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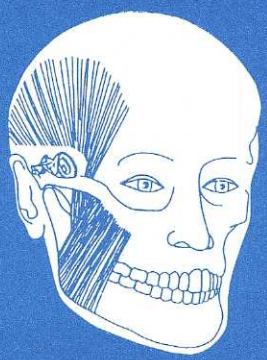
TINNITUS AND CRANIOMANDIBULAR DISORDERS



IS THERE A LINK?

BARBARA RUBINSTEIN

GÖTEBORG 1993

TINNITUS AND CRANIOMANDIBULAR DISORDERS - IS THERE A LINK?

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I Rubinstein B, Carlsson GE. Effects of stomatognathic treatment on tinnitus: a retrospective study. *Journal of Craniomandibular Practice*, 1987, 5, 254-259.

II Rubinstein B, Axelsson A, Carlsson GE. Prevalence of signs and symptoms of craniomandibular disorders in tinnitus patients. *Journal of Craniomandibular Disorders: Facial & Oral Pain*, 1990, 4, 186-192.

III Rubinstein B, Erlandsson SI. A stomatognathic analysis of patients with disabling tinnitus and craniomandibular disorders (CMD). *British Journal of Audiology*, 1991, 25, 77-83.

IV Erlandsson SI, Rubinstein B, Carlsson SG. Tinnitus: evaluation of biofeedback and stomatognathic treatment. *British Journal of Audiology*, 1991, 25, 151-161.

V Rubinstein B, Österberg T, Rosenhall U. Longitudinal fluctuations in tinnitus as reported by an elderly population. *The Journal of Audiological Medicine*, 1992, 1, 149-155.

VI Rubinstein B, Österberg T, Rosenhall U, Johansson U. Tinnitus and craniomandibular disorders in an elderly population. *The Journal of Audiological Medicine*, (1993, accepted for publication).

ABSTRACT

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by

Barbara Rubinstein

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Associations between tinnitus and craniomandibular disorders (CMD) were investigated in an epidemiological sample, in tinnitus patients, and in patients attending a 'CMD-clinic'. Natural course of tinnitus was explored in a longitudinal epidemiological study of an elderly population.

Several findings indicating a relatively strong relationship between CMD, tinnitus and subjective hearing loss were noted. This relationship seemed to be independent of objectively assessed degree of hearing loss, occupational noise exposure, general morbidity, medication or socioeconomic status. The prevalence of frequent headaches and fatigue or tenderness in jaw muscles was higher in tinnitus patients than would be expected if these conditions were unrelated. About one third of the individuals affected by tinnitus reported influence on tinnitus by jaw movements or pressure on the temporomandibular joint (TMJ). Diurnal bruxism and jaw fatigue appeared to be related to fluctuating tinnitus, vertigo, and hyperacusis.

Stomatognathic and biofeedback treatment seemed to be able to reduce or eliminate tinnitus in some patients. Relatively low severity of tinnitus, normal hearing, fluctuating tinnitus, and some signs and symptoms of CMD are believed to constitute predictors of successful treatment outcome.

Substantial longitudinal fluctuations with a high occurrence of spontaneous remissions of tinnitus were found in elderly poeple.

Key words: *biofeedback, epidemiology, headache, hearing disorders, longitudinal study, myofascial pain syndrome, stomatognathic system, temporomandibular joint syndrome, tinnitus.*

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Every kind of research is replacement of ignorance with confusion.

Mark Twain

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PREFACE

TINNITUS AND CRANIOMANDIBULAR DISORDERS - IS THERE A LINK?

This thesis is based on the following papers, which will be referred to by their Roman numerals:

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INTRODUCTION

Tinnitus is a Latin word derived from the verb *tinnire* which means to buzz, to hum, to jingle, to peal, to ring, to sound, to tinkle, or to whirr. It is an auditory symptom, commonly associated with hearing loss, with, in most cases, uncertain aetiology. It can be defined as the conscious experience of a sound that originates in the head of its owner (McFadden 1982).

Craniomandibular disorders (CMD), also called temporomandibular disorders or dysfunction (TMD), myofascial pain dysfunction (MPD), temporomandibular joint syndrome, etc, consist of musculoskeletal discomfort or dysfunction in the stomatognathic system, which is aggravated by mandibular function (Solberg 1986). The signs and symptoms of CMD are generally described as pain or tenderness in the region of temporomandibular joints or masticatory muscles, limitation or deviation of mandibular movements, joint sounds (clicking or crepitus), locking, stiffness or fatigue in the jaws. Headache has also been suggested to be part of the symptom panorama of CMD (Heiberg 1980, Magnusson 1981, Solberg 1986).

The relationship between tinnitus and CMD-symptoms is investigated in the present thesis. The attempts have also been made to find parameters which could define a CMD-related tinnitus, and to identify predictors of stomatognathic and biofeedback treatment outcome in this group of patients. In one of the papers the longitudinal fluctuations of tinnitus reports were explored.

BACKGROUND INFORMATION

TINNITUS

Historical aspects

Tinnitus has troubled humanity from time immemorial. It has been mentioned in ancient Egyptian papyri, on Assyrian clay tablets, in documents of Babylonian medicine, in the Ayur-Veda, the book of ancient Indian medicine, as well as in the Corpus Hippocraticum. Ringing in the ears is used in the Bible (Jeremiah, chapter 19, verse 3) to describe the state of being stuck with horror. In ancient Greek and Roman poetry tinnitus appeared as a symptom of passionate love, jealousy and telepathy. The historical aspects of tinnitus have been reviewed by Politzer (1907), Stephens (1984, 1987) and Feldmann (1987a, 1987b, 1991).

Epidemiology

Tinnitus is a very frequent complaint with estimates of prevalence varying from 2.3% to 44.5% according to different epidemiological studies, different definitions, and in different age-groups (Hinchcliffe 1961, Nodar 1972, MRC(IHR) 1981, Leske 1981, Davis 1983, 1989, OPCS 1983, Hale et al 1986, Mills et al 1986, Ostri et al 1986, Rosenhall et al 1987, Wänman & Agerberg 1988, Axelsson & Ringdahl 1989, Nagel & Drexel 1989, Campbell Brown 1990, Stephens et al 1990, Ostri &

Parving 1991, Rosenhall & Karlsson 1991, Parving et al 1992, Stouffer et al 1992, Wänman & Wigren 1993) with a median value in the range 15-20%.

Tinnitus is often accompanied by some degree of hearing loss but may also be present in normally hearing individuals (Jones & Knudsen 1928, Heller & Bergman 1953, Graham & Newby 1962, Nodar 1972, MRC(IHR) 1981, OPCS 1983, Kuyper et al 1987, Nagel & Drexel 1989, Stouffer & Tyler 1990). It has been proposed that tinnitus patients with so-called 'normal hearing test' in reality reveal a hearing loss located at frequencies beyond the normal testing range (Berlin & Shearer 1981, Tonndorf 1987, Shulman 1991). On the other hand in a study by Barnea et al (1990) neither auditory brainstem response (ABR) nor extended high-frequency audiometry revealed any significant differences between a group of subjects with tinnitus and normal hearing sensitivity, compared with a matched group of control subjects without tinnitus. In a study by Domènech et al (1992) five of seventy patients with subjective tinnitus were found to have normal thresholds, with regard to their age group, both with conventional and high-frequency audiometry.

Attempts have been made to find other determinants for tinnitus. In an epidemiological study in the UK age and reported occupational noise exposure appeared to be independent additive determinants for the risk of having tinnitus (MRC(IHR) 1981, Coles 1984). According to several studies tinnitus is slightly more prevalent in women than in men (Leske 1981, OPCS 1983, Coles 1984) and women are also more severely annoyed by tinnitus (Elithorn 1953, Leske 1981, Davis 1983, Coles 1984, Axelsson & Ringdahl 1989, Stouffer & Tyler 1990). The prevalence of tinnitus has also been reported higher with lower socio-economic status (MRC(IHR) 1981, Davis 1983, Coles 1984, Campbell Brown 1990) and with higher general levels of morbidity (OPCS 1983, Hale et al 1986, Campbell Brown 1990).

In spite of extensive investigations and examinations of a large population, Coles (1984) found it very difficult to find repeatable pathological correlates for tinnitus in the general population. Furthermore, no pathomorphological correlates for tinnitus could be established in histological studies of temporal bones (Oliveira et al 1990, Reisser & Schuknecht, 1991). In a demographic study of older Americans (Campbell Brown 1990) hearing- and health-related variables were found to be more associated with tinnitus than demographic and socionomic variables were. In many cases controlling for hearing and health status reversed the effects of the demographic and socioeconomic variables. Davis et al (1992) found that, after hearing impairment, reported hearing disability and history of possible ear pathology are the two most pervasive factors that influence prevalence of prolonged spontaneous tinnitus (PST). Signs of depression, high blood pressure, and present drug intake were, in the same study, found to be associated with bilateral PST. A higher incidence of cobolamine deficiency in subjects with chronic tinnitus and noise

induced hearing loss (NIHL) when compared with subjects without tinnitus was found by Shemesh et al (1992).

Characteristics

The term tinnitus is used to describe the sensation of any sound which appears to arise in the head or ears of the affected person. Tinnitus has many different manifestations. The character and intensity of the sound vary considerably both inter- and intraindividually. The noise may appear to come from one ear, both ears or elsewhere in the head. Occasionally, it may be perceived as if coming from outside the head. The onset may be gradual or sudden. The loudness of tinnitus may vary. Often, it is not one noise, but a number of noises, simple or compound in nature. They may be described by a wide range of verbal labels. The sounds may be high- or low-pitched, loud or soft, continuous or intermittent, constant, pulsatile, or fluctuating in intensity and pitch, increasing or diminishing in severity, and they may disappear completely. They may be different in each ear, and may change in character and in quality from day to day and even from hour to hour. The tinnitus may be insignificant and only discernible when one listens for it or it can be extremely severe, persistent, and intolerable. It can result in various degrees of discomfort, from mild awareness to reduced work capacity, total disability, suicidal desires, and even suicide (Tyler & Baker 1983, Lewis et al 1992). Tinnitus may be associated with considerable psychological distress such as anxiety, depression, irritability, and sleep disturbance (House 1981, Halford & Anderson 1991, Wilson et al 1991).

Tinnitus seems to affect the left ear more commonly than the right (MRC(IHR) 1981, Coles 1984, Axelsson & Ringdahl 1989), with the difference most pronounced in clinical reports (Reed 1960, Meikle & Taylor-Walsh 1984, Small et al 1986, Meikle & Griest 1987, Stouffer & Tyler 1990). In clinical studies approximately half of individuals affected by tinnitus localize the noise to one ear, while the other half identify it as coming from both ears or the head in general (Reed 1960, Meikle & Taylor-Walsh 1984, Small et al 1986, Claussen & Claussen 1987, Schönweiler 1987, Chole & Parker 1992).

In the majority of tinnitus patients the loudness of tinnitus is fluctuating (Meikle et al 1987, Erlandsson et al 1992), and can be altered, most often exacerbated, by exposure to loud sound, nervous tension, increased awareness, fatigue and various chemicals, like drugs, alcohol, caffeine and tobacco, while relaxation and diminished awareness make it less disturbing (Kafka 1934, Meikle et al 1987, Stouffer et al 1991). Hallam et al (1984) have observed that patients who had lived with tinnitus for many years began to find it intolerable during periods of life-stress.

Classification and aetiology

The oldest known classification of tinnitus is the one specified on Assyrian tablets, where the different types were named: *singing*, *whispering* and *speaking* of the ears (Thompson 1931 /quoted by Stephens

1984). But there are numerous terminologies and taxonomic systems that can be found in the literature. The classifications have been made according to cause, structure involved, location, quality, audiometric features, presumptive mechanism, masking pattern, psychological impact, etc.

Tinnitus has, however, traditionally been classified into two general types: *subjective* (real, static, nonvibratory, true, intrinsic) and *objective* (false, dynamic, vibratory, pseudo, extrinsic). Subjective tinnitus is perceived by the patient only, and objective tinnitus is audible to the patient and/or an observer (Du Verney 1683 /quoted by Feldmann 1987c).

A number of pathological states are known which produce enough mechanical energy to stimulate the auditory system, resulting in the perception of sound (vibratory tinnitus). If these entities produce enough mechanical energy to be heard by others, they become objective, if not, they remain subjective (Meyerhoff & Cooper 1980). The division is thus limited by the diagnostic ability of the examiner (Williamson & Amadee 1990).

Objective tinnitus is rare. According to Longridge (1979), it is present in about one per cent of patients who have tinnitus as a main complaint. It is usually either vascular or muscular in origin. It may be caused by vascular abnormalities within the arteries of the head and neck or it may be due to an anomaly of the venous system of the base of the skull (Du Verney 1683, Hentzer 1968, Glanville et al 1971, Trujillo et al 1990). Clonic muscle contractions of the tensor tympani, stapedius, tensor veli palatini, levator veli palatini or salpingopharyngeal muscles may result in a clicking sound which can be heard by the patient and examiner alike (Schwartze 1867, Smith 1943, Leventon et al 1968, Quarry 1972, Pulec et al 1978, Virtanen 1983, Toland et al 1984, Slack et al 1986, Le Pajolec 1990). Acoustic energy generated within the cochlea, termed spontaneous otoacoustic emissions (SOAE) were thought to be related to tinnitus (Kemp 1981, Penner 1988). However, Zwicker (1987) noted that the source of these emissions seemed to be a healthy cochlea and he could not find any correlation between SOAE and tinnitus. The current opinion is that SOAE may be responsible for tinnitus in a very small percentage of tinnitus cases (Penner 1990).

There is no single explanation for the aetiology of *subjective* tinnitus. It has been considered a symptom which results from a great number of causes, in very many conditions, which may or may not be primarily concerned with the hearing organ itself. Practically every known disease and pathological condition, from impacted earwax to sexual over-indulgence, has been mentioned in the literature as a suggested cause of tinnitus (Kafka 1934, Seltzer 1947). In the dental field unerupted molars, periapical infections, carious teeth, as well as amalgam fillings, have been blamed for causing tinnitus (Barr 1909, Harris 1922, Tousey 1922, Mollison 1926, Kafka 1934, Uhr & Uhr 1986).

There are many different kinds of tinnitus and attempts have been made to relate specific kinds of tinnitus to specific causes. In general, it has

been found that the central pitch of the tinnitus, and verbal descriptions of the noises and their severity, do not correlate with diagnosis or probable site of the lesion, although tinnitus associated with conductive hearing loss or Ménière's disease in the active phase tends to be low-pitched and is usually described as a 'roaring sound' (Reed 1960, Graham & Newby 1962, Nodar & Graham 1965, Vernon et al 1980).

The pathophysiology of tinnitus is as yet not well-understood. Many ingenious hypotheses describing the origin of tinnitus and the underlying mechanisms responsible for tinnitus have been proposed, many of them in very early papers cited by more recent authors.

Rivinus in 1717 and Cotugno in 1760 postulated that convulsive contractions of the middle ear muscles and the tympanic membrane could produce tinnitus (Politzer 1907, Feldmann 1987b).

Lothrop (1923) believed that involuntary contractions of the tensor tympani and stapedius muscles were responsible for tinnitus. He held that disruption of the normal balance of the middle ear structures might cause tinnitus, that the tensor tympani and stapedius muscles continously try to modify any displacement through involuntary contraction, and when they fail and normal balance is destroyed, the tinnitus supervenes. He proposed a theory of abnormal position of the middle ear structures exerting abnormal pressure on the intralabyrinthine fluid. In trying to re-establish an equilibrium, the middle ear muscles are brought to vibrate by pulling against the ever contracting adhesions and the unbalanced and dislocated ossicular chain, and the vibration is conveyed to the labyrinthine fluid, causing the subjective symptom of tinnitus.

Fowler (1941) and Atkinson (1944, 1947) regarded tinnitus as an auditory paraesthesia in which the nerve endings of the cochlea are stimulated in the same manner as those in other parts of the body during the sensation of itching. Fowler (1948a) postulated that anoxia, due to or exaggerated by hydrops of the labyrinth, anaemia, cardiovascular disease, inflammation, toxines, hematological, endocrine, nutritional, emotional or metabolic disorders, is a fundamental cause of nonvibratory tinnitus. Fowler & Fowler (1950) proposed that psychoneurovascular factors cause tinnitus by blood sludging resulting from emotional episodes.

Fowler (1948b) and Heller & Bergman (1953) suggested that complaints of head noises are not necessarily symptoms of pathology but may simply reflect attention to ubiquitous body signals of no general significance.

Both increased spontaneous activity of the VIIIth nerve, as well as destruction of hair cells with resulting lack of nerve activity have been considered to result in tinnitus.

The current hypotheses describing the origin of sensorineural tinnitus have been recently reviewed by Jastreboff (1990). They include partial mechanical decoupling of hair cells (sterocilia) from the tectorial membrane with increase of the thermal noise of the system and cochlear nerve deafferentiation, occurrence of 'cross-talk' between demyelinated audi-

tory nerve fibres or hair cells, caused by a breakdown of electrical insulation between them, and changes in calcium ion concentration in the perilymph as well as within hair cells.

Animal models have been developed, using salicylates in doses that produce blood concentrations evoking tinnitus in humans, for some forms of tinnitus (Sasaki et al 1981, Kauer et al 1982, Evans 1983, Schreiner & Snyder 1987). Jastreboff (1987, 1990) developed an animal model of tinnitus using both behavioural and electrophysiological approaches.

An analogy between tinnitus and abnormalities of other special senses have been discussed by Du Verney (1683) and by Williams (1928). Several studies have pointed to an analogy between chronic tinnitus in the auditory system and chronic intractable pain in the somato-sensory system, and tinnitus has been considered the equivalent of pain. Tonndorf (1987) proposed a physiological basis for chronic tinnitus deriving from deafferentiation of the sensory nerve fibres, such as has been found with pain. Jastreboff (1990) described tinnitus as a phantom auditory perception. Goebel et al (1991, 1992) analyzed the medical, behavioural, cognitive, emotional, psychosocial, and psychotherapeutic aspects of tinnitus and pain, and proposed a multifactorial psychophysiological model of tinnitus with a concept similar to those proposed for chronic pain. Melzack (1992), in an article published in 'Scientific American' discussed the phenomenon of phantom limbs, phantom seeing, and phantom hearing = tinnitus. He suggested existence of a cerebral neuromatrix, involving the somatosensory system, the limbic system, and the cognitive system, and held that in the absence of input from a sensory organ, as in hearing loss, cells in the central nervous system become more active, and the brain's intrinsic mechanisms may transform that neuronal activity into experiences like tinnitus.

There is clearly a variety of different conceivable explanations regarding the cause of tinnitus and the mechanisms of its generation but most of them are speculative and none of the hypotheses has been proven yet. As Jastreboff wrote 1990: *'There is consensus that tinnitus is the result of aberrant neural activity within the auditory pathways and that such activity is erroneously interpreted as sound by auditory centres, but the agreement ends here'.*

However, tinnitus deserves careful evaluation because it may represent the earliest indication of a serious disease process. Unilateral tinnitus may be the only sign of a cerebellopontine angle tumour (e. g. acoustic neurinoma, meningeoma etc) or a glomus tumour of the middle ear, while bilateral tinnitus, in very rare cases, may be caused by intracranial processes, that cause increased intracranial pressure.

Measurement

Psychoacoustic methods, matching and masking, neuromagnetic measurements, self-recording, and psychometric methods may be employed for the assessment of tinnitus.

Tinnitus *matching* is performed by presenting pure tones, narrow or wide band noise over a sweep frequency audiometer. The patient identifies the components of the presented sound which seem to be identical to his tinnitus or portions of his tinnitus. The frequency and loudness of the patient's tinnitus is determined (Fowler 1940, Reed 1960, Nodar & Graham 1965). The value of these measurements has, however been questioned because the sensation level of the tinnitus, the level of loudness of the tinnitus above the patient's threshold of hearing, seldom exceeds 15 dB, and is often less than 5 dB, and because the sensation level of tinnitus shows poor correlation with the distress and the subjective tinnitus grading (Fowler 1943, Vernon et al 1977, Jackson 1983, Axelsson & Sandh 1985, Hulshof & Vermeij 1986).

Masking is performed by presenting test tones at several frequencies to the patient. The intensity of these tones is raised until the patient indicates that his own tinnitus has been covered up (Josephson 1931). Often after the masking noise is turned off the tinnitus will remain inaudible for a short period of time, a phenomenon known as *residual inhibition* (Urbantschitsch 1883 /quoted by Feldmann 1987d/, Feldmann 1971, Vernon 1977).

Hoke et al (1989) have shown that the waveform of the *auditory evoked magnetic field (AEF)* in patients suffering from tinnitus differs in a very characteristic way from that in normal-hearing individuals without tinnitus. However, other investigations have failed to support these findings (Jacobson et al 1992, Colding-Jørgensen et al 1992).

A variety of *tinnitus specific psychometric scales*, recently reviewed by Erlandsson (1992) have been developed in the last few years, with the measured factors being: sleep disturbance, emotional distress, audiological perceptual difficulties, intrusiveness, hearing ability, use of medication, interference with work, avoidance, perceived attitudes, social support, and disability/handicap.

Treatment

The treatment of tinnitus of an identifiable aetiology is directed toward the underlying cause. However, the correctable causes account for a very small percentage of the tinnitus cases and mostly the nonsevere or nondesperate cases (Vernon 1977). The treatment of tinnitus of obscure aetiology remains less exact. Just about every treatment modality one can think of has been proposed and tried as a remedy for the relief of tinnitus. Many forms of therapy have been presented as successful, only to prove disappointing as time passed by.

The current approach to the treatment of tinnitus has two orientations. Firstly, the abolition or reduction of the symptom with specific therapy wherever and whenever possible. Secondly, the change of an uncompensated tinnitus into a compensated tinnitus by alleviation of the psychological effects arising from it. In both of these contexts, the treatments described can be reasonably divided into the following categories: *instru-*

mental, *pharmacological*, *psychophysiological* or *psychological*, *electrostimulatory*, *surgical*, and *other treatment methods*.

The instrumental treatment is palliative in its nature and may include: tinnitus *maskers*, conventional amplification by *hearing aids*, and *tinnitus instruments*.

Tinnitus *masking* therapy is based on the concept that tinnitus can be covered up by the presence of another sound. Masking for the relief of tinnitus was mentioned at least as early as in the twelfth-thirteenth centuries AD in the writings of the Salerno School (Stephens 1984). Jones & Knudsen (1928) constructed the first electric masking device while the first wearable tinnitus maskers were introduced in 1976 by The Tinnitus Clinic in Portland (Vernon et al 1977). Tinnitus maskers are devices, very similar in appearance to hearing aids, worn in or behind the ear, which incorporate a noise generator producing a masking noise. Some advocates of this therapy reported a success rate of 73-82% in patients evaluated for tinnitus masking (Pulec et al 1978, Vernon & Schleuning 1978, Longridge 1979, Hazell & Wood 1981, Small et al 1986). However, von Wedel et al (1989) reported a success rate of only 17%, and Erlandsson et al (1987) found no dramatic differences between masker and placebo treatment effects.

Saltzman & Ersner (1947) were the first to recommend the use of a conventional *hearing aid* in patients whose chief complaint is tinnitus. The purpose was to amplify the ambient noise such as effectively to mask the tinnitus. Hearing aids have been found effective in reducing or eliminating tinnitus in 50-75% of the patients fitted with them (Vernon & Schleuning 1978, Small et al 1986, von Wedel et al 1989), while Melin et al (1987) showed that hearing aids alone are not effective for reducing tinnitus, and ascribed the positive effects found in other studies to more unspecific interventions, such as counselling.

A combination device consisting of a tinnitus masker and a hearing aid, contained in the same postaural case has been developed and is known as a *tinnitus instrument* (Vernon & Schleuning 1978).

An infinite variety of *drugs* has been investigated at one time or another as possible treatments for the symptom of tinnitus. The first effective drugs to be found were *local anaesthetics*. In 1935, Bárány reported temporary relief from tinnitus after injection of local anaesthetics into the inferior nasal conchae for intranasal surgery. Subsequently, local anaesthetic agents have, by many different investigators, been reported to produce significant suppression on a short-term basis. In experimental studies of tinnitus treatment with intravenous lidocaine 57-85% of patients have reported good or partial relief (Melding et al 1978, Goodey 1981, Shea et al 1981, Wood et al 1983, Schönweiler 1987). Furthermore, several controlled trials have shown a *temporary* disappearance or amelioration of tinnitus following intravenous lidocaine injections (Martin & Colman 1980, Israel et al 1982, Majumdar et al 1983, Ueda 1992). A dose-response relation between the serum level of lidocaine and the amount of

relief from tinnitus has been shown by Perucca & Jackson (1981). Analogy with pain of central origin suggests that the beneficial effect of lidocaine may be due to its anticonvulsant action (Goodey 1981). It must be emphasized, however, that the exact mechanism whereby these anaesthetics relieve tinnitus and their actual site of action are still unknown. In a study by Duckert & Rees (1983) 30% of patients given intravenous lidocaine reported *increased* tinnitus after administration of the drug.

A number of oral drugs having *anticonvulsant*, antidysrythmic or 'membrane-stabilizing' properties have been tried (e. g. carbamazepine, amylobarbitone, tocainide, mexilitene, phenytoin sodium, sodium valproate and flecainide acetate). Various *benzodiazepines* have been tried (e. g. diazepam, oxazepam, clonazepam; Goodey 1981, 1987). All these drugs seem only occasionally to alleviate the tinnitus, often at the cost of unacceptable high incidence of side-effects (Fortnum & Coles 1991). *Tricyclic antidepressants* (e g nortriptyline) have an anticonvulsant as well as an antidepressant action and have been effective in reducing tinnitus in individual cases and often potentiate the action of anticonvulsants (Goodey 1981, Sullivan et al 1989). However, in a double-blind randomized clinical trial of nortriptyline versus placebo no significant differences in audiometric or self-report measures were found between active drug and placebo (Dobie et al 1992). *Vitamins*, primarily vitamin A (Baron 1951, Graham 1965), vitamin C (Seltzer 1947), nicotinic acid (Atkinson 1944), thiamine and niacin (Travell & Simons 1983) and vitamin B_{12}=cobolamine (Shemesh et al 1992) have been advocated for their beneficial effects on tinnitus.

This list of drugs, tested in an attempt to suppress tinnitus, is by no means complete, and many, if not all, of the mentioned drugs may actually sometimes cause or aggravate existing tinnitus (Fowler 1948b, Goodey 1981, 1987). No drug has been yet approved by the US Food and Drug Administration for the treatment of tinnitus.

As in chronic pain syndromes, *psychological* factors may have a crucial role in patients' response to tinnitus. The psychotherapeutic measures range from simple reassurance to psychoanalysis. The effort is not so much to cure the tinnitus as it is to make it less unbearable.

Tinnitus is a very common symptom in the population at large, and the vast majority of individuals with tinnitus never seek medical attention for it. Of those who do a majority is fairly satisfied with an explanation of the harmless nature of their complaint and the *reassurance* that serious, progressive disease has been ruled out, and that very little is known about the origin of tinnitus, mainly due to its benign character (Longridge 1979, Hazell & Wood 1981). Helpful advice is to use *'sound-sedation'* - music or radio at the bedside at night (Pang et al 1979).

Relaxation and *biofeedback* techniques have been used to modify the stress response, reduce the patients' adverse reaction to tinnitus and in some cases relieve the tinnitus. The rationale behind the use of relaxation and/or stress-management techniques in tinnitus is based upon the assump-

tion that tinnitus can be induced or exacerbated by stress, nervousness, or fatigue. Furthermore, tinnitus itself can be stress-producing, thereby creating a vicious cycle of stress with a consequent exacerbation of the tinnitus (Kirsch et al 1989). The principle of EMG-biofeedback training is to induce the patients to relax by registering action potentials in, for example, the frontalis muscle, and making this electrical activity visible or audible to the patient. House et al (1977) and Carmen & Svihovec (1984) reported improvement or disappearance of tinnitus in 80% of patients following biofeedback treatment, while Haralambous et al (1987) did not find any significant treatment effects, when a waiting-list control group was included in the study. Other biofeedback studies showed an improvement ranging from 14% to 60% (Grossan 1976, White et al 1986, von Wedel et al 1989).

Traditional *psychotherapeutic* and various *cognitive* and *behavioural modification techniques*, treating the patients reaction to tinnitus rather than the tinnitus itself, and altering maladaptive behaviours, have been tried (Lindberg et al 1984, 1988, Sweetow 1984a, 1986). In a study by Scott et al (1985) treatment with a combination of relaxation techniques and *perceptual restructuring* resulted in reduction of both subjective tinnitus loudness and discomfort from tinnitus, when compared to the patients in the waiting-list control group, who deteriorated.

Marlowe (1973) reported some cases of disturbing tinnitus successfully treated with *hypnotherapy*. Hypnosis has also been used in a controlled trial to relieve tinnitus in a selected group of patients (Marks et al 1985). Brattberg (1983) reported improvement in 69% of patients treated with *relaxation-hypnotherapy* through home use of recorded audio cassette. Seventy-three percent of patients using *self-hypnosis* reported disappearance of tinnitus during treatment sessions (Attias et al 1990).

A *matching to sample feedback* procedure described by Ince et al (1987) has been found to decrease the tinnitus loudness in 84% of patients, but no long-term effects were reported. Participation in *self-help groups* and *support/education groups* has been advocated by Sweetow (1984b) and Reich (1987). Positive effects on several factors of tinnitus distress have been reported in a study of 138 patients treated according to a *multimodal psychotherapy concept*, including counselling, tinnitus-diary, emotional and cognitive therapy, group therapy and psychoanalysis (Goebel et al 1991). An extensive review of both uncontrolled and controlled studies of psychophysiological and psychotherapeutic treatment of tinnitus has been presented by Goebel (1992).

Many attempts to suppress tinnitus using electrical currents have been reported. Static electricity was used in the first trials, described by Wibel (1786 /quoted by Stephens 1984). *Electrical stimulation* has since that been applied to ear and other sites, with iontophoresis, transcutaneous, transtympanic and intracochlear electrodes. The reported success rates in studies of the effects of electrical stimulation on tinnitus have been highly variable. The success rates in following studies ranged from 29% to 72%

(Hatton et al 1960, Chouard et al 1981, Latkowski et al 1983, House 1984, Graham et al 1987, Kaada et al 1989, Kuk et al 1989, von Wedel et al 1989, Okusa et al 1993). In a double-blind cross-over study by Dobie et al (1986) reduction in severity of tinnitus was reported by 10% of patients with the active device and by 20% of patients with placebo device.

The *cochlear implant* is an auditory prosthesis for the profoundly deaf with an electrical current that stimulates the auditory nerve, producing a sensation of sound (House et al 1976, House & Brackmann 1981, Fraysse 1983, Graham et al 1987). The relief of tinnitus was reported in up to 83% of cases (House 1984, Berliner et al 1987, McKerrow et al 1991).

Surgical interventions including destructions of the cochlea, the labyrinth or the auditory nerve (Lake 1909, Harris 1922, Pulec et al 1978), lobotomies (Elithorn 1953, Beard 1965), sympathectomies (Passe 1951), sectioning of the cochlear nerve (Pulec et al 1978), and microvascular decompression of the cochlear nerve (Jannetta 1987, Møller 1987) have been tried as treatments for tinnitus. Even with such serious interventions, the results are varied and generally poor, with a large number of patients actually becoming considerably worse. The effects of otological surgery on tinnitus, as reported in the literature, have been reviewed by Hazell (1990).

Other modalities of therapy which have been explored in the search for a remedy to tinnitus include *acupuncture*, *ultrasound*, *hyperbaric oxygen* therapy, and use of *leeches*. In general, such methods have had conflicting results reported. *Acupuncture* is a classic form of Chinese medicine. The theory of its mechanism is based on activation of different endogenous systems that act upon the neural pathways for pain. Mann (1974) postulated that only about 5% or less of patients with tinnitus may be cured or have more than a marginal improvement by acupuncture. Hansen et al (1982) found no effect on tinnitus of acupuncture when compared with placebo, while in a double-blind cross-over controlled trial of acupuncture as treatment for tinnitus 35% of patients described some benefit from the active treatment only (Marks et al 1984). Thomas et al (1988) reported transient effects in half of their patients but no positive long-term results. Podoshin et al (1991) reported amelioration in the level of tinnitus in 30% of patients treated with acupuncture, compared to 0% and 5% in two control groups, while Nilsson et al (1992) found no significant treatment effects, with 20% improvement and 25% deterioration rate in 51 patients treated with acupuncture.

Low-powered *ultrasound* has been shown to reduce tinnitus in 40% of patients in a controlled pilot study (Carrick et al 1986). Schumann & Fischer (1992) reported *hyperbaric oxygen* therapy being effective in 62% of their patients with severe tinnitus. Local blood-letting by *leeches* and cupping has been advocated in the pulsating forms of tinnitus (Itard 1821, Wilde 1853 /both quoted by Stephens 1984). Successful use of medicinal leeches for treatment of tinnitus caused by inner-ear affections has also recently been reported by Seleznev et al (1992).

In most of the studies mentioned only short-term results are reported, follow up reports being rare. In a questionnaire-study by Gefken & Kurth (1992) 165 patients with chronic tinnitus evaluated their own tinnitus treatment experiences. The percentage of positive experiences was generally low, with the highest figures for self-help-groups and psychotherapy.

To sum up, most of the treatment modalities mentioned do not provide a cure but rather a control of tinnitus. The number of remedies is in inverse proportion to their value, and none of the therapeutic strategies mentioned has been found to be consistently successful, being still on a trial and error basis. Some patients may respond to one or more of the approaches mentioned while others will respond to none. Babbit said 1932 about therapeutic measures for tinnitus: *'The one definite impression, both from experience and review of the literature, is that all methods advocated have had a modicum of success. Some cases were apparently cured, some cases here and there relieved for a time but more often relapsed. Often the secondary state of comfort would appear to be a sort of auto-suggestion tolerance, sometimes a fatigue reaction to treatment.'* His words have, alas, not lost much in actuality 60 years later.

CRANIOMANDIBULAR DISORDERS

'Craniomandibular disorders' (CMD) is a collective term, synonymous with the term temporomandibular disorders, being a cluster of joint and muscle disorders in the orofacial area characterized primarily by pain, joint sounds and irregular or deviating jaw function (American Academy of Craniomandibular Disorders 1990, De Boever & Carlsson 1993). Thus, CMD include various subjective symptoms and clinical signs ranging from minor, transient discomfort to severe, chronic pain and limitation of function. Recurrent headache has also been elevated to the list of major symptoms of CMD on the basis of several studies showing a clear association between CMD and headache (Agerberg & Carlsson 1977, Heiberg 1980, Magnusson 1981, Lous & Olesen 1982, Forssell 1985, Solberg 1986, Wänman 1987, Wänman & Agerberg 1987, Schokker 1989).

History

In the past, around 1920, it was claimed that loss of posterior teeth could cause damage to the temporomandibular joint (TMJ; Prentiss 1918, Summa 1918, Monson 1920). In 1934 Costen, described a combination of signs and symptoms dependent upon disturbed function of the TMJ. It included hearing impairment, stuffiness, tinnitus, dizziness, TMJ-clicking, pain within and around the ears, headache, sensations of burning in the throat, tongue, side of the nose and sinuses. According to Costen the ear symptoms were caused by overclosure of the mandible following loss of posterior teeth. He believed the overclosure caused retrodisplacement of the mandibular condyles, with resulting erosions of bone and direct and

indirect pressures on the chorda tympani and auriculotemporal nerves, the eustachian tube, and the skull base. Bite raising was the treatment recommended (Bleiker 1938).

Several clinicians have questionned the correctness of Costen's conclusions regarding aetiology and treatment (Junemann 1941, Schwartz 1955, Brookes et al 1980), and anatomic studies have demonstrated that the anatomic basis for Costen's syndrome was not acceptable, that the postglenoid process does not allow the retrodisplacement of the condyle, and that the auriculotemporal and chorda tympani nerves pass in such a position that no effect upon the nerves by the head of the condyle can be considered likely (Sicher 1948, Zimmerman 1951). Therefore the mechanism proposed by Costen has been discredited over several decades. However, a renaissance of some of Costen's ideas could be seen in a radiographic and histological study that revealed two different anatomic possibilities for irritation of the auriculotemporal nerve within the medial and posteromedial TMJ-region (Johansson et al 1990).

Schwartz (1955), renamed Costen's syndrome the 'TMJ pain-dysfunction syndrome', and described it as being caused by trauma or subconscious oral activity with resulting painful, self-perpetuating spasm of the masticatory muscles. Laskin (1969) described four cardinal symptoms of the pain-dysfunction syndrome as being: pain of unilateral origin, usually felt in the ear or preauricular area, muscle tenderness, clicking or popping noise in the TMJ, and limitation of jaw function. *Tinnitus*, impaired hearing and dizziness were considered as being *unrelated* to these symptoms. Since the studies of Schwartz and Laskin most dentists have worked with a myofascial pain dysfunction syndrome (MPD) trimmed down to these four cardinal symptoms: *pain*, *muscle tenderness*, *joint sounds*, and *limitation of jaw function.*

Epidemiology

Epidemiological studies indicate that between 28% and 88% of the adult population has one or more sign or symptom of CMD (Helkimo 1979, Carlsson 1984, Rugh & Solberg 1985, Szentpetery et al 1986, Wänman 1987, Agerberg & Bergenholtz 1989, Agerberg & Inkapööl 1990, de Boever & Steenks 1991, Mazengo & Kirveskari 1991). The features that distinguish patients requiring treatment from individuals displaying clinical abnormalities or being aware of symptoms are according to Solberg (1986) frequency, persistence, and severity of symptoms. It is, however, estimated that for approximately 5% of the total population in western countries, the condition represents a significant problem at some period in their lives (Rugh & Solberg 1985, Dworkin et al 1990, Schiffman et al 1990,). Signs and symptoms of CMD have been found to correlate with poor general health (Agerberg & Carlsson 1975), and a tendency towards more symptoms among lower socio-economic groups has been reported by Agerberg et al (1977).

Aetiology

A diversity of aetiological concepts of CMD may be found in the literature. De Boever (1979) organized them into the following five basic theories: *mechanical displacement* theory, *neuromuscular* theory, *muscle* theory, *psychophysiological* theory, and *psychological* theory

The *mechanical displacement* theory proposed that CMD are caused by the displacement of the condyle(s) because of a dental malocclusion. The *neuromuscular* theory proposed that any type of occlusal disharmony can cause parafunctional activity such as grinding or clenching the teeth, leading to incoordination and spasm of some of the muscles of mastication. The *muscle* theory claims that the primary aetiological factor lies in the muscles of mastication themselves with stress-induced muscle hyperactivity being the cause of painful spasm and muscles acting as trigger points, occlusion being unimportant. The *psychophysiological* theory maintained that masticatory muscle spasm, initiated by muscular overextension, muscular overcontraction or muscle fatigue, caused by chronic oral habits such as bruxism is the primary factor responsible for the signs and symptoms of CMD. Emotional disturbances and stress induce parafunctional habits which are viewed as tension relieving mechanisms. The oral habits may lead to occlusal interferences which in turn may act as sustaining factors on muscle and joint disorders. Finally, the proponents of the *psychological* theory suggested that CMD are psychogenic in origin without a significant associated physical disorder, and that emotional disturbances centrally initiate muscular hyperactivity.

Schwartz (1955) divided factors associated with the onset of the 'TMJ-syndrome' as follows: *predisposing* - constitutional or temperamental, *contributing* - occlusal 'abnormalities', *precipitating* - stretch, changes in proprioception, and *aggravating* - physiological or psychological.

Since the 1980s there has been a general agreement on a multifactorial aetiology of CMD, but there are different concepts regarding main contributing factors. McNeill et al (1990), in the position paper of the American Academy of Craniomandibular Disorders, divided both central and peripheral risk factors for CMD into three categories:

Predisposing factors - pathophysiological, psychological or structural processes that alter the masticatory system sufficiently to increase the risk of developing CMD. Pathophysiological processes are primarily caused by different systemic conditions (e g neurological disorders, autoimmune diseases). The psychological factors may include emotional, personality, and attitude characteristics (anxiety, somatization, depression, introversion, coping behaviour). And finally, the structural factors may be genetic, developmental or iatrogenic (skeletal and occlusal factors).

Initiating factors - related to trauma or adverse loading of the masticatory system, including that caused by oral and occlusal parafunctions.

Perpetuating factors - factors that sustain the disorder, can be any one or a combination of the predisposing or initiating factors. They can be pathophysiological, structural, behavioural, social, emotional or cognitive.

Solberg (1986) differentiated between *morphofunctional* (occlusion, bruxism) and *psychological* (anxiety, tension) factors causing temporomandibular disorders.

A somewhat different multifactorial concept of CMD-pathology presented by de Boever & Steenks (1991) include aetiological factors divided into three main groups: *neuromuscular* factors influenced by parafunctional activity, *occlusal anatomic* factors, that may be influenced by overload and TMJ-pathology, and *psychological* factors, that may be influenced by stress and possibly the female gender.

Assessment

The evaluation for CMD consists of a careful history taking and clinical examination of the stomatognathic system. The clinical examination comprises measurement of the range of motion of the mandible, function of the TMJs, palpation of the TMJs and masticatory muscles, recording of pain on movement of the mandible, recording of the contact relations between the teeth of the upper and lower jaw in different positions, and inspection of signs of occlusal and oral parafunctions. In some cases imaging of the TMJ and craniofacial structures may be necessary.

Treatment

Various therapeutic procedures, ranging from reassurance to TMJ-surgery are used for CMD. In general terms the common conservative treatment methods in CMD can be described as counselling, occlusal splints (stabilization splints and repositioning splints), occlusal equilibration, relaxation or biofeedback training, physical medicine, pharmacotherapy, TENS, acupuncture, and mandibular manipulation, as reviewed by Dahlström (1992). It is generally agreed that most CMD-patients should be treated conservatively, and he vast majority seem to benefit from such an approach.

The surgical techniques in treatment of CMD have been osteoarthrotomy, condylectomy, high condylectomy, condylotomy, eminectomy, zygomectomy, diskectomy, placement of various implants, arthroplasty, repair and repositioning of the disc (House et al 1984, Weinberg 1984), myotomies of one of the muscles of mastication (Jonck 1978), arthroscopic lysis, lavage, and arthrocentesis (Nitzan et al 1991). Additional therapeutic measures may constitute of prosthetic reconstruction or orthodontic treatment.

TINNITUS AND CRANIOMANDIBULAR DISORDERS

Phylogenetical aspects

Phylogenetically, the middle ear bones are interpreted as jaw bones passed over to the service of hearing. A close relationship exists between the development of the jaw joints (masticatory apparatus) and ears (the locomotor system of the sound-conducting apparatus). Fossil evidence shows that the articulare bones of reptiles became incorporated into the mam-

malian middle ear as part of the incudomallear joint, *os articulare* becoming *malleus*, *os quadratum* - *incus*, and *os hyomandibulare* or columella - *stapes*. The mandible, malleus and incus are of common embryological origin being derivatives of the mandibular arch, while the stapes is derived from the second branchial arch. The tensor tympani and tensor veli palatini muscles develop from the first branchial arch, are innervated by the fifth cranial nerve - as are all muscles of mastication - and are, likewise, interpreted as originally being masticatory muscles. As the embryo develops, the pterygoid mass splits into three distinct muscles: the medial (internal) pterygoid, the tensor veli palatini, and the tensor tympani. These three muscles interdigitate: the fibres of the medial pterygoid mix with those of the tensor veli palatini, and the fibres of tensor veli palatini mix with those of the tensor tympani. The tensor veli palatini and tensor tympani muscles are thus part of the same muscular complex related to the muscles of mastication, with a common nerve supply from the trigeminal nerve. The stapedius muscle is an integral part of the mimetic muscles, the facial nerve being the common nerve supply (Du Brul 1964, Levy 1964, Arlen 1977).

Anatomical, physiological and clinical findings

Many attempts have been made to link TMJ and ear disturbances on anatomical basis, and conflicting opinions regarding coherence of disturbances in the masticatory apparatus and the middle ear have appeared in the dental and otological literature.

About 1920 several investigators claimed that deafness and tinnitus could be influenced by malposition of the jaws and 'loss of vertical dimension' (Monson 1920, Wright 1920, Decker 1925). In 1934 Costen proposed that malpositioning of the mandibular condyle could lead to Eustachian tube blockage and symptoms of aural pain, tinnitus, and vertigo.

Shapiro & Truex (1943), in an extensive review of the earlier literature regarding defects in auditory function that have been attributed to various changes within the TMJ, more or less disproved most of the theories claiming that deafness was caused by TMJ-disorders. As previously mentioned Costen's explanation of the mechanism whereby TMJ-dysfunction could produce otological problems was challenged by Sicher (1948) and Zimmerman (1951).

However, several authors have made the clinical observation that tinnitus is common in patients with CMD (although they named it differently). Goodfriend (1933) stated that deafness, tinnitus and vertigo, caused by pathological factors of the TMJ abnormality, are among the primary complaints of patients with TMJ diseases. In 1947 he postulated that tinnitus of long duration can be favourably altered in type and intensity, while that of short duration can be cured by dental treatment. According to Curtis (1980) the otological symptoms: aural fullness, subjective hearing loss, tinnitus and vertigo, were among the most common of the nonmasticatory symptoms in CMD.

The consequent theories of relationship between tinnitus and the masticatory apparatus were more functional and took into consideration the neuromuscular mechanisms. Fowler (1948b) postulated that contractions of the muscles about and within the head and the neck may all cause tinnitus. Shore (1959) stated that the ear is often affected by disturbances of the TMJ, with tinnitus, change in hearing ability and excessive wax formation as common symptoms. Travell (1960) has noted the association of unilateral tinnitus with a trigger point in the deep layer of the ipsilateral masseter muscle. Salomon & Starr (1963) have demonstrated electromyographically in humans that tensor tympani activity could be elicited with reflex periorbital muscle contraction and other facial movements such as grimacing and smiling. Profound activity also occurred with swallowing and talking. Furthermore, the stapedius muscle was active during jaw movements and vocalization. Such movement-associated middle ear activity was considered nonacoustic in nature. Kelly & Goodfriend (1964) postulated that traumatic dental malocclusion can cause blurring of vision, otalgia, tinnitus, hyperacusis, 'aerotitis media', deafness and vertigo.

Myrhaug (1964, 1969, 1981) coined the expressions 'oto-dental syndrome' and 'otognathic syndrome'. He reasoned that hyperactivity of masticatory muscles associated with occlusal factors and clenching would reflexively lead to disturbances of the tensor tympani because of their common nerve supply and thus cause such ear symptoms as tinnitus. Functional diminition of hearing, sense of fullness in the ear, paracusis and 'hyperaesthesia acoustica' where thought to have similar origin. Myrhaug postulated that malocclusion and dysfunction of the jaw cause both subjective and objective tinnitus, and referred to several cases of objective tinnitus related to mandibular dysfunction reported in the literature. He considered tinnitus to represent a fatigue reaction that arose as an autogenous vibration in the sound-conducting system due to tremor or myoclonus in the tensor muscle of the middle ear. The tensor muscles are reportedly stimulated along with the pterygoid muscle during jaw function and thereby affect the tympanic membrane and patency of the Eustachian tube. In the rare occasions of objective tinnitus the sound was thought to arise by means of rythmic opening and shutting movements of the pharyngeal opening of the eustachian tube, and both tensor muscles being involved. He concluded that the vibratory tinnitus must be considered as being produced by oscillations of the stapedial footplate creating fluid motions in the labyrinth which are adequate to stimulate the perceptive elements in the cochlea. Chole & Parker (1992) considered this line of reasoning as illogical because the muscles would be contracting at an unrealistically high frequency to cause a high-pitched tinnitus.

A presumed relationship between tinnitus and tensor tympani muscle contractions has been suggested by Klockhoff & Westerberg (1971) who described a psychosomatic syndrome caused by increased psychic tension due to mental stress. The 'tensor tympani syndrome' is characterized by aural fullness, tinnitus, dysacusis, tension headache, vertigo and otalgia.

Myrhaug (1981) proposed that the tensor tympani phenomenon is part of the 'otognathic syndrome'.

A frequent disturbance of the active opening of the tube during swallowing in patients with TMJ-problems has been reported by Philipp et al (1972). They proposed that this disturbance as well as unfavourable sensations in the ear were caused by an irritation within the trigeminal nerve distribution area, leading to incoordination of masticatory muscles. Arlen (1977) described the 'otomandibular syndrome' that included pain in and around the ear, fullness in the ear, hearing loss, tinnitus, and a loss of equilibrium. He proposed that patients with TMJ problems are tensing their muscles of mastication e. g. by clenching and/or bruxing, and that increased tension of tensor tympani muscles could result in a complaint of an ear problem or a subjective experience of hearing loss.

However, Brookes et al (1980), after examining 45 patients with TMJ dysfunction, otalgia and other aural symptoms (deafness, tinnitus, pressure/blockage and vertigo), argued that there is no direct aetiological basis to link TMJ dysfunction and other aural symptoms apart from otalgia.

According to House (1981) masticatory muscle spasm can potentiate and might be an origin of tinnitus, being an auditory 'pain'. Malkin (1987) reasoned that hypertonia of the internal (medial) pterygoid muscle, caused by neuromuscular dysfunction, produces a reflex hypertonia of the tensor tympani muscle and a relative hypotonia of the tensor veli palatini muscle, which results in an ineffective opening of the Eustachian tube and poor ventilation of the middle ear cavity, which in turn could induce pathological changes resulting in tinnitus and other symptoms.

Two structural grounds for connection between the ear and the TMJ were provided by Thonner (1953) and Pinto (1962). Thonner described a vascular supply from the internal maxillary artery to the inner ear, passing through the fissure system in the glenoid fossa, suggesting that being a possible anatomical base for disturbances in the inner ear caused by malocclusion and arthrosis of the TMJ. Pinto found a ligamentous structure connecting the neck and anterior process of the malleus to the mediposterosuperior part of the capsule, the interarticular disc, and the sphenomandibular ligament. It has been hypothesized that this structure, also named the malleomandibular ligament or anterior malleolar ligament, in cases of TMJ-dysfunction, can be deformed beyond its limit, resulting in increased pressure in the middle ear which would cause pain and annoying tinnitus (Mercuri et al 1982). Tasanen & Jokinen (1981) proposed that the condition of this ligament may be altered after closed condylotomy, with alleviation of middle ear derangements and ear symptoms. Here again, Chole & Parker (1992) argue that the mechanical energy that could be transmitted by these ligamentous structures to malleus cannot account for high-frequency subjective tinnitus.

An inflammatory state in the TMJ has been designated as cause of tinnitus by some authors. Morgan (1973) mentioned tinnitus among symptoms of a form of arthritis. Jonck (1978) suggested that tinnitus was related to

occlusal disharmony causing abnormal muscular spasms eventually resulting in arthritis of the TMJ. House et al (1984) postulated that pressure in the TMJ capsule causes middle and inner ear symptoms, and Myers (1988) hypothesized that in cases of so called retrodiskitis in the TMJ inflammatory products can penetrate the TMJ capsule and proceed to the carotid sheath area causing tension on the whole carotid sheath with any movement of the neck or jaw. If this tension was to be transmitted to the jugular foramen, the saccus endolymphaticus would be bound down against the bone above the sigmoid sinus and therefore unable to expand to perform any possible regulation of the endolymphatic pressure, and increased endolymphatic pressure on the hair cells of the cochlea could cause tinnitus.

Lusk & Babin (1985) described a case with tinnitus due to synkinesis involving the masseter muscle and the tensor tympani secondary to neurotmesis (a total traumatic disconnection) of the motor division of the trigeminal nerve. Shulman (1990) considered TMJ syndrome to be a contributory factor influencing particularly the parameters of tinnitus intensity and quality, but not the primary cause of tinnitus. Ash & Pinto (1991) proposed that functional (eustachian tube and muscles) and structural (retrodiskal changes associated with disk derangement) causes are to be considered for some subjective ear symptoms, such as tinnitus. Cooper & Cooper (1991) mentioned tinnitus among the painful symptoms of CMD and proposed that common innervation of the masticatory muscles and the two tensor muscles as well as compression or stretching of the posterior attachment fibres of the temporomandibular disk could be links in the TMJ/ear symptom phenomenon.

A hypothesis of excessive somatic concern, that would explain the high prevalences of tinnitus and vertigo in CMD-patients has been discussed by Chole & Parker (1992) but could not be supported by the findings in their study. The excessive somatic concern would be a syndrome in which complaints are disproportionate to the severity of physical findings.

Dauman & Tyler (1992) considered muscular problems associated with temporomandibular joint disorders being a sound-producing condition, heard as tinnitus.

Several authors advise identification and treatment of CMD as possible cause of, especially intermittent or fluctuating, tinnitus (Goodey 1981, Hazell 1987, Neuhauser 1992), while, for example, in the whole latest edition of 'The TMJ - a biological basis for clinical practice' by Sarnat & Laskin (1992) the symptom of tinnitus is not even mentioned other than as a sign of primary ear disease.

In conclusion: the opinions on existence of, and nature of a possible association between tinnitus and CMD are legion, and any consensus in that matter seems still very distant. Hopefully, the present study and future well-designed investigations will spread more light over this topic.

THE PRESENT INVESTIGATION

AIMS

- to review the effects of stomatognathic treatment on tinnitus in patients referred to a TMJ clinic (*paper I*)
- to determine the prevalence of signs and symptoms of CMD in subjects with disabling tinnitus as the main complaint (*paper II*)
- to examine the distribution of signs and symptoms of CMD in a group of patients with severe tinnitus and self-reported CMD-symptoms and/or headaches, and to describe how these symptoms are related to different aspects of mood and to audiological data (*paper III*)
- to define subgroups of patients with tinnitus related to CMD (*paper III*)
- to evaluate effects of stomatognathic treatment and clinical biofeedback-training on patients with disabling tinnitus (*paper IV*)
- to find background variables that could predict the outcome of biofeedback and stomatognathic treatment on tinnitus patients (*paper IV*)
- to analyze the longitudinal fluctuations of tinnitus (*paper V*)
- to explore associations between tinnitus, CMD and headache (*paper VI*)

SUBJECTS AND METHODS

Study I included in total 376 consecutive patients referred to the clinic of the Department of Stomatognathic Physiology at the University of Göteborg, Sweden. All patients, at their first visit to the clinic, answered a self-administered questionnaire that among others included the question if they suffered from ear noises. A few months later, a new brief questionnaire was sent to those who stated that they did so, concerning the characteristics that their tinnitus might possess, the stomatognathic treatment they had received, and its effect on tinnitus. Two years later those patients who initially reported a marked decrease of tinnitus after the stomatognathic treatment were followed up by a telephone interview.

Study II was based on 102 tinnitus patients, (40 women and 62 men with a mean age of 56 years, range 15 - 84), who attended the Department of Audiology at the Sahlgrenska Hospital in Göteborg. The patients underwent a clinical dental examination that included palpation of muscles and joints, measurements of mandibular mobility, determination of the anamnestic and clinical dysfunction indices (Ai and Di) according to

Helkimo (1974), assessment of the dentition, materials in dental restorations, occlusal factors and signs of parafunction. Furthermore, the patients answered a questionnaire about the occurrence and frequency of headache, fatigue and tenderness in chewing muscles, difficulties in moving the mandible, TMJ-clicking and bruxism. They were asked about possible influence on their tinnitus of mandibular movements, pressure on the TMJ or dental therapy. They also accomplished a tension test consisting of voluntary mouth opening and protrusion against resistance applied by the examiner, while the subjects were asked to report any changes in intensity of the tinnitus during the test.

Study III and *IV* comprised 42 patients (15 women and 27 men) with consistent and bothersome tinnitus, who originally attended the Department of Audiology, Sahlgrenska Hospital in Göteborg. Their mean age was 50 years (range 24-67). They were selected for participation in the study because of reported frequent headaches, clenching or grinding of the teeth or other subjective symptoms of CMD. The patients had been subjected to an otological examination by an ENT-specialist and audiological standard tests by an audiologist at the Department of Audiology. They were interviewed by a psychologist and examined and classified according to the clinical dysfunction index (Helkimo 1974). The patients were instructed to complete two weeks of three daily self-ratings of tinnitus intensity and severity, and three subscales of the Mood Adjective Check-list (MACL) reflecting *pleasantness*, *activation* and *relaxation*. Patients were randomly assigned for participation in the stomatognathic or the biofeedback treatment according to a cross-over design. The EMG biofeedback was supplemented with progressive relaxation. The stomatognathic treatment included occlusal splints, occlusal adjustment, and jaw exercises. After the completion of each treatment period, the patient accomplished the above described 'diary' assessments thrice daily for two weeks. Six months following the last treatment, the patients completed further 'diary' ratings during a two-week period.

Studies V and *VI* are parts of the gerontological and geriatric population study in Göteborg, Sweden (H70; Rinder et al 1975, Svanborg 1977, Eriksson et al 1987). *Study V* was based on a subsample, comprising 377 individuals, of one cohort of 70-years old inhabitants of Göteborg, who completed questionnaires concerning hearing problems, where one of the questions dealt with tinnitus. The investigation was repeated when the subjects were 75 and 79 years old. *Study VI* was based on subsamples, comprising totally 1005 individuals, of two cohorts of 70-year old inhabitants of Göteborg. The general medical, audiological and odontological investigations in the study were based on interviews by questionnaires, clinical examinations, and laboratory tests. Moreover, for estimation of socioeconomic status, data on officially registered incomes were obtained from official registers (Rinder et al 1975).

Statistical methods

In *study I* a *chi-square* analysis was used to determine whether there were any significant correlations between the reported effect of treatment and the other items in the questionnaire.

In *study II*, *III*, and *IV*, *Kendall's rank correlation coefficient* (τ) was used to examine intercorrelations between variables. Differences between subgroups in *study III* and *IV* were examined using *chi-square* analysis for dichotomous variables, *Mann-Whitney U-test* for other discrete variables, and *Student's t-test* for continuous variables. In comparisons between pre- and posttreatment ratings in *study IV*, *paired t-test* was applied for continuous variables, and *Wilcoxon matched pairs signed-ranks test* for discrete variables.

For group comparisons in *study VI odds ratios* were used for dichotomous or dichotomized variables, *Mann-Whitney U-test* for other discrete variables, and *Student's t-test* for continuous variables. Moreover, two *logistic models* with presence of tinnitus as the response variable were fitted. The object of statistical modelling is to present a simplified or smoothed representation of the underlying population. This is done by separating systematic features of the data from random variation. The systematic features are represented by a regression function involving parameters which can be simply related to important variables measured on each observational unit (Aitkin et al 1990).

The computer analyses were performed using the Statistical Package for the Social Sciences for personal computers (SPSS PC+; Norusis 1988), and the statistical package for Generalized Linear Interactive Modelling (GLIM3) developed by the Working Party on Statistical Computing of the Royal Statistical Society in the UK.

RESULTS

In *study I* 46% of the patients who had received stomatognathic treatment reported no tinnitus or reduced tinnitus a few months after the treatment. A two-year follow-up showed that the improvement remained in most of those who had benefitted from the treatment.

The results of *study II* indicated that frequent headaches, fatigue/tenderness in jaw muscles, clinical findings of pain on palpation of masticatory muscles, impaired mandibular mobility and signs of parafunctions were more prevalent in tinnitus patients than found in epidemiological samples. About one-third of the patients reported influence on tinnitus by mandibular movements and/or pressure applied to the TMJs.

Results from *study III* indicated that awareness of diurnal bruxism and feeling of jaw tenderness/fatigue may be related to fluctuating tinnitus, vertigo and hyperacusis. (fig 1). The results relating to mood dimensions were not conclusive.

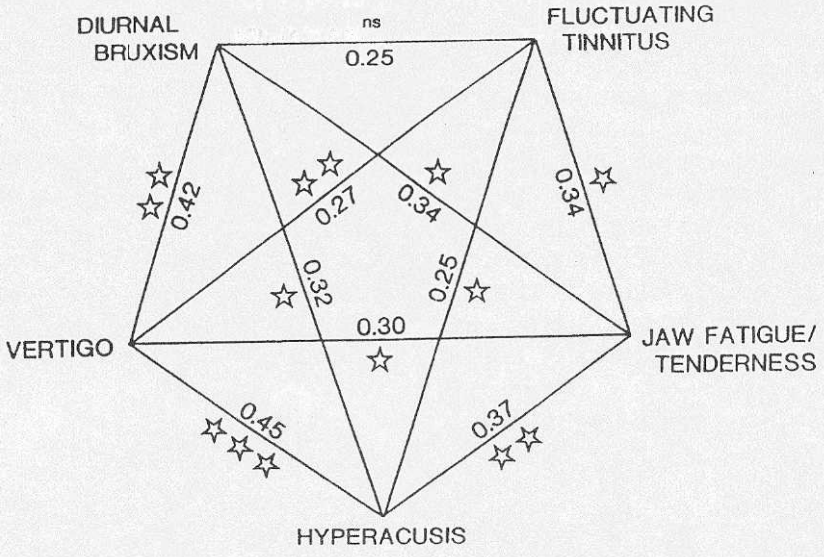

Figure 1. Intercorrelations between some stomatognathic, vestibular, and audiological symptoms in 42 tinnitus patients.

In *study IV* the evaluation of treatment outcomes showed some improvements at the group level: decrease of tinnitus intensity, mood improvement and reduction of clinical signs of dysfunction in the masticatory system. Possible predictors of stomatognathic and biofeedback treatment outcome, in sense of reducing tinnitus intensity, were observed. Such predictors would be: comparatively low severity of tinnitus, normal hearing or compensated hearing loss, occlusal interferences, jaw fatigue, awareness of diurnal bruxism and fluctuations in tinnitus intensity. Patients with left-sided tinnitus seemed to be poor candidates for stomatognathic and biofeedback treatment.

Study V revealed substantial longitudinal fluctuations in tinnitus with a high occurrence of spontaneous remissions (fig 2a-d), with the major changes between the ages 75 and 79 years, where the total prevalence of tinnitus increased from 28% to 42%, although 76% of those with continuous tinnitus at the age of 75 reported occasional or no tinnitus at the age of 79. Of the 153 subjects followed over the whole age range (70-79), only four (< 3%) reported continuous tinnitus at each investigation, whereas 39% reported no tinnitus at each investigation and occasional or continuous tinnitus was reported on each occasion by 13%.

Study VI showed a strong relationship between tinnitus complaints and several symptoms of CMD. The prevalence of headache, fatigue/tenderness in jaw muscles and several other symptoms and signs of CMD was

markedly higher in an elderly population with than one without tinnitus. This relationship seemed to be independent of the degree of hearing loss, occupational noise exposure, general morbidity, medication or socioeconomic status. Here again, about one third of the tinnitus population reported influence on tinnitus by mandibular movements, pressure on the TMJ or changes in the occlusion. Despite the fact that hearing loss on average was more pronounced in the tinnitus complainers than in others in this study, a majority of tinnitus complainers seemed to have had hearing within the range expected from their age group. The prevalences of headache and CMD-symptoms were, with a few exceptions, almost identical in persons with no or slight hearing loss and those with pronounced hearing impairment. However, in those with self-estimated hearing deficiency the prevalences of headache and CMD-symptoms were significantly higher.

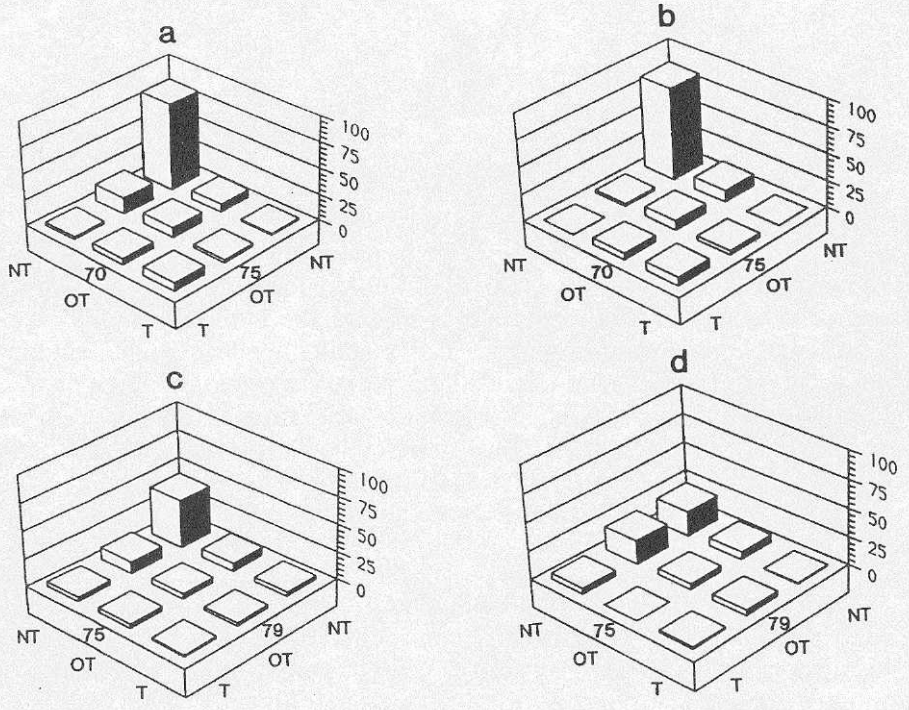

Figure 2. Fluctuation of tinnitus between age of 70 and 75 years (a,b), respectively 75 and 79 years (c,d), in women (a,c) and men (b,d). NT = no tinnitus, OT = occasional tinnitus, T = continuous tinnitus.

DISCUSSION

Comments on methods

There are no simple measures for separating individuals affected by tinnitus from those who are not, and there is a lack of universally accepted classification schemes with diagnostic criteria of tinnitus. The problems are similar for CMD, especially the lack of accepted classification. Most people have at some time or another in their lives experienced some of those symptoms in a more or less pronounced way. In spite of the fact that several attempts have been made to quantify the severity of both tinnitus and CMD, this possibility is still not satisfactorily met. Instead we have to rely on qualitative descriptions.

Because of the subjective character of the tinnitus complaint as well as of several symptoms of CMD the means of investigation must be: interviews, questionnaires, or rating scales. All these methods have been employed in this study, and they all have both advantages and disadvantages. The validity and reliability of results from interview- or questionnaire-studies have been investigated and discussed by several authors (Collen 1969, Meltzer & Hochstim 1970, Kopp 1976, Rieder 1977, Agerberg & Helkimo 1987, Wänman 1987, Baskill et al 1991) and have in general been found acceptable. Collen ranked 204 questions, commonly used in self-administered medical history questionnaires, as to their relative reliability. Question "Have you, in the past 6 months, often had noises (buzzing or ringing) in the ear?" was the fifth of totally 31 head-eye-ENT questions ranked in descending order of reliability, and was considered to have good reliability. Meltzer & Hochstim found in a study of reliability a general high consistency between responses to two identical questionnaires on physical complaints, self-administered about a week apart. Baskill et al reported an acceptable test-retest reliability of a tinnitus questionnaire.

Tinnitus matching was initially performed on some patients participating in *study III* and *IV*. It has, however, not been used in the correlation analyses or treatment evaluation because of the subjective character of the complaint, and the potentially high measurement error in relation to the audiometrically estimated loudness level. Self-ratings of tinnitus were also preferable because they could be repeated several times, and during a long period, thus diminishing the influence of the inherent variability of this symptom.

The clinical dysfunction index (D_i) or clinical dysfunction score (CDS) according to Helkimo have been used in *studies II-IV*. In a study by Carlsson et al (1974) good agreement was found between three trained observers in assessment of the D_i. The D_i has also been found to have high correlation with both subjective symptoms and other indices for objective signs of CMD. Several studies have been performed in order to assess the inter- and intraobserver variability in estimation of clinical

signs of CMD. The results of these studies have been reasonably satisfactory regarding summary indices, measuring the overall presence of a clinical sign (Carlsson et al 1980, Magnusson 1981, Kopp & Wenneberg 1983, Duinkerke et al 1986, Fricton & Schiffman 1987, Dworkin et al 1990). Thus, both D_i and CDS should be regarded as fairly reliable. The assessment of validity of clinical examination for CMD is more problematic, because of lack of external criteria against which to assess it. In fact, the results of clinical examinations are often themselves used as criteria for validating investigations done by questionnaires and interviews, and they have, in clinical practice, been useful as guides to treatment and prognosis of CMD.

The dropouts and missing data on some participants in *study I* and *study IV* may have led to an over- or an underestimating of treatment effects, which certainly call for caution when interpreting the results. In *study IV* a high proportion of the dropouts had Ménière's disease, but they did not differ significantly from the participants in any other background variable or in baseline ratings. In the remaining studies there are no reasons to assume that missing data would have biased the results.

Tinnitus and headache

High prevalences of headache in tinnitus patients, ranging from 23% to 77%, have been reported from several studies (Hazell 1981, Graham & Butler 1984, Claussen & Claussen 1987, Scott et al 1990). Strong correlation between headache frequency and the severity of tinnitus have been noted by Lindberg et al (1984) and Erlandsson et al (1992), and high prevalence of tinnitus (46%) has been found in a population of chronic headache sufferers (Gelb & Tarte 1975). The most convincing evidence for an association between headache and tinnitus are findings from epidemiological studies. The highly significant correlation between headache and tinnitus found in *study VI* is supported by a similar finding noted in a study of the male population in Copenhagen (Parving et al 1992).

Tinnitus in CMD-patients and CMD in tinnitus patients

High prevalences of tinnitus, ranging from 33% to 67%, some thus by far exceeding those found in epidemiological studies have been observed among patients attending TMJ-clinics (Dolowitz et al 1964, Gelb et al 1967, Gelb & Tarte 1975, Morgan 1975, Curtis 1980, Fricton et al 1985, Gelb & Bernstein 1983, Bush 1987). Tinnitus and other otological symptoms are reported to be common in patients with symptomatic internal derangements of the TMJ, and frequently perceived ipsilaterally to the internal derangement (Mercuri et al 1982, McKenna & Hall 1990). Chole & Parker 1992 found significantly higher prevalences of tinnitus and vertigo in patients with TMJ-pain and clicking than in two comparable age-matched control groups, and concluded this association being indisputable but its mechanism unknown.

Signs and symptoms of CMD seem to occur frequently in patients who consult audiological clinics for tinnitus. In *study II* 84% of the patients had signs and 46% had symptoms of CMD. Schleuning (1989, 1991) reported that 45% of patients with tinnitus seen at the Tinnitus Clinic in Portland described active TMJ problems at one time or another. Of patients who had severe tinnitus, 38% described it as occurring concurrently with the increase of severity of their complaint and over 30% of patients with TMJ-symptomatology related the tinnitus to the onset of their jaw symptoms. Morgan (1992) examined twenty patients with tinnitus as the chief complaint and found a high frequency of clinical, electromyographic and radiographic indications of a temporomandibular disorder. Moreover, in a prospective study of patients with inner ear dysfunction, 47% of all patients, and 72% of patients with isolated tinnitus had signs or symptoms of CMD (Kempf et al 1993).

The influence on tinnitus by jaw movements or pressure to the TMJ has been observed by Melding et al (1978), and by Berlin, House & House (1981), and was frequently reported both in the tinnitus patients in *study II* and the tinnitus population in *study VI*. Moreover, in the Tinnitus Clinic in Portland about 13% of patients reported jaw movements altering their tinnitus (Vernon et al 1992). This may indicate that increased activity in masticatory muscles increases or even causes the perception of tinnitus. The relatively frequent awareness and signs of parafunctions found in individuals affected by tinnitus in *study II* and *VI* indicate a muscular hyperactivity in their masticatory systems.

Tinnitus and CMD in an epidemiological perspective

Both tinnitus and signs and symptoms of CMD are common in the general population, so even assuming that these conditions are unrelated to each other the prevalence of coincidental tinnitus and symptoms of CMD may be fairly high in selected samples, particularly if the selection factor is uncontrolled. Clinical samples of CMD-patients are probably not representative for all individuals with symptoms of CMD, as well as clinical samples of tinnitus patients not representing all individuals affected by tinnitus. Multiple complaints may influence individuals to seek advice and help from one or other specialist. That makes even a case-control study design insufficient to verify hypotheses of association between these complaints. For that purpose use of cross-sectional sampling design in the general population is needed.

Study VI is a cross-sectional study of two cohorts of an elderly population, where a strong relationship between tinnitus complaints and several symptoms of CMD has been found. This relationship seems to be independent of the degree of hearing loss, occupational noise exposure, general morbidity, medication or socioeconomic status. The study was carried out on a sample of 70-76 years old individuals. Therefore, one must bear in mind that the results obtained and the interpretations made relate to the population from which the sample was taken. However, results from

study II and other studies (Wänman & Agerberg 1987, 1990, Chole & Parker 1992) make similar findings in other, younger, populations plausible. Despite limitations mentioned the results are of sufficient magnitude to warrant further studies. Since a cross-sectional study cannot establish a causal connection, there is a need of long-term, large-scale epidemiological studies, investigating many potential factors that could influence on tinnitus. An analysis of longitudinal data on tinnitus, hearing and CMD may give a base for better understanding of the underlying mechanisms. Longitudinal epidemiological studies can show when preventive intervention techniques are needed as well as how to determine potential aetiological factors.

Natural history of tinnitus and CMD

It is of the utmost importance to know the natural course of the symptom before influence of other factors is studied. There exists established knowledge that substantial fluctuations and spontaneous remissions of headache and CMD-symptoms are common (Wänman 1987, Wänman & Agerberg 1988, 1990, Schiffman et al 1990, Hampf 1992). The present knowledge about the natural history of tinnitus is markedly poorer and there exists a great risk for a pessimistic bias concerning prognosis for tinnitus, due to the fact that individuals free from symptoms seldom have reason to attend a medical clinic. This state of things may lead to self-compliant prophecy: a physician, who tells the patients that there is nothing that can be done and that they have to learn to live with their tinnitus may contribute to vicious circles whereby the muscle tension increases and aggravates the tinnitus, a nocebo effect.

Jackson (1983) stated *'In general, once tinnitus is troublesome it can be expected to persist so that its prevalence in the population increases with age. Most elderly patients with tinnitus will have had this symptom for many years'.* Slater & Terry (1983) estimated the likelihood of spontaneous remission for all forms of tinnitus to be about one in 20, and for intermittent type of tinnitus of short duration they estimated the chance of spontaneous recovery to increase slightly but still be less than one in ten. Meikle & Taylor-Walsh (1984) mentioned that many patients report their tinnitus started at a mild level and gradually grew louder, to a distressing level, over a period of years. Scott et al (1990) observed in a retrospective questionnaire survey, covering patients from 52 hearing centres in Sweden, a significant increase in discomfort from tinnitus and tinnitus loudness over time. Further, in a study of 528 tinnitus patients, Stouffer et al (1991) noted that tinnitus loudness and severity increased as a function of years since onset. They discussed that due to the strong relationship between tinnitus and hearing loss, and because hearing loss usually is progressive, it was unlikely that tinnitus symptoms would decrease dramatically or disappear. They admitted, however, the lack of data on the prevalence of spontaneous remission and/or dramatic improvement of tinnitus.

However, in the last few years some attention has been paid to the natural course of tinnitus in non-clinical samples, and the perspective became less pessimistic. From retrospective population statistics on the natural history of tinnitus Smith & Coles (1987) have concluded that, soon after the onset period, the general pattern of severity of tinnitus is likely to decrease gradually and sometimes the tinnitus even disappears altogether; it does not often get markedly worse, and if there is any change in loudness at all, it is more likely to involve a gradual decrease. Moreover, in the UK epidemiological study 25% of those who reported having experienced tinnitus at one time, denied having it 'nowadays' at the time of investigation (MRC(IHR) 1987). *Study V* gives an indication of substantial longitudinal fluctuations in the tinnitus reports with a high occurrence of spontaneous remissions of tinnitus in elderly people. A 9 year-study of a smaller sample of 19-year-old adolescents showed a similar high rate of remissions of tinnitus (Rubinstein et al 1992).

Effects of stomatognathic treatment on tinnitus

There is no simple way of evaluating treatment effects in a heterogeneous group of tinnitus patients. It is important to keep in mind that tinnitus is a symptom and not a disease, and no single modality of therapy will be universally successful. In *study I* and in several other studies (table 1) stomatognathic treatment intending to reduce CMD had positive side-effects on tinnitus, reducing its intensity or eliminating it totally in some patients.

Table 1: Reported effects on tinnitus of dental and stomatognathic treatment in 13 studies.

Study	Number of evaluated patients	Percentage improved or symptomfree
Junemann 1941	9	78
Gelb & Arnold 1959	12	50
Hankey, 1962	6	50
Dolowitz et al 1964	43	93
Kelly & Goodfriend 1964	46	80
Gelb et al 1967	26	96
Bernstein et al 1969	28	75
Gelb & Tarte 1975	38	82
Koskinen et al 1980	8	63
Ioannides & Hoogland 1983	2	50
Cooper et al 1986	?	~50
Rubinstein & Carlsson 1987	61	46
Bush 1987	35	73

The arbitrary determined improvement rate in *study IV* was 41%. Alleviation or relief of tinnitus has also been described following TMJ

surgery (Morgan 1975, House et al 1984, Bell et al 1990, Anderson et al 1991). Principato & Barwell (1978) reported resolution of tinnitus in 71% of patients with CMD treated with EMG biofeedback. Dental and stomatognathic treatment in patients with inner ear dysfunction was reported to improve the otological symptoms, including tinnitus in 57% of cases (Kempf et al 1993).

However, one should be cautious interpreting these results. None of these studies included any control group and one part of the achieved treatment effects could be accounted for by regression to the mean of a fluctuating tinnitus, and another part of the effects could consist of unspecific ones: giving attention to the patient, giving him general counselling and possibility to express his concerns about how the tinnitus is affecting his life, understanding of the nature of his problem, the reassurance and emotional support, and so called placebo-effect. The placebo effect can be considerable in the treatment of tinnitus. In a study reported by Fowler (1942) subcutaneous injection of normal saline solution resulted in recorded improvement of tinnitus in 23% of cases. Duckert & Rees (1984) investigating the contributions of a placebo effect in clinical tinnitus studies, reported that 40% of patients who received a placebo saline solution injection reported a change in their tinnitus following injection. Erlandsson et al (1987) showed in their experiment that 24% of 21 patients treated with a placebo equipment reported reduction of tinnitus following the treatment. Myers (1975) suggested that the use of placebos is probably the best known treatment for tinnitus. This hypothesis is still not disproved.

The positive predictors of stomatognathic and biofeedback treatment outcome suggested on basis of the results of study IV were: comparatively low severity of tinnitus, normal hearing or compensated hearing loss, occlusal interferences, jaw fatigue, awareness of diurnal bruxism and reported fluctuations in tinnitus intensity. These findings are in accordance with those observed in other tinnitus treatment outcome studies (House 1981, Meikle et al 1984, Haralambous et al 1987, Bush 1987) and *study I* in the present thesis. Attempts to identify predictors of tinnitus therapy outcome have also been made by other authors. On evaluating effects of reassurance, relaxation training and distraction on chronic tinnitus sufferers Jakes et al (1986) found that the 'non-improvers' could be distinguished from the 'improvers' by higher pre-treatment scores of distress, hearing difficulties, intrusiveness, tinnitus loudness and annoyance, and more continuous awareness of tinnitus. All 'non-improvers' in their study had hearing impairment, to be compared with 44% of the 'improvers'. The prerequisites for tinnitus that could be positively influenced by cervical therapy, as listed by Biesinger (1992) were: normal hearing, unilateral, fluctuating tinnitus, young patient, influence on tinnitus by head movements, and onset following trauma to the cervical spine.

Interestingly, Goodey (1981) reported that the group of patients not responding to either lidocaine or masking included a high proportion with

normal hearing. Furthermore, Ueda (1992) noted a better outcome of intravenous lidocaine therapy in cases with hearing levels above 40 dB.

Thus, it seems that relatively low severity of tinnitus, normal hearing, and fluctuations in tinnitus intensity are good predictors of psychological, stomatognathic and cervical treatment outcome. The positive predictive value of low severity of tinnitus contradicts partially the assumption that regression to the mean has influenced the results reported here.

Tinnitus, hyperacusis, subjective hearing loss, and CMD

Hyperacusis or 'phonophobia' is often reported by tinnitus patients. Sood & Coles (1988) found the mean uncomfortable loudness levels distinctly lower in tinnitus subjects (both in the tinnitus ear and the non-tinnitus ear) when compared with non-tinnitus subjects. The authors concluded that this 'phonophobia' is probably due to psychological rather than pathophysiological disorder. In a survey of 104 hyperacusic patients Reich & Griest (1992) reported that 63.5% had problems associated with their jaw or bite (jaw or face pain, 'bad bite', grinding of teeth, jaw clicking), being a larger percentage than observed in the tinnitus population without hyperacusis. In most cases the jaw problems were present before onset of hyperacusis. Hazell & Sheldrake (1992) studied retrospectively, in 128 patients referred with a primary complaint of tinnitus, the prevalence of hyperacusis before tinnitus started, and compared it with the prevalence of hyperacusis at the time of investigation. They found that significant hyperacusis occurs in the pre-tinnitus state and that it increases substantially after tinnitus develops.

Sense of impaired hearing has previously been reported to occur frequently in cases with temporomandibular joint dysfunction (Costen 1944, Dolowitz et al 1964, Bernstein et al 1969). The results of *study VI* indicated that subjective hearing loss may be associated with both tinnitus and CMD. Hinchcliffe (1992) described a condition characterized by perceived auditory disability in association with an essentially normal manual pure-tone audiogram and termed it King-Kopetzky syndrome. He suggested that this condition was primarily an auditory stress disorder. As shown in *study VI* there seems to exist a certain association between CMD-symptoms, headache, and both tinnitus and subjective hearing loss, whereas no such association has been found with objective hearing loss. This may imply that the King-Kopetzky syndrome could be related to tinnitus, headache, CMD-symptoms, and probably hyperacusis. The existence of a stress-related condition including several of following symptoms: fluctuating tinnitus, subjective hearing loss, hyperacusis, dizziness/vertigo, diurnal bruxism, jaw fatigue/stiffness and headache, is implicated by the results of *study III* and *study VI* together with the results of studies on hyperacusis (Reich & Griest 1992). The conceivable common cause may be a generally increased muscle tension in the oro-facial region. But further studies are needed to establish or reject the hypotheses emanating from the present study.

CONCLUSIONS

- Several findings in the studies indicate an association between signs and symptoms of CMD and tinnitus.
- The prevalence of frequent headaches and fatigue/tenderness in jaw muscles seems to be higher in tinnitus patients than in epidemiological samples. The prevalence of clinical findings, such as pain on palpation of masticatory muscles, impaired mandibular mobility and signs of parafunctions, seems also to exceed the prevalence of these findings in epidemiological studies.
- About one third of the individuals affected by tinnitus report influence on tinnitus by mandibular movements and/or by pressure on the TMJs.
- Awareness of diurnal bruxism and feeling of jaw tenderness/fatigue seem to be related to fluctuating tinnitus, vertigo and hyperacusis.
- The present study shows that in a community based sample of elderly there is a strong relationship between tinnitus complaints and several symptoms of CMD, including frequent headaches.
- This relationship seems to be independent of the degree of hearing loss, occupational noise exposure, general morbidity, medication, and socioeconomic status.
- The association between objective hearing loss and tinnitus seems not to be much stronger than the one between CMD and tinnitus, while subjective hearing loss seems to be associated with both tinnitus and CMD.
- Stomatognathic and biofeedback treatment seem to be able to reduce or eliminate tinnitus in selected groups of patients.

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