-induced hearing loss? (yes/no)

14) Would you be interested in further examination or treatment of tinnitus, if such should be available? (yes/no)

15) May we send you a more detailed tinnitus questionnaire or contact you by telephone? (yes/no).

13 References

Adams P.F. & Marano M.A. 1995. Current estimates from the National Health Interview Survey, 1994. *Vital Health Stat 10*, 1-260.

Alberti P.W. 1987. Tinnitus in occupational hearing loss: nosological aspects. *J Otolaryngol*, 16, 34-5.

Al-Otaibi S.T. 2000. Occupational hearing loss. *Saudi Med J*, 21, 523-30.

Alster J., Shemesh Z., Ornan M. & Attias J. 1993. Sleep disturbance associated with chronic tinnitus. *Biol Psychiatry*, 34, 84-90.

Anttonen H., Hassi J., Riihikangas P. & Sorri M. 1980. Impulse noise exposure during military service. *Scand Audiol Suppl*, 17-24.

Arora R., Thakur J.S., Azad R.K., Mohindroo N.K., Sharma D.R. & Seam R.K. 2009. Cisplatin-based chemotherapy: Add high-frequency audiometry in the regimen. *Indian J Cancer*, 46, 311-7.

Arslan E. & Orzan E. 1998. Audiological management of noise induced hearing loss. *Scand Audiol Suppl*, 48, 131-45.

ATA. 2010. American Tinnitus Association. Support for vets: One of our top priorities. http://www.ata.org/action-alliance/support-for-veterans.

Atherley G.R., Hempstock T.I. & Noble W.G. 1968. Study of tinnitus induced temporarily by noise. *J Acoust Soc Am*, 44, 1503-6.

Attias J., Reshef I., Shemesh Z. & Salomon G. 2002. Support for the central theory of tinnitus generation: a military epidemiological study. *Int J Audiol*, 41, 301-7.

AudiologyOnline. 2006. http://www.audiologyonline.com/news/news_detail.asp?news_id=2281.

Axelsson A. & Sandh A. 1985. Tinnitus in noise-induced hearing loss. *Br J Audiol*, 19, 271-6.

Axelsson A. & Hamernik R.P. 1987. Acute acoustic trauma. *Acta Otolaryngol*, 104, 225-33.

Axelsson A. & Ringdahl A. 1989. Tinnitus--a study of its prevalence and characteristics. *Br J Audiol*, 23, 53-62.

Axelsson A. & Barrenäs M.-L. 1992. Tinnitus in noise-induced hearing loss. *Noise induced hearing loss*, Mosby Year Book, 269-276.

Axelsson A. & Prasher D. 2000. Tinnitus induced by occupational and leisure noise. *Noise Health*, 2, 47-54.

Baguley D.M. 2002. Mechanisms of tinnitus. *Br Med Bull*, 63, 195-212.

Baguley D.M., Humphriss R.L., Axon P.R. & Moffat D.A. 2006. The clinical characteristics of tinnitus in patients with vestibular schwannoma. *Skull Base*, 16, 49-58.

Balatsouras D.G., Homsioglou E. & Danielidis V. 2005. Extended high-frequency audiometry in patients with acoustic trauma. *Clin Otolaryngol*, 30, 249-54.

Baldo P., Doree C., Lazzarini R., Molin P. & McFerran D.J. 2006. Antidepressants for patients with tinnitus. *Cochrane Database Syst Rev*, CD003853.

Barbotte E., Guillemin F. & Chau N. 2001. Prevalence of impairments, disabilities, handicaps and quality of life in the general population: a review of recent literature. *Bull World Health Organ*, 79, 1047-55.

Barrs D.M., Althoff L.K., Krueger W.W. & Olsson J.E. 1994. Work-related, noise-induced hearing loss: evaluation including evoked potential audiometry. *Otolaryngol Head Neck Surg*, 110, 177-84.

Bartels H., Staal M.J. & Albers F.W. 2007. Tinnitus and neural plasticity of the brain. *Otol Neurotol*, 28, 178-84.

Bartels H., Middel B., Pedersen S.S., Staal M.J. & Albers F.W. 2010. The distressed (Type D) personality is independently associated with tinnitus: a case-control study. *Psychosomatics*, 51, 29-38.

Bartels H., Pedersen S.S., van der Laan B.F., Staal M.J., Albers F.W. & Middel B. 2010. The impact of Type D personality on health-related quality of life in tinnitus patients is mainly mediated by anxiety and depression. *Otol Neurotol*, 31, 11-8.

Benson V. & Marano M.A. 1998. Current estimates from the National Health Interview Survey, 1995. *Vital Health Stat 10*, 1-428.

Bohne B. & Harding G.W. 1999. Noise-induced hearing loss. http://oto2.wustl.edu/bbears/noise.htm.

Cahani M., Paul G. & Shahar A. 1983. Tinnitus pitch and acoustic trauma. *Audiology*, 22, 357-63.

Cahani M., Paul G. & Shahar A. 1984. Tinnitus asymmetry. *Audiology*, 23, 127-35.

CDC. 2003. Centers for Disease Control and Prevention. Explosions and blast injuries. http://www.bt.cdc.gov/masscasualties/explosions.asp.

Cernak I., Savic J., Ignjatovic D. & Jevtic M. 1999. Blast injury from explosive munitions. *J Trauma*, 47, 96-103; discussion 103-4.

Chaloner E. 2005. Blast injury in enclosed spaces. *Bmj*, 331, 119-20.

Chen R., Classen J., Gerloff C., Celnik P., Wassermann E.M., Hallett M. & Cohen L.G. 1997. Depression of motor cortex excitability by low-frequency transcranial magnetic stimulation. *Neurology*, 48, 1398-403.

Chen X.L., Wang Y.J., Wang C.R. & Li S.S. 2002. Gunpowder explosion burns in fireworks factory: causes of death and management. *Burns*, 28, 655-8.

Chermak G.D. & Dengerink J.E. 1987. Characteristics of temporary noise-induced tinnitus in male and female subjects. *Scand Audiol*, 16, 67-73.

Chiossoine-Kerdel J.A., Baguley D.M., Stoddart R.L. & Moffat D.A. 2000. An investigation of the audiologic handicap associated with unilateral sudden sensorineural hearing loss. *Am J Otol*, 21, 645-51.

Chung D.Y., Gannon R.P. & Mason K. 1984. Factors affecting the prevalence of tinnitus. *Audiology*, 23, 441-52.

Clark W.W. 1992. Hearing: the effects of noise. *Otolaryngol Head Neck Surg*, 106, 669-76.

Clark W.W. & Bohne B.A. 1999. Effects of noise on hearing. *Jama*, 281, 1658-9.

Cohen J.T., Ziv G., Bloom J., Zikk D., Rapoport Y. & Himmelfarb M.Z. 2002. Blast injury of the ear in a confined space explosion: auditory and vestibular evaluation. *Isr Med Assoc J*, 4, 559-62.

Coles R. 1984. Epidemiology of tinnitus: (2) demographic and clinical features. *J Laryngol Otol, Suppl*, 9, 195-201.

Coles R.R., Garinther G.R., Hodge D.C. & Rice C.G. 1968. Hazardous exposure to impulse noise. *J Acoust Soc Am*, 43, 336-43.

Cox H.J. & Ford G.R. 1995. Hearing loss associated with weapons noise exposure: when to investigate an asymmetrical loss. *J Laryngol Otol*, 109, 291-5.

Cruickshanks K.J., Klein R., Klein B.E., Wiley T.L., Nondahl D.M. & Tweed T.S. 1998. Cigarette smoking and hearing loss: the epidemiology of hearing loss study. *Jama*, 279, 1715-9.

Da Costa E.A., Castro J.C. & Macedo M.E. 2008. Iris pigmentation and susceptibility to noise-induced hearing loss. *Int J Audiol*, 47, 115-8.

Dancer A., Grateau P., Cabanis A., Vaillant T. & Lafont D. 1991. Delayed temporary threshold shift induced by impulse noises (weapon noises) in men. *Audiology*, 30, 345-56.

Daniell W.E., Fulton-Kehoe D., Cohen M., Swan S.S. & Franklin G.M. 2002. Increased reporting of occupational hearing loss: workers' compensation in Washington State, 1984-1998. *Am J Ind Med*, 42, 502-10.

Davies H., Marion S. & Teschke K. 2008. The impact of hearing conservation programs on incidence of noise-induced hearing loss in Canadian workers. *Am J Ind Med*, 51, 923-31.

Davis P.B., Wilde R.A., Steed L.G. & Hanley P.J. 2008. Treatment of tinnitus with a customized acoustic neural stimulus: a controlled clinical study. *Ear Nose Throat J*, 87, 330-9.

De Ridder D., De Mulder G., Walsh V., Muggleton N., Sunaert S. & Moller A. 2004. Magnetic and electrical stimulation of the auditory cortex for intractable tinnitus. Case report. *J Neurosurg*, 100, 560-4.

Dejonckere P.H. & Lebacq J. 2005. Medicolegal decision making in noise-induced hearing loss-related tinnitus. *Int Tinnitus J*, 11, 92-6.

Dias A. & Cordeiro R. 2008. Association between hearing loss level and degree of discomfort introduced by tinnitus in workers exposed to noise. *Braz J Otorhinolaryngol*, 74, 876-83.

Dille M.F., Konrad-Martin D., Gallun F., Helt W.J., Gordon J.S., Reavis K.M., Bratt G.W. & Fausti S.A. 2010. Tinnitus onset rates from chemotherapeutic agents and ototoxic antibiotics: results of a large prospective study. *J Am Acad Audiol*, 21, 409-17.

Eggermont J.J. 2005. Tinnitus: neurobiological substrates. *Drug Discov Today*, 10, 1283-90.

Eichhammer P., Langguth B., Marienhagen J., Kleinjung T. & Hajak G. 2003. Neuronavigated repetitive transcranial magnetic stimulation in patients with tinnitus: a short case series. *Biol Psychiatry*, 54, 862-5.

Engineer N.D., Riley J.R., Seale J.D., Vrana W.A., Shetake J.A., Sudanagunta S.P., Borland M.S. & Kilgard M.P. 2011. Reversing pathological neural activity using targeted plasticity. *Nature*, 470, 101-4.

Erlandsson S.I. & Hallberg L.R. 2000. Prediction of quality of life in patients with tinnitus. *Br J Audiol*, 34, 11-20.

Erlandsson S.I., Hallberg L.R. & Axelsson A. 1992. Psychological and audiological correlates of perceived tinnitus severity. *Audiology*, 31, 168-79.

EU. 2003. Directive 2003/10/EC of the European Parliament and of the Council of 6 February 2003 on the minimum health and safety requirements regarding the exposure of workers to the risks arising from physical agents (noise) (Seventeenth individual Directive within the meaning of Article 16(1) of Directive 89/391/EEC).

Fausti S.A., Erickson D.A., Frey R.H., Rappaport B.Z. & Schechter M.A. 1981. The effects of noise upon human hearing sensitivity from 8000 to 20 000 Hz. *J Acoust Soc Am*, 69, 1343-7.

Folmer R.L. 2002. Long-term reductions in tinnitus severity. *BMC Ear Nose Throat Disord*, 2, 3.

Folmer R.L. & Griest S.E. 2003. Chronic tinnitus resulting from head or neck injuries. *Laryngoscope*, 113, 821-7.

Folmer R.L., Griest S.E., Meikle M.B. & Martin W.H. 1999. Tinnitus severity, loudness, and depression. *Otolaryngol Head Neck Surg*, 121, 48-51.

Frykberg E.R. 2002. Medical management of disasters and mass casualties from terrorist bombings: how can we cope? *J Trauma*, 53, 201-12.

Garth R.J. 1995. Blast injury of the ear: an overview and guide to management. *Injury*, 26, 363-6.

Gerber K.E., Nehemkis A.M., Charter R.A. & Jones H.C. 1985. Is tinnitus a psychological disorder? *Int J Psychiatry Med*, 15, 81-7.

Goodwin P.E. & Johnson R.M. 1980. The loudness of tinnitus. *Acta Otolaryngol*, 90, 353-9.

Griest S.E. & Bishop P.M. 1998. Tinnitus as an early indicator of permanent hearing loss. A 15 year longitudinal study of noise exposed workers. *Aaohn J*, 46, 325-9.

Gristwood R.E. & Venables W.N. 2003. Otosclerosis and chronic tinnitus. *Ann Otol Rhinol Laryngol*, 112, 398-403.

Halford J.B. & Anderson S.D. 1991. Tinnitus severity measured by a subjective scale, audiometry and clinical judgement. *J Laryngol Otol*, 105, 89-93.

Hallam R.S., Jakes S.C. & Hinchcliffe R. 1988. Cognitive variables in tinnitus annoyance. *Br J Clin Psychol*, 27 (Pt 3), 213-22.

Hallberg L.R. & Carlsson S.G. 1991. Hearing impairment, coping and perceived hearing handicap in middle-aged subjects with acquired hearing loss. *Br J Audiol*, 25, 323-30.

Hallberg L.R. & Erlandsson S.I. 1993. Tinnitus characteristics in tinnitus complainers and noncomplainers. *Br J Audiol*, 27, 19-27.

Hallberg L.R., Johnsson T. & Axelsson A. 1993. Structure of perceived handicap in middle-aged males with noise-induced hearing loss, with and without tinnitus. *Audiology*, 32, 137-52.

Hamernik R.P. & Hsueh K.D. 1991. Impulse noise: some definitions, physical acoustics and other considerations. *J Acoust Soc Am*, 90, 189-96.

Hamernik R.P. & Ahroon W.A. 1999. Sound-induced priming of the chinchilla auditory system. *Hear Res*, 137, 127-36.

Hamernik R.P., Patterson J.H., Turrentine G.A. & Ahroon W.A. 1989. The quantitative relation between sensory cell loss and hearing thresholds. *Hear Res*, 38, 199-211.

Han B.I., Lee H.W., Kim T.Y., Lim J.S. & Shin K.S. 2009. Tinnitus: characteristics, causes, mechanisms, and treatments. *J Clin Neurol*, 5, 11-9.

Hanley P.J. & Davis P.B. 2008. Treatment of tinnitus with a customized, dynamic acoustic neural stimulus: underlying principles and clinical efficacy. *Trends Amplif*, 12, 210-22.

Hannaford P.C., Simpson J.A., Bisset A.F., Davis A., McKerrow W. & Mills R. 2005. The prevalence of ear, nose and throat problems in the community: results from a national cross-sectional postal survey in Scotland. *Fam Pract*, 22, 227-33.

Hart C.W. 1999. Medicolegal aspects of tinnitus. *Int Tinnitus J*, 5, 63-6.

Hart C.W. & Rubin A.G. 1996. Medicolegal aspects of neurotology. *Otolaryngol Clin North Am*, 29, 503-20.

Hazell J.W. 1981. Patterns of tinnitus: medical audiologic findings. *J Laryngol Otol Suppl*, 39-47.

Hazell J.W. 1981. Measurement of tinnitus in humans. *Ciba Found Symp*, 85, 35-53.

Heinsalmi K. & Mattila M. 2008. Toimialan onnettomuudet 2007. Tukes.

Heller M.F. & Bergman M. 1953. Tinnitus aurium in normally hearing persons. *Ann Otol Rhinol Laryngol*, 62, 73-83.

Henderson D. & Hamernik R.P. 1986. Impulse noise: critical review. *J Acoust Soc Am*, 80, 569-84.

Henderson D., Bielefeld E.C., Harris K.C. & Hu B.H. 2006. The role of oxidative stress in noise-induced hearing loss. *Ear Hear*, 27, 1-19.

Henderson D., McFadden S.L., Liu C.C., Hight N. & Zheng X.Y. 1999. The role of antioxidants in protection from impulse noise. *Ann N Y Acad Sci*, 884, 368-80.

Henry J.A. & Meikle M.B. 2000. Psychoacoustic measures of tinnitus. *J Am Acad Audiol*, 11, 138-55.

Henry J.A., Zaugg T.L. & Schechter M.A. 2005. Clinical guide for audiologic tinnitus management II: Treatment. *Am J Audiol*, 14, 49-70.

Henry J.A., Zaugg T.L., Myers P.J. & Schechter M.A. 2008. Using therapeutic sound with progressive audiologic tinnitus management. *Trends Amplif*, 12, 188-209.

Henry J.A., Flick C.L., Gilbert A., Ellingson R.M. & Fausti S.A. 1999. Reliability of tinnitus loudness matches under procedural variation. *J Am Acad Audiol*, 10, 502-20.

Henry J.A., Schechter M.A., Zaugg T.L., Griest S., Jastreboff P.J., Vernon J.A., Kaelin C., Meikle M.B., Lyons K.S. & Stewart B.J. 2006. Clinical trial to compare tinnitus masking and tinnitus retraining therapy. *Acta Otolaryngol Suppl*, 64-9.

Henry J.L. & Wilson P.H. 1998. The Psychometric Properties of Two Measures of Tinnitus Complaint and Handicap. *Int Tinnitus J*, 4, 114-121.

Hiller W. & Goebel G. 1992. A psychometric study of complaints in chronic tinnitus. *J Psychosom Res*, 36, 337-48.

Hirshberg B., Oppenheim-Eden A., Pizov R., Sklair-Levi M., Rivkin A., Bardach E., Bublil M., Sprung C. & Kramer M.R. 1999. Recovery from blast lung injury: one-year follow-up. *Chest*, 116, 1683-8.

Hobson J., Chisholm E. & El Refaie A. 2010. Sound therapy (masking) in the management of tinnitus in adults. *Cochrane Database Syst Rev*, 12, CD006371.

Hoet P. & Lison D. 2008. Ototoxicity of toluene and styrene: state of current knowledge. *Crit Rev Toxicol*, 38, 127-70.

Holgers K.M. & Finizia C. 2001. Health profiles for patients with Meniere's disease. *Noise Health*, 4, 71-80.

Horrocks C.L. 2001. Blast injuries: biophysics, pathophysiology and management principles. *J R Army Med Corps*, 147, 28-40.

House P.R. 1981. Personality of the tinnitus patient. *Ciba Found Symp*, 85, 193-203.

Huang M.Y. & Schacht J. 1989. Drug-induced ototoxicity. Pathogenesis and prevention. *Med Toxicol Adverse Drug Exp*, 4, 452-67.

Jakes S.C., Hallam R.S., Chambers C. & Hinchcliffe R. 1985. A factor analytical study of tinnitus complaint behaviour. *Audiology*, 24, 195-206.

Jakes S.C., Hallam R.S., Chambers C.C. & Hinchcliffe R. 1986. Matched and self-reported loudness of tinnitus: methods and sources of error. *Audiology*, 25, 92-100.

Jastreboff P.J. 1990. Phantom auditory perception (tinnitus): mechanisms of generation and perception. *Neurosci Res*, 8, 221-54.

Jastreboff P.J., Gray W.C. & Gold S.L. 1996. Neurophysiological approach to tinnitus patients. *Am J Otol*, 17, 236-40.

Jayne T.R. 1993. Legal remedies for hearing loss. *In: Medical-legal evaluation of hearing loss*, R.A. Dobie Ed. New York: Van Nostrand Reinhold, 222-242.

Job A., Grateau P. & Picard J. 1998. Intrinsic differences in hearing performances between ears revealed by the asymmetrical shooting posture in the army. *Hear Res*, 122, 119-24.

Johansson M.S. & Arlinger S.D. 2003. Prevalence of hearing impairment in a population in Sweden. *Int J Audiol*, 42, 18-28.

Jokitulppo J. 2009. Non occupational noise - sources, exposure and effects on hearing, Natural and environmental sciences, Kuopio University.

Jokitulppo J., Toivonen M. & Bjork E. 2006. Estimated leisure-time noise exposure, hearing thresholds, and hearing symptoms of Finnish conscripts. *Mil Med*, 171, 112-6.

Kaksonen R., Pyykkö I., Kere J., Starck J. & Toppila E. 2000. Hereditary hearing loss--the role of environmental factors. *Acta Otolaryngol Suppl*, 543, 70-2.

Kaltenbach J.A., Zhang J. & Finlayson P. 2005. Tinnitus as a plastic phenomenon and its possible neural underpinnings in the dorsal cochlear nucleus. *Hear Res*, 206, 200-26.

Karjalainen A., Aalto L., Jolanki R., Keskinen H., Mäkinen I. & Saalo A. 2001. Ammattitaudit 2000. Työterveyslaitos.

Karjalainen A., Aalto L., Jolanki R., Keskinen H., Mäkinen I. & Saalo A. 2002. Ammattitaudit 2001. Työterveyslaitos.

Karjalainen A., Palo L., Saalo A., Jolanki R., Mäkinen I. & Kauppinen T. 2008. Ammattitaudit ja ammattitautiepäilyt 2006. Työterveyslaitos.

Karl A., Muhlnickel W., Kurth R. & Flor H. 2004. Neuroelectric source imaging of steady-state movement-related cortical potentials in human upper extremity amputees with and without phantom limb pain. *Pain*, 110, 90-102.

Katz E., Ofek B., Adler J., Abramowitz H.B. & Krausz M.M. 1989. Primary blast injury after a bomb explosion in a civilian bus. *Ann Surg*, 209, 484-8.

Kauppinen T., Heikkilä P., Lehtinen S., Lindström K., Näyhä S., Seppälä A., Toikkanen J. & Tossavainen A. 2000. Työ ja terveys Suomessa v.2000. Työterveyslaitos.

Kellerhals B. 1981. Acute acoustic trauma. *Adv Otorhinolaryngol*, 27, 114-20.

Kerr A.G. & Byrne J.E. 1975. Concussive effects of bomb blast on the ear. *J Laryngol Otol*, 89, 131-43.

Khalfa S., Morlet T., Micheyl C., Morgon A. & Collet L. 1997. Evidence of peripheral hearing asymmetry in humans: clinical implications. *Acta Otolaryngol*, 117, 192-6.

Kodama A. & Kitahara M. 1990. Clinical and audiological characteristics of tonal and noise tinnitus. *ORL J Otorhinolaryngol Relat Spec*, 52, 156-63.

Koefoed-Nielsen B. & Tos M. 1982. Posttraumatic sensorineural hearing loss. A prospective long-term study. *ORL J Otorhinolaryngol Relat Spec*, 44, 206-15.

Kuk F.K., Tyler R.S., Russell D. & Jordan H. 1990. The psychometric properties of a tinnitus handicap questionnaire. *Ear Hear*, 11, 434-45.

Kuokkanen J., Aarnisalo A.A. & Ylikoski J. 2000. Efficiency of hyperbaric oxygen therapy in experimental acute acoustic trauma from firearms. *Acta Otolaryngol Suppl*, 543, 132-4.

Kuokkanen J., Virkkala J., Zhai S. & Ylikoski J. 1997. Effect of hyperbaric oxygen treatment on permanent threshold shift in acoustic trauma among rats. *Acta Otolaryngol Suppl*, 529, 80-2.

Kuttila S. 2003. Secondary aural symptoms in relation to cranio-cervical and general disorders, University of Turku.

König O., Schaette R., Kempter R. & Gross M. 2006. Course of hearing loss and occurrence of tinnitus. *Hear Res*, 221, 59-64.

Lamm K., Lamm H. & Arnold W. 1998. Effect of hyperbaric oxygen therapy in comparison to conventional or placebo therapy or no treatment in idiopathic sudden hearing loss, acoustic trauma, noise-induced hearing loss and tinnitus. A literature survey. *Adv Otorhinolaryngol*, 54, 86-99.

Landgrebe M., Zeman F., Koller M., Eberl Y., Mohr M., Reiter J., Staudinger S., Hajak G. & Langguth B. 2010. The Tinnitus Research Initiative (TRI) database: a new approach for delineation of tinnitus subtypes and generation of predictors for treatment outcome. *BMC Med Inform Decis Mak*, 10, 42.

Langguth B., Eichhammer P., Wiegand R., Marienhegen J., Maenner P., Jacob P. & Hajak G. 2003. Neuronavigated rTMS in a patient with chronic tinnitus. Effects of 4 weeks treatment. *Neuroreport*, 14, 977-80.

Le Prell C.G., Hughes L.F. & Miller J.M. 2007. Free radical scavengers vitamins A, C, and E plus magnesium reduce noise trauma. *Free Radic Biol Med*, 42, 1454-63.

Leibovici D., Gofrit O.N. & Shapira S.C. 1999. Eardrum perforation in explosion survivors: is it a marker of pulmonary blast injury? *Ann Emerg Med*, 34, 168-72.

Leibovici D., Gofrit O.N., Stein M., Shapira S.C., Noga Y., Heruti R.J. & Shemer J. 1996. Blast injuries: bus versus open-air bombings--a comparative study of injuries in survivors of open-air versus confined-space explosions. *J Trauma*, 41, 1030-5.

Liang Z.a. 1992. Parametric relation between impulse noise and auditory damage. *Noise induced hearing loss*, A. Dancer, D. Henderson, R.J. Salvi & R.P. Hamernik (eds.). St Louis: Mosby Year Books.

Lockwood A.H., Salvi R.J., Coad M.L., Towsley M.L., Wack D.S. & Murphy B.W. 1998. The functional neuroanatomy of tinnitus: evidence for limbic system links and neural plasticity. *Neurology*, 50, 114-20.

Loeb M. & Smith R.P. 1967. Relation of induced tinnitus to physical characteristics of the inducing stimuli. *J Acoust Soc Am*, 42, 453-5.

Londero A., Langguth B., De Ridder D., Bonfils P. & Lefaucheur J.P. 2006. Repetitive transcranial magnetic stimulation (rTMS): a new therapeutic approach in subjective tinnitus? *Neurophysiol Clin*, 36, 145-55.

Lu J., Cheng X., Li Y., Zeng L. & Zhao Y. 2005. Evaluation of individual susceptibility to noise-induced hearing loss in textile workers in China. *Arch Environ Occup Health*, 60, 287-94.

Lutman M.E. 2000. What is the risk of noise-induced hearing loss at 80, 85, 90 dB(A) and above? *Occup Med (Lond)*, 50, 274-5.

Man A. & Naggan L. 1981. Characteristics of tinnitus in acoustic trauma. *Audiology*, 20, 72-8.

Markou K., Lalaki P., Barbetakis N., Tsalighopoulos M.G. & Daniilidis I. 2001. The efficacy of medication on tinnitus due to acute acoustic trauma. *Scand Audiol Suppl*, 180-4.

Mattila M. & Rusanen M. 2006. Toimialan onnettomuudet. Tukes.

May J.J. 2000. Occupational hearing loss. *Am J Ind Med*, 37, 112-20.

Mazurek B., Olze H., Haupt H. & Szczepek A.J. 2010. The more the worse: the grade of noise-induced hearing loss associates with the severity of tinnitus. *Int J Environ Res Public Health*, 7, 3071-9.

McKee G.J. & Stephens S.D. 1992. An investigation of normally hearing subjects with tinnitus. *Audiology*, 31, 313-7.

McShane D.P., Hyde M.L. & Alberti P.W. 1988. Tinnitus prevalence in industrial hearing loss compensation claimants. *Clin Otolaryngol*, 13, 323-30.

Meikle M. & Taylor-Walsh E. 1984. Characteristics of tinnitus and related observations in over 1800 tinnitus clinic patients. *J Laryngol Otol Suppl*, 9, 17-21.

Meikle M.B., Vernon J. & Johnson R.M. 1984. The perceived severity of tinnitus. Some observations concerning a large population of tinnitus clinic patients. *Otolaryngol Head Neck Surg*, 92, 689-96.

Meikle M.B., Creedon T.A. & Griest S.E. 2004. Tinnitus Archive, second edition. http://www.TinnitusArchive.org.

Melinek M., Naggan L. & Altman M. 1976. Acute acoustic trauma - a clinical investigation and prognosis in 433 symptomatic soldiers. *Isr J Med Sci*, 12, 560-9.

Meric C., Gartner M., Collet L. & Chery-Croze S. 1998. Psychopathological profile of tinnitus sufferers: evidence concerning the relationship between tinnitus features and impact on life. *Audiol Neurootol*, 3, 240-52.

Meyer J.D., Chen Y., McDonald J.C. & Cherry N.M. 2002. Surveillance for work-related hearing loss in the UK: OSSA and OPRA 1997-2000. *Occup Med (Lond)*, 52, 75-9.

Miller I.S., McGahey D. & Law K. 2002. The otologic consequences of the Omagh bomb disaster. *Otolaryngol Head Neck Surg*, 126, 127-8.

Mirz F., Gjedde A., Ishizu K. & Pedersen C.B. 2000. Cortical networks subserving the perception of tinnitus--a PET study. *Acta Otolaryngol Suppl*, 543, 241-3.

Moon I.S. 2007. Noise-induced hearing loss caused by gunshot in South Korean military service. *Mil Med*, 172, 421-5.

Mühlnickel W., Elbert T., Taub E. & Flor H. 1998. Reorganization of auditory cortex in tinnitus. *Proc Natl Acad Sci U S A*, 95, 10340-3.

Mäkitie I. & Pihlajamäki H. 2006. Fatal explosion injuries in Finland: a twenty-year nationwide survey. *Scand J Surg*, 95, 180-4.

Mäkitie I., Paloneva H. & Tikka S. 1997. Explosion injuries in Finland 1991-1995. *Ann Chir Gynaecol*, 86, 209-13.

Möller A.R. 2000. Similarities between severe tinnitus and chronic pain. *J Am Acad Audiol*, 11, 115-24.

Nageris B.I., Attias J. & Raveh E. 2010. Test-retest tinnitus characteristics in patients with noise-induced hearing loss. *Am J Otolaryngol*, 31, 181-4.

Nageris B.I., Raveh E., Zilberberg M. & Attias J. 2007. Asymmetry in noise-induced hearing loss: relevance of acoustic reflex and left or right handedness. *Otol Neurotol*, 28, 434-7.

Neuberger M., Korpert K., Raber A., Schwetz F. & Bauer P. 1992. Hearing loss from industrial noise, head injury and ear disease. A multivariate analysis on audiometric examinations of 110,647 workers. *Audiology*, 31, 45-57.

Newman C.W., Jacobson G.P. & Spitzer J.B. 1996. Development of the Tinnitus Handicap Inventory. *Arch Otolaryngol Head Neck Surg*, 122, 143-8.

Newman C.W., Sandridge S.A. & Jacobson G.P. 1998. Psychometric adequacy of the Tinnitus Handicap Inventory (THI) for evaluating treatment outcome. *J Am Acad Audiol*, 9, 153-60.

Newman C.W., Wharton J.A., Shivapuja B.G. & Jacobson G.P. 1994. Relationships among psychoacoustic judgments, speech understanding ability and self-perceived handicap in tinnitus subjects. *Audiology*, 33, 47-60.

Nicolas-Puel C., Akbaraly T., Lloyd R., Berr C., Uziel A., Rebillard G. & Puel J.L. 2006. Characteristics of tinnitus in a population of 555 patients: specificities of tinnitus induced by noise trauma. *Int Tinnitus J*, 12, 64-70.

Nondahl D.M., Cruickshanks K.J., Wiley T.L., Klein R., Klein B.E. & Tweed T.S. 2002. Prevalence and 5-year incidence of tinnitus among older adults: the epidemiology of hearing loss study. *J Am Acad Audiol*, 13, 323-31.

Nondahl D.M., Cruickshanks K.J., Dalton D.S., Klein B.E., Klein R., Tweed T.S. & Wiley T.L. 2006. The use of hearing protection devices by older adults during recreational noise exposure. *Noise Health*, 8, 147-53.

Norena A.J. & Eggermont J.J. 2003. Changes in spontaneous neural activity immediately after an acoustic trauma: implications for neural correlates of tinnitus. *Hear Res*, 183, 137-53.

Ojanen O.-P. & Vainio T. 2003. Räjähdys kauppakeskus Myyrmannissa: sisäasiainministeriön asettaman tutkijaryhmän raportti. Sisäasiainministeriö.

Okamoto H., Stracke H., Stoll W. & Pantev C. 2011. Listening to tailor-made notched music reduces tinnitus loudness and tinnitus-related auditory cortex activity. *Proc Natl Acad Sci U S A*, 107, 1207-10.

Oliveira C.A., Venosa A. & Araujo M.F. 1999. Tinnitus program at Brasilia University Medical School. *Int Tinnitus J*, 5, 141-3.

Pahor A.L. 1981. The ENT problems following the Birmingham bombings. *J Laryngol Otol*, 95, 399-406.

Palmer K.T., Griffin M.J., Syddall H.E., Davis A., Pannett B. & Coggon D. 2002. Occupational exposure to noise and the attributable burden of hearing difficulties in Great Britain. *Occup Environ Med*, 59, 634-9.

Patterson M.B. & Balough B.J. 2006. Review of pharmacological therapy for tinnitus. *Int Tinnitus J*, 12, 149-59.

Perkins C., Hamernik R.P. & Henderson D. 1975. The effect of interstimulus interval on the production of hearing loss from impulse noise. *J Acoust Soc Am Suppl*, 57, S62.

Phillips J.S. & McFerran D. 2010. Tinnitus Retraining Therapy (TRT) for tinnitus. *Cochrane Database Syst Rev*, CD007330.

Phoon W.H., Lee H.S. & Chia S.E. 1993. Tinnitus in noise-exposed workers. *Occup Med (Lond)*, 43, 35-8.

Pirilä T. 1991. Left-right asymmetry in the human response to experimental noise exposure. I. Interaural correlation of the temporary threshold shift at 4 kHz frequency. *Acta Otolaryngol*, 111, 677-83.

Pirilä T. 1991. Left-right asymmetry in the human response to experimental noise exposure. II. Pre-exposure hearing threshold and temporary threshold shift at 4 kHz frequency. *Acta Otolaryngol*, 111, 861-6.

Pirilä T., Jounio-Ervasti K. & Sorri M. 1991. Hearing asymmetry among left-handed and right-handed persons in a random population. *Scand Audiol*, 20, 223-6.

Pirilä T., Sorri M., Jounio-Ervasti K., Sipilä P. & Karjalainen H. 1991. Hearing asymmetry among occupationally noise-exposed men and women under 60 years of age. *Scand Audiol*, 20, 217-22.

Plewnia C., Bartels M. & Gerloff C. 2003. Transient suppression of tinnitus by transcranial magnetic stimulation. *Ann Neurol*, 53, 263-6.

Podoshin L., Ben-David J. & Teszler C.B. 1997. Pediatric and Geriatric Tinnitus. *Int Tinnitus J*, 3, 101-103.

Price G.R. 1983. Relative hazard of weapons impulses. *J Acoust Soc Am*, 73, 556-66.

Price G.R. 1986. Hazard from intense low-frequency acoustic impulses. *J Acoust Soc Am*, 80, 1076-86.

Price G.R., Kim H.N., Lim D.J. & Dunn D. 1989. Hazard from weapons impulses: histological and electrophysiological evidence. *J Acoust Soc Am*, 85, 1245-54.

Puolustusvoimat. 1979. Varomääräys kuulon suojaamiseksi ampumaharjoituksissa. 1686/Dbcb PEkoul-os PMK D 39., Pääesikunta/koulutusosasto/ohjesääntötoimisto.

Puolustusvoimat. 1985. Kuulonsuojelu taisteluharjoituksissa. 3153/Dbc 18.11.1985., Pääesikunta/koulutusosasto/ohjesääntötoimisto.

Puolustusvoimat. 1989. Kuulon suojaaminen. 869/Dbcb PE-koul-os PMK D 1.6., Pääesikunta/koulutusosasto/ohjesääntötoimisto.

Pääkkönen R. 1988. Low frequency high level noise impulses near weapons and explosions. *J Low Freq Noise Vibr*, 7, 42-49.

Rauschecker J.P., Leaver A.M. & Muhlau M. 2010. Tuning out the noise: limbic-auditory interactions in tinnitus. *Neuron*, 66, 819-26.

Richmond D.R., Yelverton J.T., Fletcher E.R. & Phillips Y.Y. 1989. Physical correlates of eardrum rupture. *Ann Otol Rhinol Laryngol Suppl*, 140, 35-41.

Ries P.W. 1994. Prevalence and characteristics of persons with hearing trouble: United States, 1990-91. *Vital Health Stat 10*, 1-75.

Riihikangas P., Anttonen H., Hassi J. & Sorri M. 1980. Hearing loss and impulse noise during military service. *Scand Audiol Suppl*, 292-7.

Risey J., Briner W., Guth P.S. & Norris C.H. 1989. The superiority of the Goodwin procedure over the traditional procedure in measuring the loudness level of tinnitus. *Ear Hear*, 10, 318-22.

Roberto M., Hamernik R.P. & Turrentine G.A. 1989. Damage of the auditory system associated with acute blast trauma. *Ann Otol Rhinol Laryngol Suppl*, 140, 23-34.

Roberto M., Ahroon W.A., Hamernik R.P., Davis R.I. & Case C.J. 1994. The effects of primed and interrupted noise exposure paradigms on hearing loss. *J Acoust Soc Am*, 95, 3005.

Rosenberg S.I., Silverstein H., Rowan P.T. & Olds M.J. 1998. Effect of melatonin on tinnitus. *Laryngoscope*, 108, 305-10.

Rosenhall U. 2003. The influence of ageing on noise-induced hearing loss. *Noise Health*, 5, 47-53.

Rosenhall U. & Karlsson A.K. 1991. Tinnitus in old age. *Scand Audiol*, 20, 165-71.

Rubak T., Kock S., Koefoed-Nielsen B., Lund S.P., Bonde J.P. & Kolstad H.A. 2008. The risk of tinnitus following occupational noise exposure in workers with hearing loss or normal hearing. *Int J Audiol*, 47, 109-14.

Rusanen M. & Laanti T. 2003. Turvatekniikan keskuksen tietoon tulleet onnettomuus- ja vaaratilanteet vuonna 2002. Tukes.

Rösler G. 1994. Progression of hearing loss caused by occupational noise. *Scandinavian Audiology*, 23, 13-37.

Sakata E., Ito Y. & Itoh A. 1997. Clinical Experiences of Steroid Targeting Therapy to Inner Ear for Control of Tinnitus. *Int Tinnitus J*, 3, 117-121.

Sakata T., Esaki Y., Yamano T., Sueta N. & Nakagawa T. 2008. A comparison between the feeling of ear fullness and tinnitus in acute sensorineural hearing loss. *Int J Audiol*, 47, 134-40.

Salmivalli A. 1967. Acoustic trauma in regular army personnel. Clinical audiologic study. *Acta Otolaryngol*, Suppl 222:1-85.

Sardesai M.G., Tan A.K. & Fitzpatrick M. 2003. Noise-induced hearing loss in snorers and their bed partners. *J Otolaryngol*, 32, 141-5.

Saunders J.C., Cohen Y.E. & Szymko Y.M. 1991. The structural and functional consequences of acoustic injury in the cochlea and peripheral auditory system: a five year update. *J Acoust Soc Am*, 90, 136-46.

Savolainen S. & Lehtomäki K.M. 1996. Hearing protection in acute acoustic trauma in Finnish conscripts. *Scand Audiol*, 25, 53-8.

Savolainen S. & Lehtomäki K.M. 1997. Impulse noise and acute acoustic trauma in Finnish conscripts. Number of shots fired and safe distances. *Scand Audiol*, 26, 122-6.

Savolainen S. & Lehtomäki K. 2005. Acute acoustic trauma in the Finnish Defence Forces in 2003. *Ann Med Milit Fenn*, 80, 29-33.

Savolainen S., Pääkkönen R., Jokitulppo J., Toivonen M. & Lehtomaki K. 2008. Nuorten miesten kuulo ja korvien oireilu varusmiespalvelukseen astuttaessa. *SLL*, 63, 1935-1939.

Schlee W., Mueller N., Hartmann T., Keil J., Lorenz I. & Weisz N. 2009. Mapping cortical hubs in tinnitus. *BMC Biol*, 7, 80.

Shim H.J., Kim S.K., Park C.H., Lee S.H., Yoon S.W., Ki A.R., Chung D.H. & Yeo S.G. 2009. Hearing abilities at ultra-high frequency in patients with tinnitus. *Clin Exp Otorhinolaryngol*, 2, 169-74.

Shulman A. & Goldstein B. 2006. Pharmacotherapy for severe, disabling, subjective, idiopathic tinnitus: 2005-2006. *Int Tinnitus J*, 12, 161-71.

Smith P.A., Davis A., Ferguson M. & Lutman M.E. 2000. The prevalence and type of social noise exposure in young adults in England. *Noise Health*, 2, 41-56.

Smoorenburg G. 1992. Damage risk for low-frequency impulse noise: the spectral factor in noise-induced hearing loss. *Noise-induced hearing loss*, A. Dancer, D. Henderson, R.J. Salvi & R.P. Hamernik (eds.). New York: Mosby Year Book, 313-324.

Spoendlin H. 1971. Primary structural changes in the organ of Corti after acoustic overstimulation. *Acta Otolaryngol*, 71, 166-76.

Spoendlin H. & Brun J.P. 1973. Relation of structural damage to exposure time and intensity in acoustic trauma. *Acta Otolaryngol*, 75, 220-6.

Starck J., Toppila E. & Pyykkö I. 1999. Smoking as a risk factor in sensory neural hearing loss among workers exposed to occupational noise. *Acta Otolaryngol*, 119, 302-5.

Starck J., Toppila E. & Pyykkö I. 2003. Impulse noise and risk criteria. *Noise Health*, 5, 63-73.

Stephens S. 1987. Historical aspects of tinnitus. *In: Tinnitus*, J. Hazell Ed. London: Churchill Livingstone, 1-19.

STM. 2009. Sosiaali- ja terveysministeriön asetus tapaturmavakuutuslaissa tarkoitetusta haittaluokituksesta. *Säädöskokoelma N:o 1649.*

Stouffer J.L. & Tyler R.S. 1990. Characterization of tinnitus by tinnitus patients. *J Speech Hear Disord*, 55, 439-53.

Sutbas A., Yetiser S., Satar B., Akcam T., Karahatay S. & Saglam K. 2007. Low-cholesterol diet and antilipid therapy in managing tinnitus and hearing loss in patients with noise-induced hearing loss and hyperlipidemia. *Int Tinnitus J*, 13, 143-9.

Söderman A.C., Bagger-Sjöbäck D., Bergenius J. & Langius A. 2002. Factors influencing quality of life in patients with Meniere's disease, identified by a multidimensional approach. *Otol Neurotol*, 23, 941-8.

Tambs K., Hoffman H.J., Borchgrevink H.M., Holmen J. & Samuelsen S.O. 2003. Hearing loss induced by noise, ear infections, and head injuries: results from the Nord-Trondelag Hearing Loss Study. *Int J Audiol*, 42, 89-105.

Temmel A.F., Kierner A.C., Steurer M., Riedl S. & Innitzer J. 1999. Hearing loss and tinnitus in acute acoustic trauma. *Wien Klin Wochenschr*, 111, 891-3.

Toivonen M., Pääkkönen R., Savolainen S. & Lehtomäki K. 2002. Noise exposure at combat excersises. *Rev Inter Mil Med*, 76, 117-120.

Tonndorf J. 1981. Stereociliary dysfunction, a case of sensory hearing loss, recruitment, poor speech discrimination and tinnitus. *Acta Otolaryngol*, 91, 469-79.

Toppila E., Pyykkö I. & Starck J. 2001. Age and noise-induced hearing loss. *Scand Audiol*, 30, 236-44.

Toppila E., Pyykkö I.I., Starck J., Kaksonen R. & Ishizaki H. 2000. Individual Risk Factors in the Development of Noise-Induced Hearing Loss. *Noise Health*, 2, 59-70.

Touma J.B. 1992. Controversies in noise-induced hearing loss (NIHL). *Ann Occup Hyg*, 36, 199-209.

Tschopp K. & Probst R. 1989. Acute acoustic trauma. A retrospective study of influencing factors and different therapies in 268 patients. *Acta Otolaryngol*, 108, 378-84.

Tyler R.S. 1993. Tinnitus disability and handicap questionnaires. *Seminars in hearing*, 14, 377-383.

Tyler R.S. & Baker L.J. 1983. Difficulties experienced by tinnitus sufferers. *J Speech Hear Disord*, 48, 150-4.

Tyler R.S., Haskell G.B., Gogel S.A. & Gehringer A.K. 2008. Establishing a tinnitus clinic in your practice. *Am J Audiol*, 17, 25-37.

Walsh R.M., Pracy J.P., Huggon A.M. & Gleeson M.J. 1995. Bomb blast injuries to the ear: the London Bridge incident series. *J Accid Emerg Med*, 12, 194-8.

Vavrina J. & Muller W. 1995. Therapeutic effect of hyperbaric oxygenation in acute acoustic trauma. *Rev Laryngol Otol Rhinol (Bord)*, 116, 377-80.

Vernon J. 1995. Is the claimed tinnitus real and is the claimed cause correct. *Proceedings of the Fifth International Tinnitus Seminar*, American Tinnitus Association, Portland OR USA.

Wightman J.M. & Gladish S.L. 2001. Explosions and blast injuries. *Ann Emerg Med*, 37, 664-78.

Wilson P.H. & Henry J.L. 1998. Tinnitus Cognitions Questionnaire: Development and Psychometric Properties of a Measure of Dysfunctional Cognitions Associated with Tinnitus. *Int Tinnitus J*, 4, 23-30.

Wilson P.H., Henry J., Bowen M. & Haralambous G. 1991. Tinnitus reaction questionnaire: psychometric properties of a measure of distress associated with tinnitus. *J Speech Hear Res*, 34, 197-201.

Ylikoski J. 1986. Screening audiometry of Finnish conscripts, preliminary results. *Ann Med Milit Fenn*, 61, 141-144.

Ylikoski J. & Pääkkönen R. 1986. Hazardous impulse noise from firearms: physical characteristics. *Ann Med Milit Fenn*, 61, 176-180.

Ylikoski J. & Tuominen J. 1988. Varusmiesten kuulovammat ja kuulonsuojelu. *SLL*, 43, 481-487.

Ylikoski J., Pekkarinen J. & Starck J. 1987. The efficiency of earmuffs against impulse noise from firearms. *Scand Audiol*, 16, 85-8.

Ylikoski M.E. & Ylikoski J.S. 1994. Hearing loss and handicap of professional soldiers exposed to gunfire noise. *Scand J Work Environ Health*, 20, 93-100.

14 Original publications