

TURUN YLIOPISTON JULKAISUJA
ANNALES UNIVERSITATIS TURKUENSIS

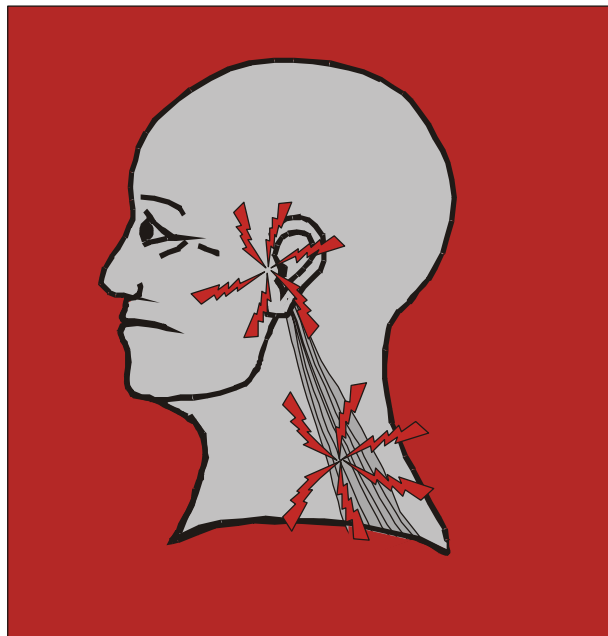
SARJA – SER. D OSA – TOM. 579

MEDICA ODONTOLOGICA

Secondary aural symptoms in relation to cranio-cervical and general disorders.

by

Seppo Kuttila



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From:

the Institute of Dentistry, University of Turku and
the Department of Otorhinolaryngology

Supervised by:

Professor Pentti Alanen
Department of Community Dentistry
Institute of Dentistry
University of Turku, Turku, Finland
and

Docent Yrsa Le Bell
Institute of Dentistry
University of Turku, Turku, Finland
and

Professor Jouko Suonpää
Department of Otorhinolaryngology
University of Turku

Reviewed by:

Professor Mauno Könönen
Chair Stomatognathic Physiology and Prosthetic Dentistry
Institute of Dentistry, University of Helsinki, Finland
Professor, Department of Restorative Sciences
Faculty of Dentistry, Kuwait University, Kuwait
and

Docent Markus Rautiainen
Department of Otorhinolaryngology
Tampere University Hospital
University of Tampere, Tampere, Finland

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To Marjaana and Jenni



You are the rhythm and music of my life



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ABSTRACT

The present thesis comprises two study populations. The first study sample (SS1) consisted of 411 adults examined and interviewed at three annual visits. The second study sample (SS2) consisted of 1720 adults who filled in a mailed questionnaire about secondary otalgia, tinnitus and fullness of ears. In the second phase of the SS2, 100 subjects with otalgia were examined and interviewed by specialist in stomatognathic physiology and otorhinolaryngology. In the third phase, 36 subjects participated in a randomized, controlled and blinded trial of effectiveness of occlusal appliance on secondary otalgia, facial pain, headache and treatment need of temporomandibular disorders (TMD).

The standardized prevalence of recurrent secondary otalgia was 6%, tinnitus 15% and fullness of ears 8%. Aural symptoms were more frequent among young than old subjects. They were associated with other, simultaneous aural symptoms, TMD pain, head and neck region pain, and visits to a physician. The subjects with aural symptoms more often had tenderness on palpation of masticatory muscles and clinical signs of temporomandibular joint than the subjects without. 85% of the subjects reporting secondary otalgia had cervical spine or temporomandibular disorder or both. In SS1, the final model of secondary otalgia included active need treatment for TMD, elevated level of stress symptoms, and bruxism. In SS2, the final models of aural symptoms included associated aural symptoms, young age, TMD pain, headache and shoulder ache. Stabilization splint more effectively alleviated secondary otalgia and active treatment need for TMD than a palatal control splint.

In patients with aural pain, tinnitus or fullness of ears, it is important to first rule out otologic and nasopharyngeal diseases that may cause the symptoms. If no explanation for aural symptoms is found, temporomandibular and cervical spine disorders should be ruled out to minimize unnecessary visits to a physician.

Key words: adults, epidemiology, secondary otalgia, tinnitus, fullness of ears, TMD, CSD

TIIVISTELMÄ

Väitöskirja koostuu kahdesta tutkimusaineistosta. Ensimmäisessä aineistossa oli 411 aikuista, jotka osallistuivat kolmeen vuosittaiseen tutkimukseen ja haastatteluun. Toinen aineisto koostui 1720 aikuisesta, jotka aluksi vastasivat kirjekyselyyn korvakivusta, korvan soimisesta ja korvien tukkoisuudesta. Tutkimuksen toisessa vaiheessa 100 tutkittavaa, joilla oli kirjekyselyn perusteella toissijainen korvakipu, osallistui klinisiin tutkimuksiin ja haastatteluihin. Kolmannessa vaiheessa 36 tutkittavaa osallistui satunnaistettuun, kontrolloituun ja sokkoutettuun tutkimukseen purentakiskon tehokkuudesta toissijaisen korvakivun, kasvosäryn, päänsäryn ja purentaelimen toimintahäiriön hoidossa.

Iän ja sukupuolen mukaan vakioitu toissijaisen korvakivun esiintyvyys oli 6 %, korvan soimisen 15 % ja korvien tukkoisuuden 8 %. Korvaoireet olivat yleisempiä nuorilla kuin vanhoilla. Korvaoireet olivat yhteydessä toisiin samanaikaisiin korvaoireisiin, kasvosärkyyn, pään ja niskojen alueen särkyyn ja lääkärissä käynteihin. Korvaoireisilla oli muita enemmän arkuutta puremalihasten ja leukanivelten palpaatiossa. Yhteensä 85 % toissijaisesta korvakivusta oli selitettävissä kaularangan ja/tai purentaelimen toimintahäiriöllä. Korvakipuisista 15 %:lla ei ollut kaularangan eikä purentaelimen toimintahäiriötä.

Seurantatutkimuksessa (n=411) toissijaisen korvakivun selitysmalliin kuuluivat purentaelimen toimintahäiriön hoidontarve, kohonnut stressioireiden määrä sekä bruxismi. Toisessa tutkimusaineistossa (n=1720) korvaoireiden selitysmalliin kuuluivat muut korvaoireet, nuoret ikäluokat, kasvokipu, pään- ja niskasärky.

Kiskohoitotutkimuksessa purentakisko oli tehokkaampi kuin kontrollikisko lievittämään tai poistamaan toissijaista korvakipua ja purentaelimen toimintahäiriötä kymmenen viikon seuranta-aikana.

Yhteenvedona totean, että korvaoireisen potilaan tutkimuksessa tulee aluksi selvittää, onko kyse korvan tai nenänielun sairaudesta, joka aiheuttaa korvaoireen. Mikäli korvaoireen syy ei selviä kliinisessä tutkimuksessa, purentaelin- ja kaularankaperäiset korvaoireiden syyt tulee selvittää. Näin toimien vältetään turhilta lääkärikäynneiltä ja lääkkehoidoilta.

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ABBREVIATIONS

The following abbreviations appear in the text:

BSI	Brief Symptom Inventory
CEXT	Cervical extension
CFLE	Cervical flexion
CFLR	Cervical flexion with rotation
Comb	Combined cervical spine and temporomandibular disorder
CSD	Cervical spine disorder
CREX	Cervical rotation with extension
D _i	Clinical dysfunction index by Helkimo (1974)
DSM-III	Diagnostic and Statistical Manual of Mental Disorders by American Psychiatric Association (1980)
NF group	Subjects with secondary otalgia not fitting into the clinical groups
RCT	Randomized, controlled trial
SS1	Study sample 1 (n=411)
SS2	Study sample 2 (n=1720)
TMD	Temporomandibular disorder
TMJ	Temporomandibular joint

LIST OF ORIGINAL PUBLICATIONS

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

- I** Kuttila S, Kuttila M, Niemi PM, Le Bell Y, Alanen P, Suonpää J. Secondary otalgia in an adult population. *Arch Otolaryngol Head Neck Surg* 2001;127:401-405.
- II** Kuttila S, Kuttila M, Le Bell Y, Alanen P, Suonpää J. Secondary otalgia, tinnitus and fullness of ears in a Finnish sample. Manuscript.
- III** Kuttila S, Kuttila M, Le Bell Y, Alanen P, Suonpää J. Characteristics of subjects with secondary otalgia. Submitted for publication.
- IV** Kuttila S, Kuttila M, Le Bell Y, Alanen P, Suonpää J. Aural symptoms and signs of temporomandibular disorder in association with treatment need and visits to physician. *Laryngoscope* 1999;109:1669-1673.
- V** Kuttila M, Le Bell Y, Savolainen-Niemi E, Kuttila S, Alanen P. Efficiency of occlusal appliance therapy in secondary otalgia and temporomandibular disorders. *Acta Odontol Scand* 2002;60:248-254.

The original papers have been published in this thesis with the permission of the copyright holders. Some additional information is also presented.

1. INTRODUCTION

Aural symptoms indicating an otologic problem are otorrhea, otalgia, fullness of ears, hearing loss, vertigo, and tinnitus (Harker 1998). However, otalgia, tinnitus and fullness of are not always diagnostic of aural disease. It is not uncommon for a patient to complain of otalgia or fullness of ears and have no identifiable pathology within the ear. Primary otalgia is aural pain originating in the ear, while secondary otalgia originates from a non-otologic source. More than 50% of aural pain is secondary otalgia (Paparella and Young 1991). It can result from referred pain of the neck or the orofacial region (Okeson 1996).

Aural symptoms like otalgia, tinnitus, impaired hearing, fullness of ears, hyperacusis and vertigo are common in functional disturbances of the masticatory system (Curtis 1980, Cooper and Cooper 1993, Rubinstein 1993, Keersmaekers et al. 1996). In patient studies, otalgia and tinnitus are often connected with temporomandibular disorders (TMD), as well as with cervical spine disorders (CSD) (Brookes et al. 1980, Curtis 1980, Koskinen et al. 1980, Friction et al. 1985, Feinmesser and Fluman 1987, Biesinger 1989). In the study by De Wijer (1995), patients with TMD more often reported aural symptoms than patients with CSD.

Occlusal splint trials have shown a positive outcome of TMD and associated aural symptoms (Okeson et al. 1982, Yap 1998, Ekberg et al. 1998, Ekberg et al. 2003) while some have shown no efficiency (Rubinoff et al. 1987, Davies and Gray 1997, Dao et al. 1994). According to Rubinstein (1993), stomatognathic treatment consisting of occlusal splint, occlusal adjustment and exercise therapy treatment reduced or eliminated tinnitus in some patients. However, according to Forssell et al. (1999), most of the studies showing a positive treatment outcome have not been based on the principles of randomized, controlled trials (RCTs).

Epidemiology has a vital role in improving our understanding of the causes, natural course and impact of, e.g. aural symptoms. Despite extensive population-based studies on tinnitus abroad, many questions about aural symptoms, especially about secondary otalgia, are yet unanswered. Furthermore, no Finnish population-based studies have been published on otalgia, tinnitus or fullness of ears.

2. REVIEW OF THE LITERATURE

2.1. ANATOMY AND PATHOPHYSIOLOGY OF PAIN IN THE EAR, HEAD AND NECK

2.1.1. Sensory innervation of ear

Sensory innervation of the auricle is derived from the great auricular nerve (C2, C3: helix, antihelix, lobule), the lesser occipital nerve (C2: upper part of the cranial surface), the auricular branch of the vagus (concavity of the concha and posterior part of the eminentia), the auriculotemporal nerve (trigeminus: tragus, crus of the helix and the adjacent part of the helix), the facial and vagal nerves (on both aspects of the auricle in the depression of the concha and over its eminence). Sensory innervation of the external ear canal is derived from the auriculotemporal branch of the mandibular nerve (anterior and superior walls of the meatus), and the auricular branch of the vagus (posterior and inferior walls) (Williams et al. 1999) (Fig. 1).

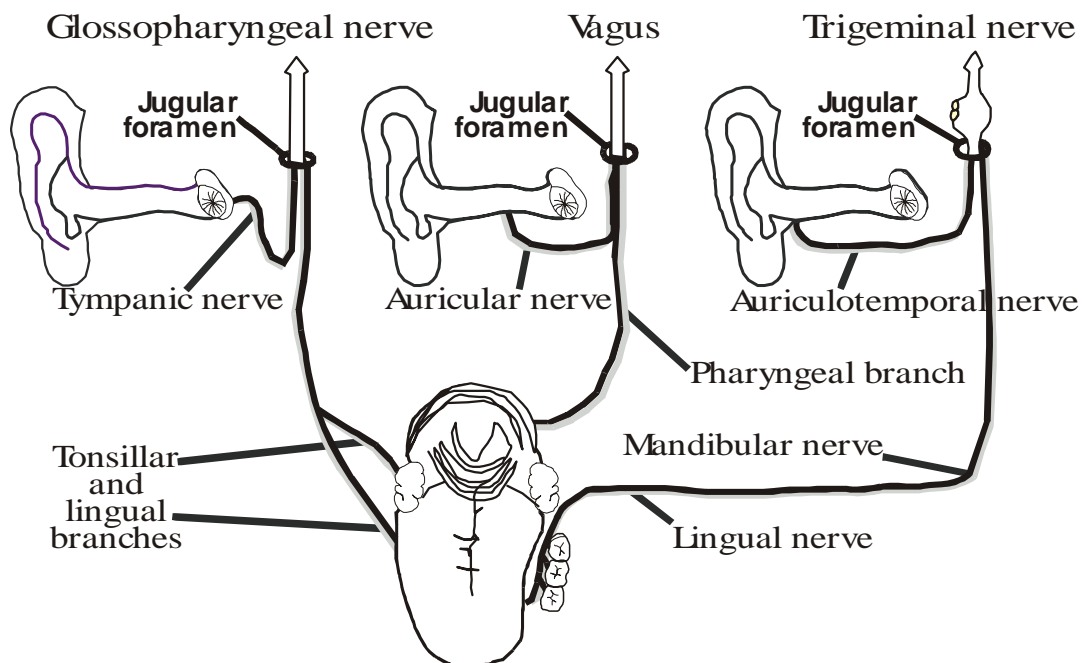


Figure 1. The schematic distribution of the sensory branches of trigeminal, glossopharyngeal and vagal nerves innervating the ear (adapted from Paparella and Jung. *Otolaryngology* (1991)).

The tympanic membrane is almost exclusively innervated by the auriculotemporal nerve, and appears to perceive only pain. There is a minor, inconstant, overlapping sensory supply from the seventh, ninth and tenth cranial nerves. The nervus intermedius is the sensory root of the facial nerve. The pharyngeal nerve (branch of maxillary nerve) transmits the principal sensory supply from the Eustachian tube and middle ear cavity. Some sensory fibres are also transmitted via the facial nerve (nervus intermedius), via Jacobsen's nerve (the glossopharyngeal and vagal nerve), and the nerve of Arnold respectively also make a contribution. All these somatic sensory afferents converge in the brainstem to synapse in the descending trigeminal sensory nucleus, and in the nucleus of the tractus solitarius.

2.1.2. Pathophysiology of pain in the ear, head and neck

Infectious diseases such as medial or external otitis are the most frequent causes of primary otalgia. Other causes of primary otalgia are trauma and tumor (Table 1a). They are all easily diagnosed by a clinical otorhinolaryngologic examination. Secondary otalgia is quite easily detected in patients with diseases of the paranasal sinuses, nose, mouth, teeth, tonsils, pharynx, larynx or thyroid gland (Harker 1998).

Table 1a. Causes of primary otalgia.

<p>Auricle</p> <ul style="list-style-type: none"> Peri- or polychondritis Relapsing chondritis Frostbite or burn Trauma 	<p>Middle ear</p> <ul style="list-style-type: none"> Myringitis Otitis media <ul style="list-style-type: none"> bacterial viral <p>Complicated otitis media</p> <ul style="list-style-type: none"> Mastoiditis Petrositis Extradural or intradural abscess Subperiosteal abscess Brain abscess Venous sinus thrombosis
<p>External meatus</p> <ul style="list-style-type: none"> Cerumen impaction Foreign body Carbuncle or furuncle Trauma Otitis externa <ul style="list-style-type: none"> bacterial fungal viral 	<p>External, middle ear or skull base</p> <ul style="list-style-type: none"> Neoplasms Metastatic tumours

Other suggested causes of secondary otalgia are referred pain from temporomandibular (TMD) or cervical spine disorders (CSD); from neural, vascular, or lymphatic structures of the jugular neck; or from oesophagus, heart, or lungs. Table 1b shows two lists of infectious and inflammatory diseases which often are quite acute in nature, and pain disorders and diseases associated with secondary otalgia, which often are prolonged or chronic in nature.

Referred pain is usually unilateral, recurrent and locates on the same side of the head as tenderness on palpation of stomatognathic muscles or TMJ (Blake et al. 1982, Kramer and Kramer 1985). Pain referral into the orofacial region has been explained by the common sensory innervation of the ear: the trigeminal, facial, glossopharyngeal and vagal cranial nerves and C1 – C3 spinal nerves all distribute sensory nerves into or near the tympanic membrane (McNeill 1993, Okeson 1996). In the referring of pain, convergence of sensory and nociceptive branches is even more important. All the nerves mentioned above converge into the subnucleus caudalis in the brainstem. Likewise, the deep structures of the neck (bones, muscles, joints, vessels and nerves) can be the source of referred pain. Convergence is possible, because the subnucleus caudalis reaches into the C1 - C2 region of the medulla oblongata where it communicates with the nociceptive ramii of the posterior spinal nerves (Okeson 1995). This explains pain referral into the TMJ and ear, e.g. in acute shoulder muscle trauma or chronic CSD (Fig. 2).

In the jugular neck, muscle pain referred to the ear may originate from digastric, hyoidal, sternocleidomastoid or scalenic muscles. Visceral referred pain to the ear may originate from lymphatic nodes, vagal branches of the thyroid gland (n. laryngicus inferioris), parasympathetic branches of the facial nerve of the submandibular gland, and the sympathetic plexus of the carotid artery.

Referral of muscle pain (C2 spinal nerve) in the ear (N. mandibularis)

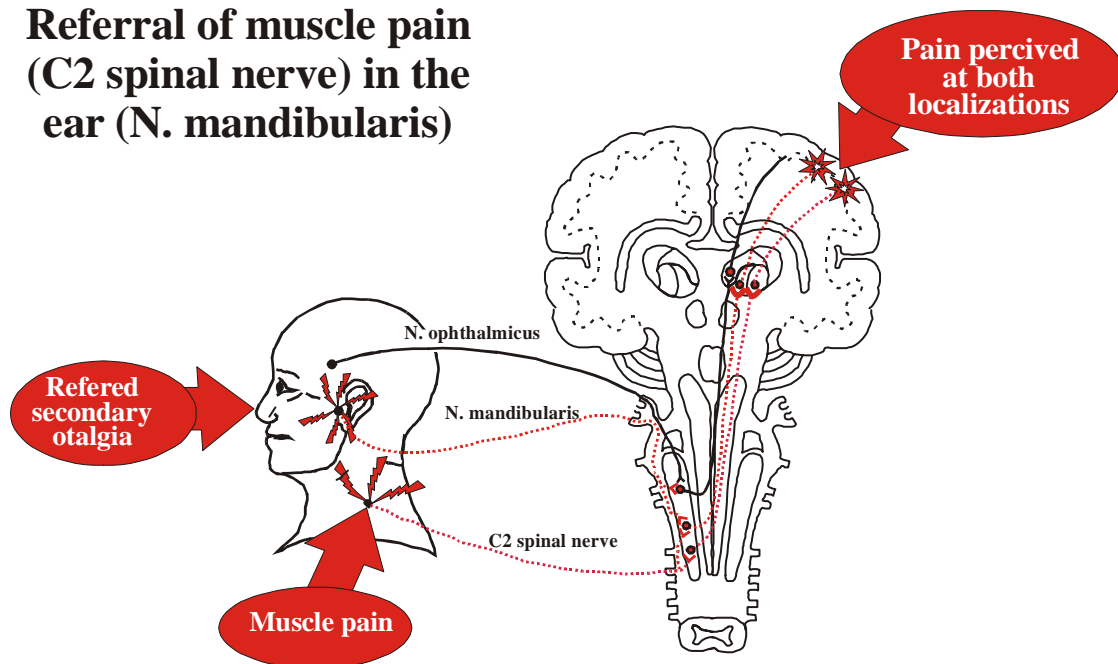


Figure 2. Schematic presentation of referred pain from trapezius muscle to the ear (adapted from Okeson. Bell's Orofacial pains. (1995)).

Table 1b. Infections or inflammatory diseases, and pain disorders or diseases associated with secondary otalgia

Infectious or inflammatory diseases associated with secondary otalgia

Viral

Primary herpetic infection
 Recurrent herpetic infection
 Acute herpes zoster

Dental

Infection
 Traumatic occlusion
 Trauma to the TMJ
 Unerupted teeth
 Impacted teeth
 Aphthous stomatitis

Nasopharyngeal

Maxillary sinusitis
 Ethmoidal sinusitis
 Nasal infection
 Pharyngeal infection
 Tonsillitis
 Peritonsillar abscess
 Post-tonsillectomy pain
 Post-adenoidectomy pain

Laryngeal

Laryngitis
 Perichondritis
 Chondritis
 Arthritis of cricoarytenoid joint

Miscellaneous

Erysipelas
 Parotiditis
 Thyroiditis
 Esophagitis

Pain disorders or diseases associated with secondary otalgia

Disorders/diseases of the neck

Whiplas injury
 Lesions of cervical spine
 Cervical radiculopathy
 Cervical arthritis
 CSD
 Carotidynia

Dental/stomatognathic

TMD
 Burning mouth
 Recent adjustment of archwires

Neuralgias

Atypical facial pain
 Trigeminal neuralgia
 Glossopharyngeal neuralgia
 Post-herpetic neuralgia
 Vagal and superior laryngeal neuralgia

Headaches

Vascular headaches
 Tension type headache
 Traction and inflammatory headache

Neoplastic disease

Carcinoma
 Sarcoma
 Lymphoma
 Metastatic disease

Miscellaneous

Hiatal hernia
 Foreign body in esophagus
 Angina pectoris
 Aneurysms of great vessel

2.2. AURAL SYMPTOMS

2.2.1. Otalgia

In 1902, Kretschman was the first to publish a patient material concerning otalgia and temporomandibular joint (TMJ) disease. In his material, 25% of patients with TMJ disease also reported otalgia. He had noticed redness in the front wall of the bony part of the external ear canal, and he concluded that arthritis of TMJ would be the cause of otalgia. He described otalgia as a sudden, blunt pain, deep in the ear that lasted from 1 to 14 days. By drawing the ear and TMJ in a horizontal plane, he pointed out the close relationship of the condyle and the middle ear.

The otologist, James Costen (1934), pointed out that the dysfunction of the temporomandibular joint could cause aural and facial pain. He also described how a flat object placed between the jaws gave marked comfort to the patient. Costen's major concern was the same as to day: secondary otalgia and facial pain were then and are now treated incorrectly as an infectious state of the ear or the maxillary sinuses. Costen's patient material comprised only 11 patients without controls. His theory that the condyle caused aural symptoms by compressing the Eustachian tube or the greater auricular nerve, aroused the most interest. Later on, his theory was proved to be false.

In the studies concerning patients with temporomandibular disorder (TMD), the occurrence of otalgia varies mainly between 40% and 90%. In the general population, the prevalence of otalgia ranges between 4% and 18% (Table 2). In the study by Conti et al. (1996), 18% of the subjects reported pain in or around the ears that had occurred sometimes. According to the studies by Agerberg and Bergenholtz (1989) and by Agerberg and Inkapööl (1990), in a population-based sample, the prevalence of pain in or near the ear was four and eight percent, respectively. However, these epidemiologic studies (Agerberg and Bergenholtz 1989, Agerberg and Inkapööl 1990, Conti et al. 1996) did not include otorhinolaryngologic examination to exclude primary otalgia.

2.2.2. Tinnitus

According to McFadden (1982), tinnitus is a conscious experience of a sound that originates in the head. Tinnitus itself is a symptom that is common to many maladies and it is also the most widespread symptom of the auditory system. It afflicts many different structures within and outside the auditory system. According to Coles (1984), there is no single condition of "tinnitus" that can be given a fixed definition and a single prevalence figure. Instead, there are various figures depending on the descriptions of the severity of the tinnitus. While many of these are not "clinical tinnitus", even the non-spontaneous or short-duration tinnitus appears to be annoying to some patients. At least 8% of patients with tinnitus report that tinnitus interferes with getting to sleep and/or makes them moderately or severely annoyed, while, 0.5% report that tinnitus severely decreases their ability to lead a normal life.

Table 2. Studies reporting prevalence of otalgia published since 1964. (int.=interview, quest= questionnaire)

Author(s)	Published (year)	(n)	Age (years)	Method	Study sample	Prevalence (%)
Dolowitz et al.	1964	66	7-56	int.	TMD-patients	85
Gelb et al.	1967	742	10-80	int.	TMD-patients	61
Bernstein et al.	1969	86	not reported	int.	TMD-patients	93
Brookes et al.	1980	45	10-79	int.	TMD-patients	82
Curtis	1980	91	31	int.	TMD-patients	99
Koskinen et al.	1980	47	not specified	int.	TMD-patients	47
Fricton et al.	1985	164	41	int.	TMD-patients	42
Agerberg and Helkimo	1987	331	11-84	quest.	TMD-patients	59
Bush	1987	105	39	quest.	TMD-patients	82
Gross et al.	1988	1109	40	int.	dental patients	6
Locker and Slade	1988	677	18 -	int.	non-patients	8
Agerberg and Bergenholz	1989	1578	44	int.	non-patients	4
Agerberg and Inkapööl	1990	637	18-65	int.	non-patients	8
Cooper and Cooper	1993	996	44	int.	TMD-patients	63
Cooper and Cooper	1993	1764	39	int.	TMD-patients	46
Ciancaglini et al.	1994	797	37	int.	TMD-patients	2
Conti et al.	1996	310	20	quest.	non-patients	18
Keersmaekers et al.	1996	400	36-39	int.	TMD-patients	42
Luz et al.	1997	843	not specified	int.	TMD-patients	11
Lam et al.	2001	776	39	quest.	TMD-patients	67

Prevalence of tinnitus in an adult population varies between 10 and 32% (Leske 1981, Coles 1984, Axelsson and Ringdahl 1989). According to Davis et al. (1992), in the adult population, one third of adults report some tinnitus, 10% report prolonged spontaneous tinnitus, 5% report prolonged spontaneous tinnitus that is moderately or severely annoying, 2-4% have been referred to hospital because of tinnitus, and 1% report that tinnitus severely decreases their quality of life. Self-report and clinical assessments of tinnitus in the general population are much in agreement.

Aetiology of tinnitus has earlier been explained, e.g. by TMD. Costen (1934) explained tinnitus by compression of the Eustachian tube. According to Myrhaug (1958), the hyperactivity of masticatory muscles could lead, by common innervation of the trigeminal nerve, to hyperactivity of the tensor tympani muscle causing tinnitus. According to Williamson (1986) and Ash and Pinto (1991), direct mechanical stimulation of the malleus via the anterior malleolar ligament could cause tinnitus. Tinnitus has also been associated with emotional disorders (Brown and Walker 1987),

somatization and psychiatric disorders (Harrop-Griffiths et al. 1987, Simpson et al. 1988). According to Coles et al. (1990), hearing problem, aging and noise exposure are the most important determinants of tinnitus in general population.

Many different pathologic changes may lead to tinnitus. It can be explained as an increase in the activity of the auditory nerve, or a decrease in the inhibitory system of hearing. The mechanisms of tinnitus in the central nervous system are not completely understood. At least some of tinnitus is of central origin (Lockwood et al. 1998). In the central auditory system, a change in neural activity may be a simple explanation of tinnitus. The change can be an abnormally enhanced activity of receptors or primary affective neurons, transient denervation hypersensitivity, accumulation of excitatory transmitters or depletion of inhibitory transmitters at a central place, or hyperactivity of resting and evoked activity in the central nuclear site. The inferior colliculus can modify the activity of the limbic system and lead to significant affective components in response to perception of tinnitus. A systematic classification of tinnitus generator and examples of pathogenetic models by Zenner (1998) is shown in Table 3.

2.2.3. Fullness of ears

Patients with fullness of ears describe it as a stuffy feeling or pressure in the ear, or a clogged or muffled sensation in the ear (Harker 1998, Cooper et Cooper 1993). In a review of the literature, six studies concerning TMD patients (Dolowitz et al. 1964, Gelb et al. 1967, Bernstein et al. 1969, Brookes et al. 1980, Curtis 1980, Cooper and Cooper 1993) reported a prevalence of fullness of ears ranging from 6 to 62%. However, no data on the prevalence of fullness of ears in the general population are available.

Fullness of ears is perceived in most infections of the middle and external ear. It can also be associated with non-infectious ear disorders like obstruction of the external ear canal, middle ear cavity or the eustachian tube, perilymphatic fistula or Meniere's disease. The cause of obstruction of the external cavity is usually cerumen, debris or foreign body. The cause of obstruction of the middle ear can be tumor or cholesteatoma. The cause of obstruction of the eustachian tube can be tumor, but also a patulous Eustachian tube can be perceived as fullness of ears. The cause of a patulous the eustachian tube can be sudden weight loss, or use of steroids or hormones.

2.2.4. Association of psychological distress with aural symptoms

No analyses concerning otalgia or fullness of ears associated with psychological distress are available. Psychological factors may play an important role in the processes of noticing, interpreting, reporting, worrying and complaining about tinnitus (Hallam 1987). In psychiatric examinations of patients with chronic tinnitus, a large share of patients fulfilled the criteria for psychiatric disorder at least once in their lives

Table 3. Tinnitus classification and a selection of pathogenetic models (Zenner 1998).

Classification of tinnitus	Pathogenetic models (examples)
OBJECTIVE TINNITUS	Glomus tumor Angiostenosis Protruding bulbus of jugular vein
SUBJECTIVE TINNITUS	
Conductive	Disturbance of tubal ventilation Middle ear myoclonia
Sensorineural tinnitus	
Type I	Hypermotility DC tinnitus Edge-effect tinnitus Regulatory disturbances of efferent nerves Noise trauma Ion channel disorders of the outer hair cells
Type II	Continuous depolarization of ion channel disorders or disturbance of the stereocilia of the inner hair cells
Type III	Release of transmitters Flooding with synaptic transmitters Swelling of the afferent nerve fibers Excitotoxic tinnitus
Type IV	Disorders of ion channels oin the stria vascularis Circulatory disorders of the cochlea Resorption disorders, osmolarity changes and hydrops of endolymf
Central tinnitus	
Primary	Brain tumors Multiple sclerosis
Secondary	Phantom tinnitus

according to the Diagnostic and Statistical Manual of Mental Disorders by the American Psychiatric Association (1980) (DSM-III). In the study by Simpson et al. (1988), the share of patients fulfilling the criteria of psychiatric disorder was 63%, while, in the study by Harrop-Griffits et al. (1987), it was 80%. Tinnitus was most frequently associated with major depression (massively pronounced depressive syndrome lasting at least 2 weeks). Among patients with complex chronic tinnitus, affective disorder was most prevalent (85%) followed by anxiety disorders and substance-related disorders (23%) (Hiller and Goebel 1998).

Seventy percent of patients with major depression reported that both tinnitus and depression had begun within the same year. The remaining 30% reported the onset of depression at a later date (Harrop-Griffits et al. 1987). In psychologically stable persons, tinnitus can be overcome without decompensation (Hiller and Goebel 1998). However, in persons with psychological vulnerability, the decompensation threshold may be crossed, leading to psychiatric disorder. Hiller and Goebel (1998) point out that psychiatric disorder must always be viewed as a function of tinnitus and a pre-existing psychological vulnerability.

2.3. TEMPOROMANDIBULAR DISORDERS

TMD is a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint (TMJ) and associated structures, or both. The most frequent presenting symptom is pain in the muscles of mastication, the preauricular area, and/or the TMJ. Patients with TMD frequently have limited or asymmetric mandibular movements and TMJ sounds. Other common related complaints of TMDs are jaw ache, earache, headache, and facial pain (McNeill 1993).

In the follow-up study of TMD treatment need by Kuttilla M. (1998), the subjects with active treatment need for TMD reported tiredness or stiffness of the mandible from 30% to 57%, and pain in TMJ or muscles in front of the ear (during mastication) from 29% to 43%. According to the clinical stomatognathic examination, tenderness on palpation of masticatory muscles varied from 87% to 100%, and on lateral palpation of the TMJ from 54% to 67%.

2.3.1. Prevalence

The earlier studies reported a high prevalence of both signs and symptoms of TMD, and also a large variance in the prevalence figures (Agerberg and Carlsson 1972, Helkimo 1979, Carlsson 1984, Rugh and Solberg 1985). According to Nilner (1992), the registration of subclinical signs and the fluctuation of signs and symptoms are two reasons for the high prevalence figures. The methodological factors can probably explain more of the variation in prevalence figures than can any real difference between samples (Carlsson and De Boever 1994). The variation in the prevalence figures is usually due to differences between samples, study designs, definitions, diagnostic criteria, or data presentation (Gross et al. 1988, Von Korff et al. 1988). The studies in the 90's have reported the prevalence of severe dysfunction to be from 1% to 3% (De Kanter 1990, Salonen et al. 1990). In population-based studies, the signs of TMD occur more frequently than the symptoms, usually in a ratio of 2:1 (Carlsson 1984).

2.3.2. Differential diagnosis

In differential diagnosis, one has to consider diseases and disorders like neuralgias (trigeminal and post-herpetic neuralgia), vascular diseases (migraine, temporal arteritis), diseases of the salivary glands and lymphatic tissue, otitis and sinusitis and tumors. One has also to remember an infected third molar, apical root infection or an impacted tooth in the differential diagnosis of TMD. As a source of orofacial pain, gingival and oral mucosal diseases, pain disorders of the tongue, burning mouth syndrome or atypical odontalgia may resemble of the symptoms of TMD.

2.3.3. Association of psychological distress with TMD

The patients with TMD may have psychological problems like stress, depression, disability and dysfunctional illness behaviour (Dworkin and LeResche 1992). They may also have difficulties in coping with increased life stress and personality characteristics causing problems in difficult life situations (Duinkerke et al. 1985, Eversole et al. 1985, Magnusson et al. 1986, Von Korff et al. 1988, Harness and Rome 1989, Southwell et al. 1990, De Leeuw et al. 1994, Rugh and Dahström 1994).

Increased stress may increase bruxism or clenching, which load the masticatory system (Okeson 1996). Stress may also modify a patient's ability to tolerate TMD pain (Zarb et al. 1994). On the other hand, chronic pain may cause stress, behavioural and emotional changes (Ecker 1984, Schwarzman and McLellan 1987), but these may also be a cause of pain (Dworkin and Burgess 1987, Rugh and Davis 1992).

2.3.4. Treatment of TMD with occlusal splint

The goals of treatment of TMD are decreased pain, decreased adverse loading, restored function, and restored normal daily activities (McNeill 1993, Okeson 1996). Treatment of TMD consists of patient education and self-care, cognitive behavioral intervention, physical therapy, orthopedic appliance therapy, pharmacotherapy, occlusal adjustment and surgery of specific articular disorders.

In clinical practice, it is commonly assumed that occlusal appliances (splints) have therapeutic value in the treatment of temporomandibular disorders (TMD). This opinion is based on several reports of a successful outcome achieved with these appliances (Greene and Laskin 1972, Dahlström et al. 1982, Okeson et al. 1982, Okeson et al. 1983, Clark 1984, Moss and Garret 1984, Sheikoleslam et al. 1986, Rubinoff et al. 1987, Wilkinson et al. 1992, Yap 1998).

According to Keersmaekers et al. (1996), 42% of TMD patients reported otalgia before treatment. One year after the treatment, 48% reported that otalgia had disappeared, and 32% reported that the intensity or the occurrence of otalgia had decreased. According to Koskinen et al. (1980), 69% of the patients with TMD reported that aural symptoms had disappeared or decreased during the treatment of

TMD. According to Bush (1987), occlusal splint treatment reduced fluctuating tinnitus but not severe tinnitus. According to Rubinstein (1993), TMD treatment together with biofeedback reduced the intensity of tinnitus in patients with severe tinnitus and TMD.

According to Forssell et al. (1999), most of these studies on occlusal splints have not been randomized, controlled trials (RCTs). They showed that scientific evidence for occlusal therapy in TMD treatment is scarce. They found only fourteen RCT studies on occlusal appliances. Many trials had clear shortcomings in study design. One study of myofascial pain patients treated with an occlusal appliance showed that the effect of a stabilization splint was non-specific, and gave no better result than a non-occluding palatal splint (Dao et al. 1994).

In two RCT studies, Ekberg et al. (1998, 2003) reported a different result. According to Ekberg et al. (1998), the difference in outcome in the study by Dao et al. (1994) may be due to different origins of pain. Ekberg et al. (1998) included TMD patients with pain of arthrogenous origin, while Dao et al. (1994) treated TMD patients with pain of myogenous origin. Later, Ekberg et al. reported good treatment outcome with stabilization splint in patients with TMD of myogenous origin (Ekberg et al. 2003). The authors conclude that the difference in treatment outcomes in these two studies may be due to different methods in selection of subjects: Ekberg et al. selected patients that had requested treatment, while, Dao et al. recruited subjects both by announcements in the local newspaper and by referrals. Also the mean of VAS scores in pretreatment examination differed: Ekberg et al. reported on average 73mm, while Dao et al. 40mm. This can mean that the study samples did not include the same type of myofascial pain patients.

2.4. CERVICAL SPINE DISORDER (CSD)

Cervical Spine Disorders consist of muscle and/or tendon disorders affecting related structures in the cervical spine and neck-shoulder region. The most common CSD are cervico-cranial syndrome, tension neck and whiplash-associated disorder. Neck pain that has lasted over six months is considered to be chronic. Patients with CSD also report neck pain referring into shoulders or arms, between scapulae and/or into the head.

According to the current opinion, neck pain originates from biomechanical overloading which causes injuries to the muscles and cervical spine. Biomechanical causes and psychosocial distress increase and extend neck symptoms. Physical factors maintaining neck pain are work requiring muscle strength, high frequency or long-lasting working, difficult or extreme working positions, too short time to recover, vibration and cold working environment (Hales and Bernard 1996). Lowered muscle strength of the shoulder region is a risk factor for neck pain in work needing high muscle strength (Jonsson et al. 1988).

2.4.1. Prevalence

Prevalence of neck pain varies from 16% to 43% (Mäkelä et al. 1991, Andersson et al. 1993, Bowim et al. 1994, Ekberg et al. 1995, Lau et al. 1996, Côté et al. 1998, Ciangaglini et al. 1999). Prevalence of disabling neck pain syndrome varies from 12% to 17% (Mäkelä et al. 1991, Andersson et al. 1993, Bowim et al. 1994). According to Ciancaglini et al. (1999), neck pain was more prevalent among subjects with temporomandibular symptomatology than without. In a follow-up study by Kirveskari et al. (1988), the functional state of the stomatognathic system was associated with mobility of the cervical spine and neck-shoulder muscle tenderness.

2.4.2. Differential diagnostics

An association of TMD with CSD has been often reported (Gelb and Tarte 1975, Rieder et al. 1983, Clark et al. 1987, Cachiotti et al. 1991, De Laat et al. 1993). According to De Wijer (1995), patients with CSD reported higher intensity, longer duration and greater impact of neck pain than the patients with TMD. Patients with CSD also reported aural symptoms less often and more often pain in the head and neck region than the patients with TMD.

Patients with temporomandibular or cervical spine disorders may have the same symptoms and differentiating between them depends on clinical examination. In the study by De Wijer (1995), CSD patients reported more often tenderness on palpation in the neck than TMD patients. Tenderness on palpation in the neck, decreased range of motion in lower parts of the cervical spine and pain in rotation of the upper part of the neck correctly classified altogether 65% of the patients with CSD (55%) or TMD (74%). According to Janda (1986), the stomatognathic system and cervical spine should be considered as a functional entity. In his opinion, the reduced function of cervical muscles and spine is a more common explanation than structural changes for referred pain and altered thresholds of reflexes in the orofacial region.

2.4.3. Association of psychological distress with CSD

Psychosocial factors in work have been associated with the occurrence of CSD (Westerling and Johnson 1980, Wallace and Buckle 1987). According to Mäkelä et al. (1991), physical and psychological distress was strongly associated with chronic CSD. According to multivariate analysis of chronic CSD, the effects of physical and psychological loading were equal and independent of each other. Subjects with mental problems had 1.4 as high a risk of CSD as the subjects without mental problems. The odds ratios for cardiovascular diseases, pulmonary diseases and diabetes were lower than that for mental problems (1.2, 1.1, and 0.9, respectively).

2.5. GENERAL HEALTH PROBLEMS

According to Aromaa et al. (1989, the Mini-Finland health survey), cardiovascular diseases are the most frequent general diseases in Finnish adults (24%) followed by musculoskeletal diseases (23%), symptoms and ill-defined diseases (15%), and metabolic diseases (6%). In the interview of the Mini-Health health survey, 58% of the subjects were chronically ill. On average, they had 2.1 chronic diseases.

2.5.1. Association of general health with aural symptoms

Secondary otalgia can occur together with infectious or neoplastic diseases in the paranasal sinuses, nose, mouth, teeth, tonsils, pharynx, larynx or thyroid gland. Musculoskeletal disorders like TMD or CSD may also present with otalgia or tinnitus. Other problems of general health that may cause referred otalgia are infectious or neoplastic diseases of neural, vascular, or lymphatic structures of the jugular neck, or oesophagus, heart, or lungs. Menière's disease consists of acute spells of tinnitus, lowered hearing and vertigo with nausea or vomiting. Tinnitus may be provoked by medications like erythromycin, aminoglycosides, cis-platinum, ethacrynic acid or furosemid. Rheumatoid arthritis, systemic lupus erythematosus, polymyositis and dermatomyositis, vasculitis, relapsing polychondritis and immunodeficiency diseases are autoimmune diseases that can present with otologic symptoms.

2.5.2. Association of general health with TMD

A higher prevalence of TMD symptoms has been found in patients with rheumatoid arthritis (Tegelberg 1987), psoriatic arthritis (Könönen 1987) and ankylosing spondylitis (Wenneberg 1983) than in the general population. Systemic joint laxity has been suggested to be a significant risk factor of TMD (Blasberg and Chalmers 1989, Westling 1992). The symptomatic TMD patients reported three or four times more other joint problems than asymptomatic subjects (Morrow et al. 1996). The patients with Menière's disease also had a much higher prevalence of signs and symptoms of TMD than the general population (Björne and Agerberg 1996).

TMD patients used more medication than controls (Wedel and Carlsson 1987). Women needed more drugs because of TMD symptoms than did men (Agerberg and Helkimo 1987). After stomatognathic treatment, the amount of prescribed medicine decreased (Kirveskari and Alanen 1984). Also the severity of signs and symptoms of TMD has been correlated with the length of sick leave, both in TMD patients and in population samples (Alanen and Kirveskari 1983, Wedel 1988). Dworkin and LeResche (1993) have estimated that 0.18 days per working adult per year were lost in the United States due to TMD disability. After occlusal adjustment of TMD, sick leaves decreased (Kirveskari and Alanen, 1984).

2.5.3. Association of general health with CSD

Musculoskeletal diseases like rheumatoid arthritis, osteitis ossificans, and fibromyalgia are frequently associated with neck pain. In cases of recent deep trauma to the neck and shoulder region, deep infection, malignant tumors, rheumatoid arthritis, compression of medulla or a nerve root, intense neck pain can be perceived and it should lead to a thorough examination of the underlying general disease.

2.5.4. Primary headache disorders

In the present study, headache refers to tension-type headache (TTH) as it is defined by the Headache Classification Committee of the International Headache Society (1988). Tension-type headache is divided into episodic and chronic type, subdivided according to whether pericranial tenderness is involved or not. TTH is bilateral or locates over the top of the cranium. Although muscle pain seems to play a part in causing TTH it does not completely explain all of it (Adams et al. 1997). According to Olesen (1991), decreased activity of the descending inhibitory system may lead to nociceptive input of myofascial and vascular structures and thus cause TTH. Moreover, limbic system activity, such as emotional stress, anxiety and depression, has been shown to have a causal relationship with TTH. Nausea and vomiting are rare with episodic TTH.

TTH is the most common type of headache. Epidemiological studies show that about 2/3 up to 3/4 of general population have experienced headache during the preceding year. TTH is most common in adolescents and middle-aged individuals, especially in women (Nikiforow 1981, Iversen et al. 1990, Rasmussen et al. 1993).

Migraine is a neuro-vascular headache with the trigeminovascular pathway mediating the nociception. It is traditionally considered to locate above the line from the eye to the ear. However, neurovascular pain may also be present in the face, jugular neck (carotidynia) or in the neck (basal migraine). According to the Headache Classification Committee of the International Headache Society (1988), migraine is classified as migraine with aura or migraine without aura.

Migraine with aura is a recurrent headache with attacks lasting from 24 to 72 hours. It is characterized as unilateral, pulsatile, of moderate or severe intensity, aggravated by physical activity, and associated with nausea, and phono- and photophobia. Neurological symptoms that may occur in migraine with aura are numbness or tingling of the lips, face and hand; slight confusion of thinking; weakness of the arm or leg; mild aphasia; dizziness and uncertainty of gait; and drowsiness. Headache, nausea and/or photophobia follow the neurological symptoms directly or after a symptom-free interval of less than an hour.

Migraine without aura is characterized as recurrent headache with neurological symptoms localized to the cerebral cortex or brain stem, developing gradually over 5 to 20 minutes and lasting less than one hour. Headache may last 4 to 72 hours, but may also be absent (Adams et al. 1997).

According to the Headache Classification Committee of the International Headache Society (1988), cluster headache occurs in patients from 20 to 50 years of age, and it is five times as prevalent in men than in women. Cluster headache consists of strictly unilateral attacks of pain orbitally, supraorbitally and/or temporally, lasting 15 to 180 minutes. Headache recurs nightly between 1 to 2 hours after onset of sleep, or several times during the night, or from once every other day to eight times per day. Pain occurs with associated autonomic signs: conjunctival injection, lacrimation, nasal congestion, rhinorrhea, forehead and facial sweating, miosis, ptosis, and eyelid oedema. Attacks occur in series lasting for weeks or months. Remission periods usually last months or years.

Chronic paroxysmal hemicrania is defined by the Headache Classification Committee of the International Headache Society (1988) as pain in the temporo-orbital region, conjunctival hyperemia, and rhinorrhea. The symptoms are much the same as those in cluster headache, but they are shorter-lasting, more frequent, mostly in women (70%), recur daily for long periods and are effectively treated with indomethacin. The majority of the cases present chronic symptoms.

In patients with TMD or CSD, several studies have reported an association with headache (Kellerhals 1984, Forssell et al. 1985, Reik 1985, Schokker et al. 1990, Zimmerman 1994, Magnusson 1995). Although an association of CSD and TMD with headache has been found, CSD and TMD have not been classified as a primary cause of headache.

2.5.5. Bruxism

Bruxism is classified as a parasomnia, which also comprises nightmares, REM-sleep behaviour disorder and sleep enuresis. According to Rugh and Harlan (1988), bruxism is considered a stress-related sleep disorder, occurring in both men and women, in children, and in adults. Sleep-related bruxism should be discriminated from bruxing or clenching while awake (Lobbezoo and Lavigne 1997). Because bruxist behaviour can be exhibited during both waking and sleeping hours, neither aspect should be ignored by the practitioner as a potential contributing factor to disturbances of the masticatory system (Attanasio 2000).

Bruxism has been suggested to be part of a sleep arousal response. Disturbances in the central dopaminergic system have also been linked to bruxism. Factors like smoking, alcohol, drugs, diseases and trauma may be involved in the bruxism aetiology. Psychological factors like stress and personality are frequently mentioned in relation to bruxism as well. In summary, bruxism appears to be mainly regulated centrally, not peripherally (Lobbezoo and Naeije 2001).

In a telephone survey by Locker and Slade (1988), the prevalence of nocturnal grinding was 18%, and habitual clenching during the day 16%. According to Cacchiotti et al. (1991), among patients seeking treatment at the UCSF Temporomandibular Disorders Clinic, the prevalence of grinding and clenching was 33% and 36%, respectively. In the epidemiologic study by Kuttilla M. (1998), the

prevalence of combined diurnal and nocturnal bruxism was 29% in the interview. According to Pow et al. (2001), tooth grinding and clenching were reported by 25% of the adult Chinese in Hong Kong. This occurred often or very often in 8% and more often at night (54%).

Differential diagnostics of sleep-related bruxism include other parasomnias like sleep-related abnormal swallowing syndrome, nocturnal paroxysmal dystonia and paroxysmal arousals. Differential diagnosis of non-sleep-related bruxism includes parafunctional jaw habits, and local and systemic myofascial pain.

2.5.6. Insomnia

Insomnia is defined as the inability to initiate or maintain sleep (Culebras 1996). The symptoms related to insomnia are being tired, being irritable, loss of motivation, poor memory, decreased concentration, vague physical manifestations, muscle pain in the morning, inability to take naps and preoccupation with getting through the night. Insomnia is often associated with general diseases like rheumatoid arthritis, osteoarthritis, gastro-duodenal ulcer, nocturnal angina, asthma, chronic obstructive pulmonary disease, cardiovascular disorders, hyperthyroidism, acromegaly, Parkinson's disease, chronic fatigue syndrome and fibromyalgia. According to Austin (1997), insomnia may be a perpetuating factor of TMD.

Women are more likely to report insomnia than men in every age group (Ford and Cooper-Patrick 2001). Sleep disturbances are powerful risk factors for the development of new episodes of major depression in the following year. Individuals who report insomnia or poor quality sleep may be at higher risk for depression throughout their lifetime (Ford and Cooper-Patrick, 2001). In the study by Goulet et al. (1995), 36% of those with jaw pain and restlessness after sleep complained of severe pain, compared with only 15% in the group with jaw pain without sleep problems.

In the report of the consensus development congress on drugs and insomnia by the National Institute of Mental Health (1984), the prevalence of insomnia was 35%. In the Nordic study of insomnia, 29% of adult Finns did reported poor sleep (Hyypä 1990). After 10 years, 75% of poorly sleeping adults still reported insomnia.

Circadian rhythm abnormalities can mimic insomnia (Barthlen and Stacy 1994). Sleep phase delay retards the appearance of sleep beyond conventional hours and can resemble insomnia. Sleep phase advance means early consumption of sleep, which can lead to early waking of the patient resembling an early morning awakening. Somatic complaints like chronic pain can prevent the entry into sleep or cause arousal in the night. Also environmental disturbances like noises, traffic, or elevated temperature can lead to insomnia.

3. AIMS OF STUDY

The hypotheses for the present study were that 1) secondary otalgia, tinnitus and fullness of ears are associated with temporomandibular disorders and cervical spine disorders and other disorders in the orofacial area, and 2) successful treatment of temporomandibular disorders should affect the occurrence of secondary otalgia.

The aims of the present study were:

- 1) to find out the prevalence figures of secondary otalgia, tinnitus and fullness of ears standardized according to age and gender
- 2) to study relationship of secondary aural symptoms with other local and general disorders
- 3) to study the efficiency of occlusal appliance therapy in TMD with active treatment need in connection with secondary otalgia
- 4) to find out the quantity of use of health care services among subjects with secondary aural symptoms.

4. SUBJECTS AND METHODS

4.1. SUBJECTS

The survey of Study sample 1 (SS1) was carried out in the municipality of Jyväskylä, Finland, starting in March 1992 and ending in December 1995. In 1992, the total number of inhabitants in the municipality was 29 272. Altogether 873 (49% of all adults) randomly selected adults born in 1927, 1937, 1947, 1957 or 1967 received a written invitation to participate in a two-year follow-up study on signs and symptoms of TMD. The stratification factors in the study were age and gender. At baseline, 59% of the adults who had received the written invitation participated in the study. The baseline population consisted of 515 subjects (♂ 246, ♀ 269, mean age 45 years). The non-participants did not differ statistically from the baseline population with regard to age or gender (358 subjects, ♂ 238, ♀ 120) (Fig. 3). The Ethics Committee of the Health Care District of Central Finland approved the study.

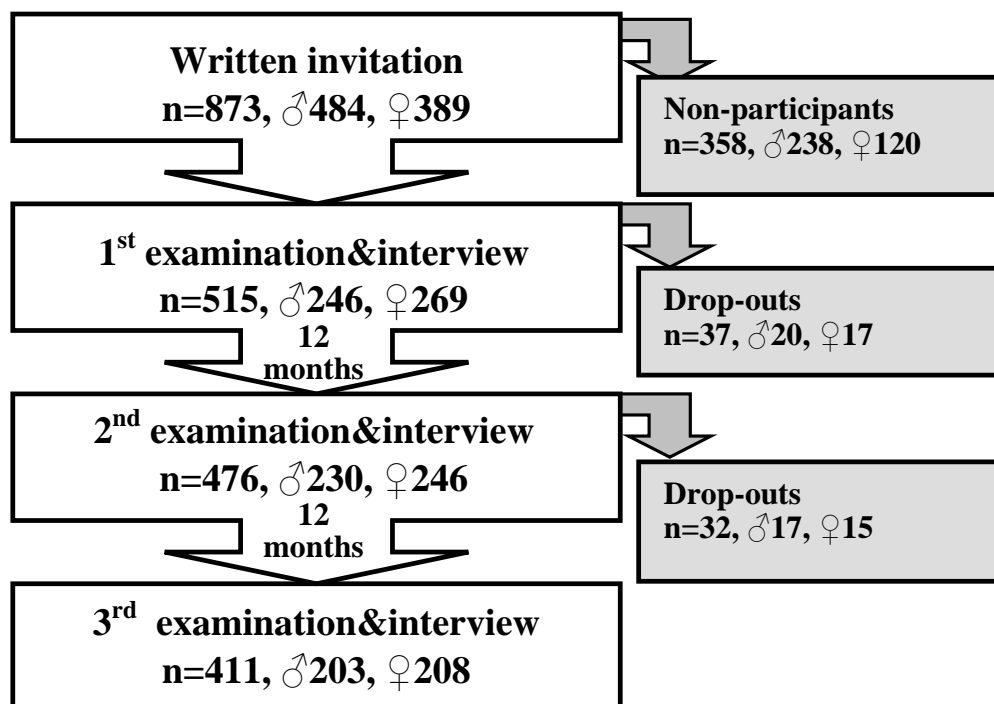


Figure 3. The flow-chart of the Study sample 1.

Studies I and IV were based on Study sample I. Study I comprised 411 subjects (♂ 203, ♀ 208) who participated in all three consecutive examinations and interviews at 12-month intervals, and were not treated for stomatognathic reasons. Study IV was based on 391 subjects (♂ 186, ♀ 205) who participated in the three examinations and interviews at 12-month intervals, completed a self-report questionnaire on symptoms of stress in the three examinations and interviews, and were not treated for stomatognathic reasons.

The survey of Study sample 2 (SS2) was carried out from March 1999 to November 2000 in Jyväskylä, Finland. The investigation comprised three phases: in the 1st phase, secondary otalgia was screened with a mailed, self-administered questionnaire (Fig. 4). A total of 2500 subjects in the age groups of 25-, 35-, 45-, 55- or 65-years were randomly selected from the database of the municipal administrative court in Jyväskylä (n= 77 867), by including every tenth name in the age group in alphabetical order, until altogether 250 men and women were selected in all age groups. The Ethics Committee of the Health Care District of Central Finland approved the study.

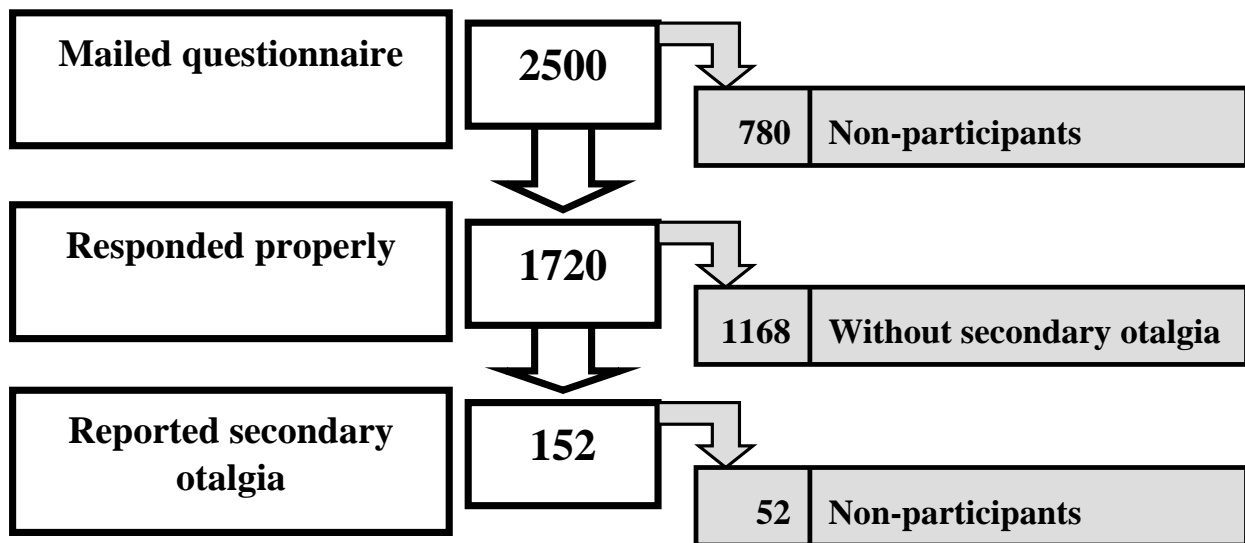


Figure 4. The flow-chart of the 1st phase of the Study sample 2.

Studies II, III and V were based on Study Sample 2. In the 1st phase of the SS2 (II), a total of 2419 subjects (97%) were approached, while 81 subjects were not approached (♂ 47, ♀ 34). Thirty-eight subjects (2%, ♂ 26, ♀ 12) returned the questionnaire unanswered and 661 subjects (26%, ♂ 392, ♀ 269) did not return it at all. Altogether 1720 (69%, ♂ 785, ♀ 935) filled in the questionnaire properly and returned it.

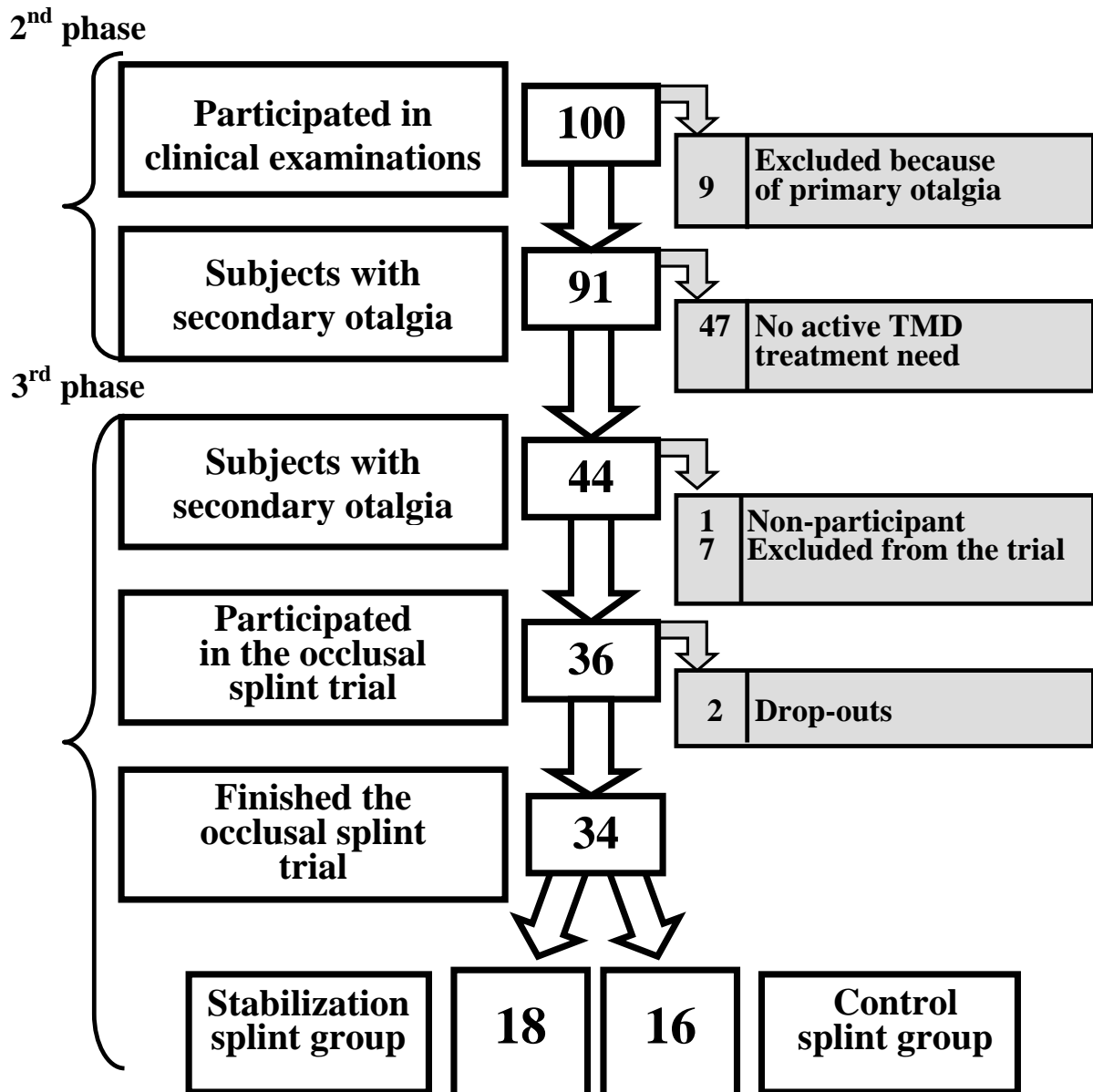


Figure 5. The flow-chart of the subjects of the 2nd and 3rd phase of the Study sample 2.

The total number of non-participants was 780 (31%, ♂ 465, ♀ 315). If the subject did not return the questionnaire, a second letter was sent four weeks later, and after that, one invitation by phone was made.

In the 2nd phase of the SS2 (III), a written description of the clinical examinations and interviews, together with an invitation to participate, was mailed to the subjects (n=152) who reported secondary otalgia in the 1st phase (9%, ♂ 63, ♀ 89, mean age 49 years) (Fig. 5). Altogether 100 subjects (66%) participated in the clinical examinations and interviews (♂ 42, ♀ 58, mean age 49 years). Informed consent was obtained from all the subjects. Non-participants consisted of 52 subjects (34%, ♂ 20, ♀ 32, mean age 48 years) who did not respond to the invitation. Of the 100 subjects who participated in the 2nd phase (III), nine had otalgia only during respiratory

infection and were excluded from further analyses because of primary otalgia. The excluded subjects consisted of seven men and two women.

In the 3rd phase of the SS2 (V), altogether 36 subjects had both secondary otalgia and active TMD treatment need. Two subjects in the control group left the study. One felt the splint was too uncomfortable and the other had continuous symptoms of respiratory infection during the follow-up period and needed medical care. Of the remaining 34 subjects, 18 (♂ 6, ♀ 12, mean age 45 years) wore a stabilization splint and 16 (♂ 3, ♀ 13, mean age 48 years) a palatal control splint. The subjects were randomly allocated by the dental assistant into the splint treatment group and the control group (Fig. 5) by randomizing the first splint type by lot and, thereafter, alternating stabilization splint and control splint treatment for the consecutive subjects. The subjects in the treatment group were treated with a stabilization splint, and the subjects in the control group with a non-occluding, thin palatal splint (Ekberg et al. 1998). Another dentist trained in stomatognathic physiology (ESN) installed and adjusted the flat stabilization splints in centric relation with canine guidance and without any mediotrusion contacts during lateral slides. Protrusion was symmetric, without molar or premolar contacts (Fig. 6). Within two weeks, the same dentist checked and readjusted the splint. The check-up was also performed in the control group.

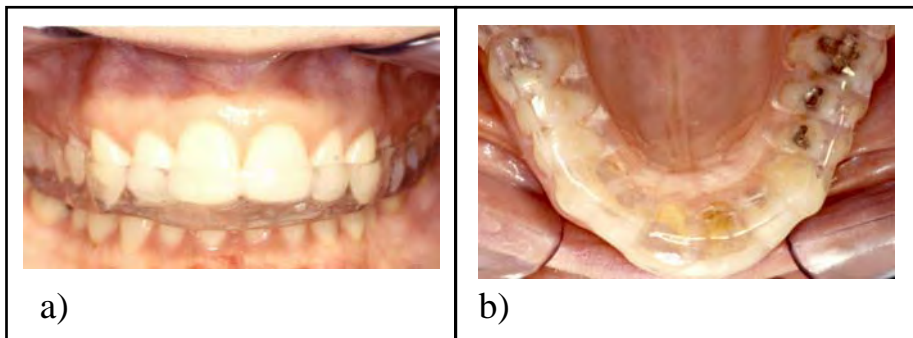
4.2. METHODS

4.2.1. Interviews

In the stomatognathic interview of the Studies I and IV, subjects were interviewed using standardized questions for symptoms related to TMD. One question was asked about whether otalgia, defined as pain in or around the ear that was not associated with infections or otitis, had occurred during the last month. The interview also included three questions about insomnia, bruxism, and recurrent head and neck region pains during the preceding month. Bruxism reported in the interview was used in the analyses. However, no clinical criteria were used. In Study IV, aural symptoms were reviewed in every interview with the question “Have you had aural symptoms not associated with infection or otitis at least once a month during the preceding six months?” The severity of aural symptoms was not estimated.

In Studies III and V, the stomatognathic interview comprised questions about earsymptoms (5 questions), clenching and bruxism (2), temporomandibular joint symptoms and trauma (8), head and neck region pain (4), facial and lingual pain (5), globus and dysphagia (2) and voice problems (1). Facial pain was defined as pain in the facial region (excluding headache, and pain in the ear or tooth). Intensity of facial pain was reported on a verbal scale from 0 to 10 (0= no pain, 10= maximal pain).

Stabilization splint viewed (a) frontally and (b) occlusally.



Control splint viewed (a) frontally and (b) occlusally.

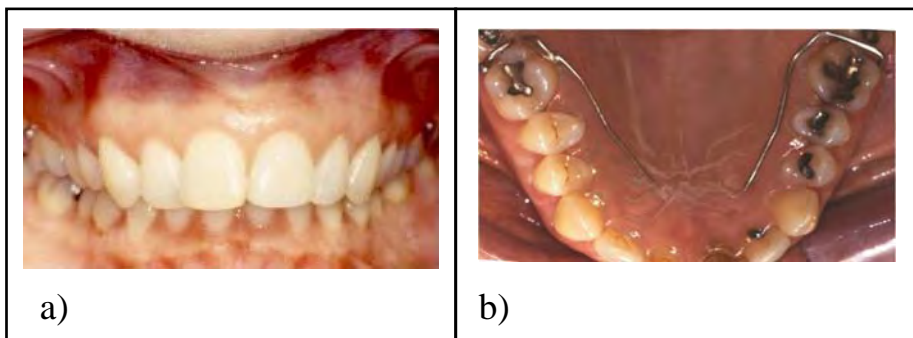


Figure 6. Stabilization and control splint.

Bruxism reported in the interview was used in the analyses. No clinical criteria for bruxism were used. The otologic interview included questions about general health and medication, whether respiratory infection preceded otalgia, physician visits and sick leaves due to otalgia, and diagnostic assessment of and therapies for otalgia. The interview about the cervical spine disorder and related symptoms comprised questions about physician visits and sick leaves due to CSD, and diagnostic assessments of and therapies for CSD.

4.2.2. Clinical examinations

A specialist in stomatognathic physiology (MK) performed all the stomatognathic examinations (I, III, IV, V). In the Studies I and IV, the temporomandibular joints were palpated laterally and posteriorly via the auditory meatus on active opening and closing movements. TMJ tenderness was recorded if palpation gave rise to a palpebral

reflex, or the subject described the palpation as painful. TMJ sounds such as clicking, reciprocal clicking and crepitus were recorded if the sound could be palpated and/or heard during maximal mouth opening and closing. TMJ sounds were also assessed with a stethoscope placed at the zygomatic arch. The loading capacity of the TMJ was tested by bimanual manipulation (Dawson 1989). The following masticatory muscles were palpated bilaterally: the anterior and posterior portion and the insertions of the temporal muscle, the superficial and the deep portions of the masseter muscle, the posterior portion of the digastric muscle, the medial pterygoid muscle and the lateral pterygoid muscle. Muscle tenderness was graded as no tenderness, tenderness if pain gave rise to a palpebral reflex, and pronounced pain, if pain gave rise to a protective reflex. The static biting test (Gottlieb et al. 1968) was performed by biting for 20 seconds on a cotton roll. The radiographic examination was based on an orthopantomogram taken from all subjects at baseline after informed consent was obtained from all subjects examined.

In the studies III and V, the clinical examination included measurement of mandibular movements, pain during guided and non-guided mandibular movements, registration of temporomandibular joint (TMJ), TMJ sounds (clicking and/or crepitation), locking, and lateral and/or posterior tenderness of the TMJ. The following muscles were palpated: the anterior and posterior temporal muscle, the attachment of the temporal muscle, the deep and superficial portion of the masseter, the medial and lateral pterygoid, and the posterior portion of the digastric muscle. The degree of muscle tenderness was evaluated according to a 4-point scale (0 = no tenderness, 1 = tenderness reported by the subject, 2 = tenderness with a palpebral reflex, 3 = withdrawal reaction).

The present author performed a standardized otolaryngologic examination. The otologic examination included inspection of ears, nose, mouth, nasopharynx, and larynx, and bilateral palpation of jugular, lateral and dorsal neck, larynx, and thyroid, submandibular and parotid glands. The examination included evaluation of maxillary sinuses with ultrasound, and evaluation of mobility of eardrums by acoustic tympanometer. The present author also performed a standardized cervical spine examination. The examination of the cervical spine consisted of guided active and passive cervical flexion, extension, flexion, and rotation with extension. Three clinical signs were recorded: 1) a rigid reduction of 50% or more in the range of cervical spine movement compared to the other side, 2) reporting pain on palpation of neck muscles (muscles in the skull base, and semispinalis capitis, trapezius, rhomboid, levator scapulae muscles) and 3) pain on guided active movements of the cervical spine (flexion, extension, flexion with rotation, rotation with extension). The exact range of movements of the cervical spine was not assessed.

4.2.3. Questionnaires

General health was assessed with an anamnesis formula of the Finnish Dental Association with questions about general health, diseases and medication (I, III, IV, V).

In the Study II, the mailed questionnaire on aural symptoms concerned about epidemiologic data (age, sex, education, occupation) and aural symptoms: frequency of secondary otalgia, tinnitus and fullness of ears; visits to a physician; TMD pain, headache, neck ache and shoulder ache. TMD pain was defined as pain or ache in the temporomandibular joint in front of the ear. In the Study III, the subjects filled in self-report questions about secondary otalgia, impact of secondary otalgia on daily living, on concentration ability at work, and on sleep, as well as twelve questions about problems related to sleep. Prolonged pain was defined as pain that has lasted for at least six months and that is located elsewhere than in the ear, e.g. in the back, stomach, neck, limbs or head. In the Study IV, after the second annual examination, a questionnaire regarding visits to a physician because of otalgia was sent to all the subjects who, at the first examination, had otalgia without infection at least once a month during the preceding six months (n=65). Of these subjects, 64 (♂14, ♀50) completed and returned the questionnaire. During the clinical follow-up, the subjects with otalgia did not know that they would retrospectively be asked about their visits to a physician.

In the Study I, the frequency of physical, behavioural, and psychological stress symptoms were assessed with the Symptoms of Stress Inventory, which is derived from the Cornell Medical Index (Beaton et al. 1978, Nakaqawa-Kogan and Betrus 1984). The reliability and validity of the Symptoms of Stress Inventory and its use as a screening instrument have been shown in both American and Finnish studies (Beaton et al. 1991, Niemi et al. 1993, Kuttilla et al. 1998). In the Study III, the participants filled in the Finnish language version of the Brief Symptom Inventory (BSI). The BSI is a shortened version of the Symptom Check List 90 (SCL-90) questionnaire (Derogatis 1977, Derogatis and Melisaratos 1983). The Finnish version of the BSI was applied and up-dated in wording according to the version used at the Social Insurance Institution in Finland. It consists of 53 questions in nine subscales (somatization, obsessive-compulsive, interpersonal sensitive, hostility, depression, anxiety, phobic anxiety, paranoid ideation, psychotism) measuring psychological distress during the previous month. The answers are graded on a 5-point scale from 0 to 4 based on the amount of perceived distress, e.g. how intense the perceived symptom was.

4.2.4. Inclusion and exclusion criteria and classification systems

In the Studies II, III and V, the inclusion criteria for secondary otalgia were pain inside or around the ear without infection, tumor or trauma; duration of at least six months; and frequency of at least once a month. The subject was considered to have secondary otalgia if he/she was aware of symptom/s meeting these criteria. The inclusion criterion for tinnitus and fullness of ears was duration of at least six months. In the Studies I and IV, inclusion criteria for secondary otalgia were pain in or around the ear that was not associated with infections or otitis, and had occurred during the last month. In the Study II, inclusion criterion for tinnitus and fullness of ears was duration of at least six months.

In the Study II, the subjects were classified into the anamnestic subgroups of secondary otalgia according to the frequency of all aural symptoms: the subjects without aural symptoms were classified into the no subgroup; the subjects with symptoms occurring less frequently than once a month into the occasional subgroup; and the subjects with symptoms occurring once a month or more often into the recurrent subgroup. The prevalences of each aural symptom were standardized according to age and gender.

In the Studies I and IV, for treatment need analyses of TMD, the classification system by Kuttilla et al. (1996) was used. Because no clear-cut criteria of TMD are available, the classification was based on anamnestic data, clinical and radiologic findings, and the clinician's judgment. The same principals have been also used by Magnusson et al. (2002). Subjects in the active TMD treatment need subgroup had moderate or severe signs and subjective symptoms of TMD, prompting them to seek help or designating them as needing care independently of other possible oral health problems (i.e. TMD alone require treatment). Subjects in the passive TMD treatment need subgroup showed some minor signs or symptoms of TMD, but were assessed as needing no stomatognathic treatment if no other dental care was considered necessary. Subjects were classified into the no TMD treatment need subgroup if TMD problems did not require treatment in any circumstances.

In the Study III, 91 subjects fulfilled the criteria for secondary otalgia used (pain inside or around the ear, occurring at least during the last six months, and absence of present or history of infection, tumor or trauma). They were classified according to signs of CSD or signs and symptoms of TMD into three groups: cervical spine disorders (CSD), temporomandibular disorders (TMD), and combined CSD and TMD (Comb). A subject was classified into the CSD group if he/she had one or more signs of CSD but no or only mild signs or symptoms of TMD. The subject was classified into the TMD group if he/she had moderate or severe signs or symptoms of TMD but no signs of CSD. The subject was classified into the Comb group if he/she had one or more signs of CSD and moderate or severe signs or symptoms of TMD. The subjects not fitting into the clinical subgroups were classified into the Not fit group (NF group) (no signs of CSD, or no or only mild signs or symptoms of TMD).

In the Study V, the subjects with secondary otalgia then classified according to Kuttilla et al. (1996) into the active TMD treatment need subgroup (n=44). The remaining 47 subjects formed another subgroup, not needing active TMD treatment. The subjects who had earlier received any treatment for their TMD, as well as the subjects with complete dentures, a history of psychiatric disorder, or symptoms of neuralgias or tooth ache (n=7) were excluded.

4.2.5. Other assessment methods

The subjects also estimated the intensity of secondary otalgia by drawing a vertical line on the visual analog scale (VAS) (0 mm-100 mm, 0 mm = no pain, 100 mm = the most intensive pain you can imagine). In the same way, the subjects also estimated the

impact of secondary otalgia on daily living (0 = not interfering, 100 = impossible to live a normal life), concentration (0 = not interfering, 100 = impossible to concentrate) and sleeping (0 = not interfering, 100 = impossible to sleep). The intensity of secondary otalgia, facial pain and the frequency of headache were estimated as before, after the 10-week' trial.

4.3. STATISTICAL METHODS

In comparisons between groups, data were analysed with χ^2 -test and Student's t-test. The χ^2 test was used for categorical variables in analyses of associations of subjects with and without secondary otalgia (I, IV), anamnestic subgroups of secondary otalgia (II), diagnostic subgroups of secondary otalgia (III), and splint therapy groups (V). Student's t-test test was used for variables on an ordinal scale in comparing means of subjects with and without secondary otalgia (I, IV), anamnestic subgroups of secondary otalgia (II), clinical subgroups of secondary otalgia (III), and splint therapy groups (V). Analysis of variance (ANOVA) was used in comparisons of age, stress-related symptoms (I) and psychological distress (III). In the within-group analyses, non-parametric χ^2 test with equal distribution expectancy was used (V). In the analyses of two consecutive assessments, the paired samples t-test, the Wilcoxon signed ranks test and the binomial test were used (V). The differences between and within groups were considered as statistically not significant if $P > 0.05$, almost significant if $.05 \leq P < .01$, significant if $.01 \leq P < .001$, and highly significant if $P \leq .001$.

Stepwise binomial logistic regression analysis was used to find independent predictors of secondary otalgia. In Study I, the variables tested were age group, gender, headache, neck ache, shoulder ache, active treatment need for TMD, total stress score over 120, bruxism, sleep problem, and general disease. In Study II, the variables tested were age group, gender, headache, neck ache, shoulder ache, TMD pain, secondary otalgia, tinnitus, fullness and visits to a physician. The tested variables were entered in the analysis as dummy variables. To calculate the final explanatory models of aural symptoms, the binomial forward stepwise logistic regression analyses were carried out with the variables identified as independent predictors of secondary otalgia together with age and gender (II).

5. RESULTS

5.1. PREVALENCE

5.1.1. Standardized prevalences of aural symptoms (II)

The standardized distributions of subjects with recurrent (\geq once a month) aural symptom among the age groups between the genders are shown in Table 4. Occasional frequencies ranged from 10% to 28%, while recurrent prevalences frequencies ranged from 6% to 15%. According to the standardized age groups, 25-year-olds reported statistically highly significantly higher frequencies of all aural symptoms than the other subjects ($P < .001$). Between men and women, statistically significant difference was found in occasional secondary otalgia (8% vs. 12%), and statistically almost significant difference in recurrent secondary otalgia (5% vs. 7%). Among the age groups, statistically almost significant differences between men and women were found in recurrent secondary otalgia in 25-year-olds (5% vs. 12%), and in occasional secondary otalgia among 55-year-olds (4% vs. 9%) and tinnitus among 25-year-olds (31% vs. 45%).

Table 4. The prevalences of recurrent aural symptoms (occurring once a month or more often) among age groups and between genders in the whole study population (n=1720, men 785, women 935). The prevalences of aural symptoms are standardized according to age and gender.

Age	Secondary otalgia			Tinnitus			Fullness		
	Men	Women	Total	Men	Women	Total	Men	Women	Total
(year)	% (n)	% (n)	% (n)	% (n)	% (n)	% (n)	% (n)	% (n)	% (n)
25	5 (7)	12 (21)	9 (28)	24 (35)	19 (32)	21 (67)	13 (18)	15 (25)	14 (43)
35	3 (5)	5 (8)	4 (13)	14 (21)	10 (17)	12 (38)	7 (10)	10 (17)	9 (27)
45	5 (7)	7 (14)	6 (21)	13 (20)	15 (30)	14 (50)	8 (12)	10 (20)	9 (32)
55	5 (8)	5 (10)	5 (18)	13 (22)	14 (28)	13 (50)	6 (10)	6 (12)	6 (22)
65	5 (8)	6 (12)	5 (20)	14 (24)	15 (30)	14 (54)	4 (7)	5 (10)	5 (17)
Total	4 (35)	7 (65)	6 (100)	16 (122)	13 (137)	15 (259)	7 (57)	9 (84)	8 (141)

5.1.2. Prevalence of aural symptoms in the two-year follow-up (IV)

In Study IV, aural symptoms were analysed without standardizing according to age and gender. During the two-year follow-up, the prevalence of otalgia varied from 12% to 16%, of tinnitus from 12% to 17%, and of fullness of ears from 5% to 9% (Table 5).

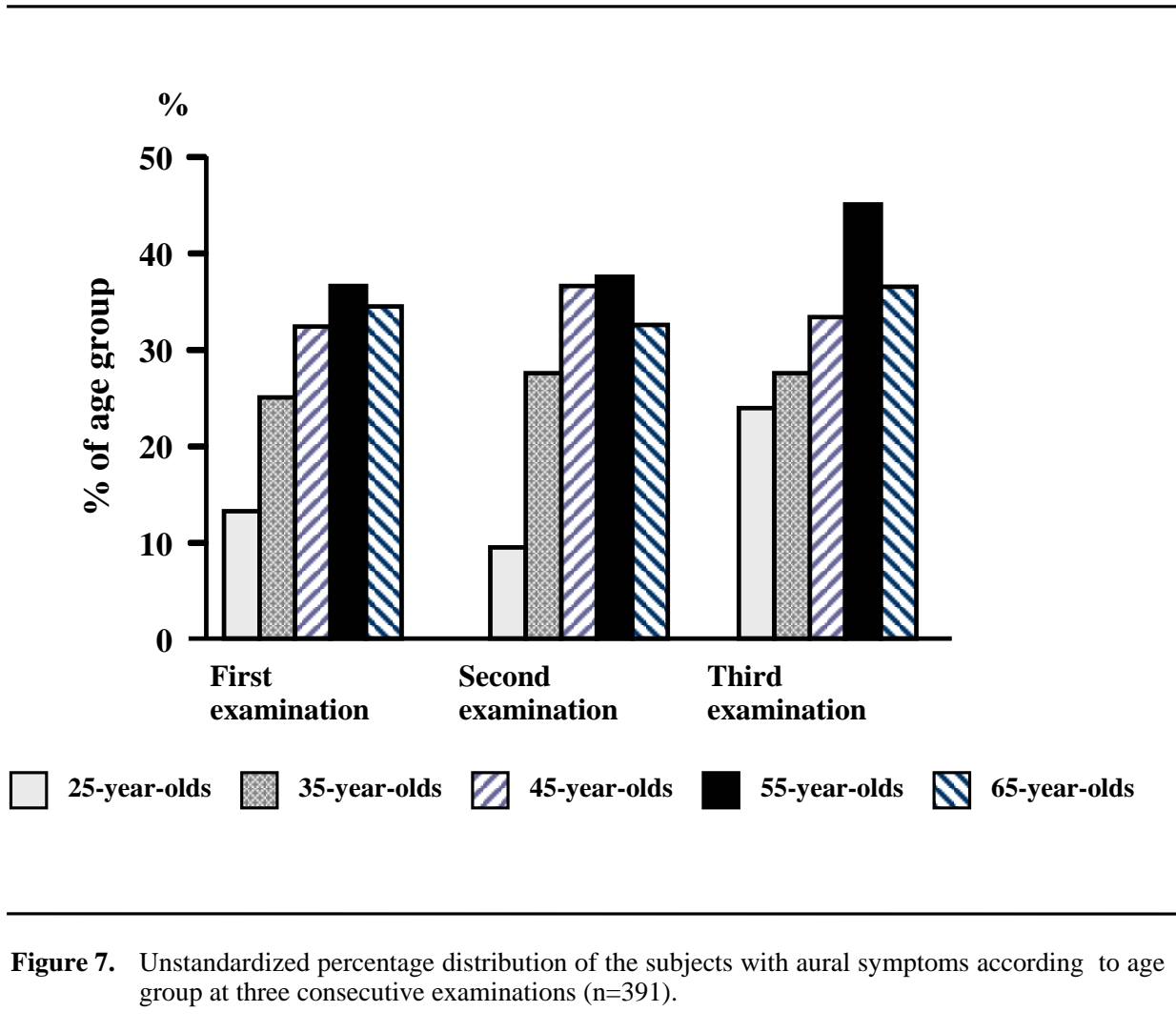
Table 5. Percentage distribution of aural symptoms and any aural symptom between genders in the study population at three consecutive examinations at 12-month intervals (n = 391).

Gender	Aural symptom							
	Secondary otalgia		Tinnitus		Fullness		Any aural symptom	
	%	(n)	%	(n)	%	(n)	%	(n)
<i>1st examination</i>								
Men	7.4	(15)	9.9	(20)	5.0	(10)	20.3	(40)
Women	23.4	(49)	17.2	(36)	5.3	(11)	33.2	(71)
Total	15.6	(64)	13.6	(56)	5.1	(21)	27.3	(111)
<i>2nd examination</i>								
Men	4.5	(9)	11.4	(23)	5.0	(10)	20.3	(40)
Women	20.1	(42)	13.4	(28)	9.6	(20)	33.6	(72)
Total	12.4	(51)	12.4	(51)	7.3	(30)	27.3	(112)
<i>3rd examination</i>								
Men	8.9	(18)	15.8	(32)	7.4	(15)	28.9	(57)
Women	16.7	(35)	18.2	(38)	10.0	(21)	34.6	(74)
Total	12.9	(53)	17.0	(70)	8.8	(36)	31.9	(131)

There was a slight tendency towards more aural symptoms with increasing age up to the age of 55 years (Fig. 7). Thereafter, the prevalence of aural symptoms decreased. Throughout the study, the subjects in the youngest age group had fewer aural symptoms than the others. The difference in the unstandardized prevalence of aural symptoms between 25-year-olds and other subjects was statistically highly

significant at the first and second examination ($P < .001$), but not significant at the third examination ($P .054$).

Women had statistically significantly more often otalgia at the first and second examination ($P < .001$), and almost significantly more often at the third examination ($P .016$) than men. A slight but not significant tendency towards the same gender difference with regard to other aural symptoms (tinnitus, fullness) was also seen.



5.2. VISITS TO PHYSICIAN

5.2.1. Anamnestic subgroups (II)

Of the anamnestic subgroups of aural symptoms, the recurrent subgroups had statistically highly significantly the highest mean number of visits to a physician during the preceding 12 months, followed by the occasional and the no subgroups. In the anamnestic subgroups of secondary otalgia, the mean of visits to a physician was

2.6 in the recurrent, 2.3 in the occasional, and 1.8 in the no subgroup (χ^2 8.5; $P < .001$). The corresponding figures in the anamnestic subgroups of tinnitus and fullness of ears were 2.4, 1.9, 1.7 (χ^2 6.7; $P .001$) and 2.6, 2.0, 1.8, (χ^2 8.0; $P < .001$), respectively.

5.2.2. Otalgia with active TMD treatment need (IV)

The subjects with active TMD treatment need and otalgia visited a physician statistically significantly more often due to aural pain than the subjects with otalgia in the other subgroups of TMD treatment need (F 19.4, $P .008$). The mean number of visits to a physician in the subgroup of active treatment need was 2½ times as high as that in the subgroup of passive treatment need, and about 14 times as high as that in the subgroup of no treatment need.

5.3 CLINICAL FINDINGS

5.3.1. Clinical groups of secondary otalgia (III)

Of the 91 subjects with secondary otalgia, 14 had no signs of CSD and no or only mild signs or symptoms of TMD (NF group). In the clinical examination of cervical spine, altogether 10 neck muscles tender to palpation, 33 stiff cervical spine movements and 33 painful cervical movements were recorded (Table 6). Altogether 35% of the subjects belonged to the CSD, 20% to the TMD, and 30% to the Combined CSD and TMD groups, while 15% belonged to the NF group (non-parametric χ^2 8.9, $P .030$). The stomatognathic signs were calculated according to Helkimo's clinical dysfunction index (1974): 29% of the subjects belonged to Di I, 43% to Di II, and 29% to Di III.

5.3.2. Tenderness on palpation of stomatognathic muscles and TM joints (IV)

In the two-year follow-up study, the subjects with aural symptoms had muscles tender to palpation 2.0-3.6 times as often as the subjects without aural symptoms (χ^2 18-50; $P < .001$). The subjects with otalgia had muscles tender to palpation from 2.4 to 2.6 times as often as the subjects without otalgia (F 26-40; $P < .001$), while the figures for the subjects with tinnitus and fullness of ears varied from 1.5 to 2.4 (F 4-8; $P .036 < .001$) and from 1.9 to 2.5 (F 9-20; $P .003 < .001$), respectively. Women had muscle and TM joint signs 2.0-2.7 times as often as men (30-41% vs. 11-21%) (χ^2 17-2; $P < .001$). The frequencies of muscle and TM joint signs were statistically significantly highest among 55-year-olds (34-42%), and lowest among 25-year-olds (7-20%) (χ^2 13-20; $P .011 < .001$).

Table 6. Distribution of subjects with neck muscles tender to palpation, stiff or painful movements of the cervical spine among the clinical groups of secondary otalgia (n=77). Also the distribution of subjects according the clinical dysfunction index (D_i) is shown. CSD= cervical spine disorder, TMD= temporomandibular disorder, Comb= combined CSD and TMD. CRFL=cervical rotation with flexion, CFLE=cervical flexion, CEXT=cervical extension, CREX= cervical rotation with extension.

	Number of muscles tender to palpation			Cervical spine movements						Clinical dysfunction index					
				Stiff			Painfull								
	0	1	2	3	CRFL	CFLE	CEXT	CREX	CRFL	CFLE	CEXT	CREX	D _i I	D _i II	D _i III
CSD	29	0	2	1	6	3	2	3	2	1	2	0	20	10	2
TMD	16	1	0	1	1	0	0	5	0	0	0	5	2	11	5
Comb	22	2	2	1	5	2	1	5	4	0	1	10	0	12	15
Total	67	3	4	3	12	5	3	13	6	1	3	15	22	33	22

5.3.3. Active TMD treatment need (IV)

The subjects with active treatment need for TMD had statistically significantly more often otalgia and tinnitus than the subjects in the other subgroups of treatment need for TMD during the two-year follow-up (Table 7). The subjects with active TMD treatment need had otalgia 1.7-3.0 times as often as the subjects with passive need, and 10.8-19.4 times as often as the subjects with no need. The subjects with active TMD treatment need had tinnitus 1.9-3.1 times as often as the subjects with passive need, and 3.9-6.9 times as often as the subjects with no need. The subjects with active TMD treatment need had fullness of ears 1.4-2.1 times as often as the subjects with passive need, and 2.0-5.1 times as often as the subjects with no need.

Table 7. Distribution of aural symptoms (secondary otalgia, tinnitus, fullness) in the TMD treatment need subgroups (active, passive, no need) at the three consecutive examinations at 12-month intervals (n = 411).

Ear symptom	TMD treatment need subgroup						χ^2	P
	Active		Passive		No need			
	%	(n)	%	(n)	%	(n)		
<i>1st examination</i>								
Any aural symptom	60.0	(18)	38.2	(73)	10.5	(20)	54.9	<.001
Secondary otalgia	40.0	(12)	23.6	(45)	3.7	(7)	43.3	<.001
Tinnitus	36.7	(11)	18.3	(35)	5.3	(10)	28.4	<.001
Fullness	13.3	(4)	6.3	(12)	2.6	(5)	7.1	.028
<i>2nd examination</i>								
Any aural symptom	68.6	(24)	36.5	(61)	12.9	(27)	59.0	<.001
Secondary otalgia	48.6	(17)	17.4	(29)	2.4	(5)	65.2	<.001
Tinnitus	31.4	(11)	16.2	(27)	6.2	(13)	21.1	<.001
Fullness	17.1	(6)	9.0	(15)	4.3	(9)	8.5	.014
<i>3rd examination</i>								
Any aural symptom	73.0	(27)	36.2	(63)	20.5	(41)	42.2	<.001
Secondary otalgia	45.9	(17)	15.5	(28)	4.1	(8)	50.2	<.001
Tinnitus	48.6	(18)	15.5	(28)	12.4	(24)	29.4	<.001
Fullness	13.5	(5)	9.9	(18)	6.7	(13)	2.4	.308

5.4. CLINICAL ASSOCIATIONS OF SECONDARY OTALGIA

5.4.1. Secondary otalgia associated with health problems (I)

The prevalence of general disease in the Study sample 1 was 44%. It was 1.4 times more prevalent in the subjects with otalgia than in those without otalgia (57% vs 43%; χ^2 4.1, P .040). The mean number of general diseases was almost two-fold in subjects with otalgia compared with those without otalgia (1.0 vs 0.6; F 12.0, P .001).

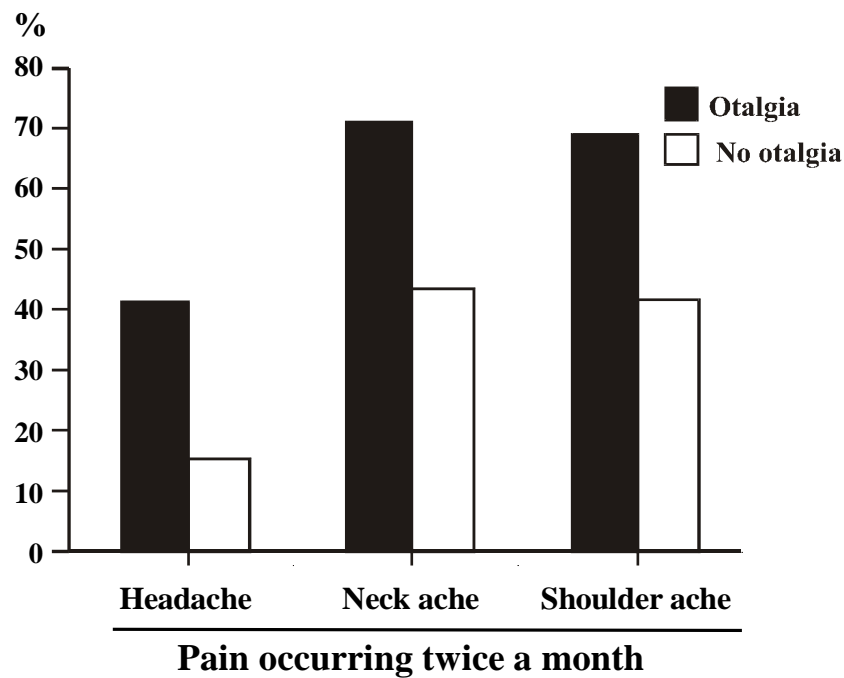


Figure 8. Percentage distribution of headache, neck ache, or shoulder ache occurring twice a month or more often among subjects with and without otalgia (n=391).

The frequencies of head and neck region pains occurring twice a month or more often were higher in the subjects with otalgia than in those without: headache was 2.7 times (χ^2 18.7; P < .001), neck pain 1.7 times (neck pain, χ^2 13.9; P < .001), and shoulder pain 1.6 times (χ^2 11.3; P .001) as prevalent (Fig. 8).

The prevalence of nocturnal bruxism was 20%; diurnal bruxism, 7%; and both types of bruxism, 4%. The subjects with otalgia reported bruxism 1.8 times as often as those without otalgia. Compared with men, women reported any type of bruxism statistically significantly more often (38% vs 23%, χ^2 10.5; P .001).

The subjects with secondary otalgia more often reported sleep problems than those without otalgia. Not falling asleep was reported 1.3 times, waking up more often than once a night 1.9 times, and waking up in pain 3.2 times as often in the subjects with otalgia as in those without otalgia (Fig. 9). The difference was highly significant in waking up more often than once a night (χ^2 13.4; $P < .001$), and almost significant in not falling asleep (χ^2 5.8; $P .020$), and waking up in pain (χ^2 6.6; $P .010$).

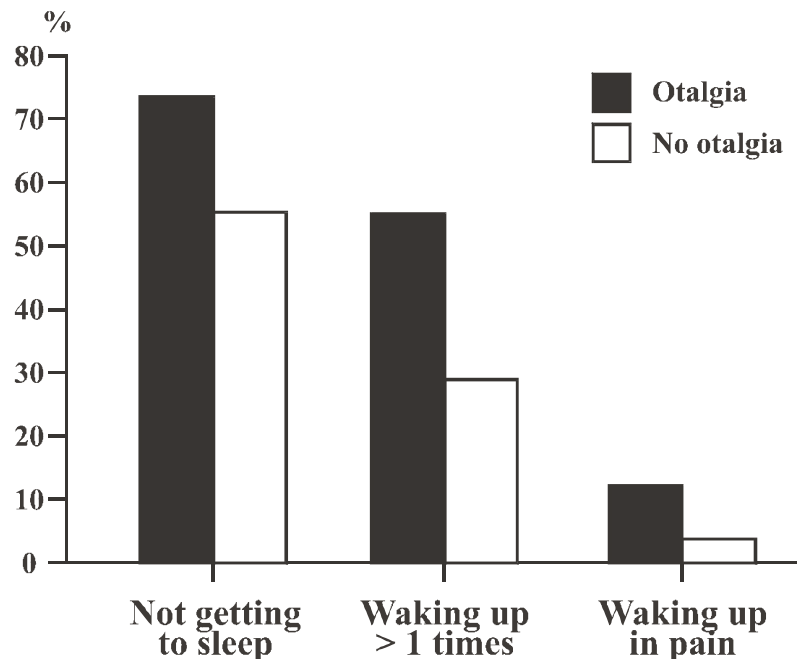


Figure 9. The percentage distribution of subjects with sleep problems among the subgroups of subjects with and without secondary otalgia (n=411).

5.4.2. Anamnestic subgroups and occurrence of associated aural symptoms (II)

An identical pattern of distribution of occurrence was seen among all combinations of associated aural symptoms: the recurrent anamnestic subgroup was most often associated with recurrent occurrence of the associated aural symptom, and least frequently associated with the occurrence of no aural symptom. All differences between the anamnestic subgroups in the occurrence of associated aural symptom were statistically highly significant (χ^2 285-442; $P < .001$).

5.4.3. Symptoms in the NF group compared with the clinical subgroups of secondary otalgia (III)

The subjects in the NF group reported statistically almost significantly less often than the others recurrent headache (14% vs. 49%; χ^2 5.9; $P .019$) and TMD pain

during the last six months (43% vs. 74%, χ^2 5.40; P .029), and statistically significantly less often shoulder ache (21% vs. 65%, χ^2 9.2; P .003) than the other subjects with secondary otalgia. The subjects in the NF group reported statistically significantly less often than the others, waking up early in the morning (57% vs. 88%, χ^2 8.4; P .004), and statistically almost significantly less often, waking up in the night because of otalgia (29% vs. 64%, χ^2 6.0; P .019). With regard to measurements on the VAS (0 mm – 100 mm), between the subjects in the NF group and the others, no statistically significant differences were found in the mean values of average (25 vs. 28) and maximal intensity (36 vs. 35) of secondary otalgia, and in the mean values of impact of secondary otalgia on daily living (14 vs. 21) and on concentration ability at work (14 vs. 21).

5.5. PSYCHOMETRIC ASSOCIATIONS

5.5.1. Symptoms of stress (SOS) Inventory (I)

In Study I, the mean of the total score of stress symptoms was 75.7 (men 71.0, women 80.1; F 5.4, P .021). The subjects with otalgia had a higher mean of the total score than those without otalgia (100.1 vs 72.3; F 20.9, P <.001). Among the subjects with otalgia, 39% had a total score higher than 120, compared with 11% of subjects without otalgia (χ^2 28.3, P <.001). The subjects with tinnitus also had a statistically highly significantly higher mean of the total score than those without tinnitus (97.9 vs 77.3; F 11.1, P .001) while the subjects with aural fullness of ears had a statistically almost significantly higher mean of the total score than those without fullness of ears (99.0 vs 79.0; F 4.0, P .046).

Women had a statistically highly significantly higher mean of the total stress score than men (87.8 vs 71.9; F 17.3, P <.001). Among the age groups, the 25-year-olds had the highest mean of the total score followed by the 65-, 45-, 55- and 35-year-olds (85 vs. 74 vs. 77 vs. 76 vs. 79, F 5.0; P <.001).

5.5.2. Brief Symptom Inventory (III)

Analyses of the Brief Symptom Inventory showed that the number of positive symptoms was statistically almost significantly lower in the NF group compared with the others (17.8 vs. 25.9) (F 4.1, P .045). When the indices of the BSI were compared, differences between the two groups were not statistically significant in the General Symptom Index (0.6 vs. 0.8), Positive Symptom Distress Index (1.3 vs. 1.4) or Positive Symptom Total (30.1 vs. 41.4). With regard to the mean values of the nine subscales of the BSI, none of the differences between the NF group and the others was statistically significant (Fig. 9). Women had a statistically almost significantly higher General Symptom Index (0.9 vs. 0.6, F 5.4; P .022) and a statistically significantly

higher Positive Symptom Distress Index (1.5 vs. 1.2, F 8.6; P .004) than men. Women also seemed to have a higher Positive Symptom Total than men, but the difference was not statistically significant (45.2 vs. 31.1, F 3.3; P .072). Among the age groups, the 45-year-olds had statistically almost significantly the highest and the 35-year-olds the lowest General Symptom Index (1.0 vs. 0.4, F 2.6; P .040), and Positive Symptom Total (54.2 vs. 18.8, 3.9; P .005).

5.6. EXPLANATORY MODELS OF AURAL SYMPTOMS

5.6.1. Explanatory model of secondary otalgia (I)

The variables of SS1 with statistically significant associations with secondary otalgia (active TMD treatment need, total stress score over 120, any type of bruxism, age of 55 years, recurrent headache, any sleep problem, female gender, recurrent neck pain, general disease, recurrent shoulder pain) were analyzed together by binomial stepwise logistic regression analysis. The variables of secondary otalgia in the final explanatory model were, active need for TMD treatment, total stress symptom score higher than 120, and bruxism (Table 8). The final model correctly classified 88% of subjects overall. However, when the logistic regression analysis was carried out separately for men and women, the final model differed. In men, the final model included recurrent neck pain, while, in women, it included active need for TMD treatment, total stress symptom score of more than 120, and the 55-year-old age group. In men, the final model correctly classified 92% of subjects, and in women, 85% of subjects.

Table 8. Final model of binomial variables predicting secondary otalgia in the subjects of SS1, who answered SOS inventory in every examination ($n=391$). The analyzed variables were active TMD treatment need, total stress score over 120, any type of bruxism, age of 55 years, recurrent headache, any sleep problem, female gender, recurrent neck pain, general disease and recurrent shoulder pain.

Variable	P	Exp(B)	95% CI for Exp(B)	
			Lower	Upper
Active TMD treatment need	.002	3.75	1.57	8.96
Total stress score over 120	.002	3.26	1.50	7.06
Any type of bruxism	.013	2.19	1.07	4.50

5.6.2. Explanatory models of recurrent aural symptoms (II)

In the step-wise logistic regression analyses of SS2, the following variables with statistically significant associations with aural symptoms were tested: age groups from 25 to 55-year-olds, female gender, secondary otalgia, tinnitus, fullness of ears, TMD pain, headache, neck ache, shoulder ache and visits to a physician. The final model of recurrent secondary otalgia included tinnitus, fullness of ears and TMD pain (Table 9). The final model of recurrent tinnitus included fullness of ears, secondary otalgia, not belonging to 65-year age group, TMD pain and headache. The final model of recurrent fullness of ears included tinnitus, secondary otalgia, TMD pain, shoulder ache and age groups of 25-, 35- and 45-year-olds.

Table 9. The final explanatory models of recurrent (\geq once a month) secondary otalgia, tinnitus and fullness of ears according to the forward stepwise logistic regression analysis. The tested variables were age group, gender, tinnitus, fullness, TMD pain, recurrent headache and shoulder ache (n=1720).

Secondary otalgia	<i>P</i>	Tinnitus	<i>P</i>	Fullness of ears	<i>P</i>
Tinnitus	<.001	Fullness of ears	<.001	Tinnitus	<.001
TMD pain	<.001	Secondary otalgia	<.001	Secondary otalgia	<.001
Fullness of ears	<.001	TMD pain	.002	Shoulder ache	<.001
		Headache	<.001	TMD pain	<.001
		25-year-olds	<.001	25-year-olds	.007
		35-year-olds	.003	35-year-olds	.011
		45-year-olds	.011	45-year-olds	.030
		55-year-olds	.045		

5.7. OCCLUSAL SPLINT TREATMENT (V)

5.7.1. Intensity and improvement of secondary otalgia

The intensity of otalgia decreased in the stabilization splint group from a VAS score of 34 to 17 (t 3.1; *P* .006), but no statistically significant decrease was seen in the control splint group (t 1.6; *P* .129). The subjects with stabilization splint reported a statistically significant improvement in their concentration ability (t 3.2; *P* .005) contrary to the subjects with control splint.

Within the stabilization splint group, 15 subjects reported improvement in secondary otalgia while three subjects reported no change or worsening of otalgia (binomial test, P .008). In the control splint group, no statistically significant difference was seen between the frequency of the subjects with (n=10) and those without (n=6) improvement in otalgia (binomial test, P .454).

5.7.2. TMD signs

The mean number of muscles tender to palpation decreased statistically significantly in the stabilization splint group (from 8.6 to 5.1), but not in the control group (from 7.8 to 5.7). The degree of tenderness on muscle palpation decreased significantly from grade 3 to 2 or less in the subjects with stabilization splint (t 3.3; 95% CI 1.3-5.8), but did not decrease in the subjects with control splint (t 2.2; 95% CI 0.1-4.2).

5.7.3. Active TMD treatment need

After 10 weeks' treatment, the number of subjects with no TMD treatment need was significantly higher in the stabilization splint group than in the control splint group (13 vs. 5, χ^2 5.71; P .017). The change from active TMD treatment need to no TMD treatment need was not associated with gender.

6. DISCUSSION

6.1. PREVALENCE OF AURAL SYMPTOMS

6.1.1. Prevalence of secondary otalgia (II, IV)

The SS2 study is the first to report prevalences of aural symptoms based on occurrence, and standardized according to age and gender. The graded aural prevalences were no, occasional (< once a month) and recurrent (\geq once a month) aural symptom. In the present study, the prevalence of recurrent secondary otalgia was assessed in a cross-sectional (II) and a longitudinal study (IV). In the cross-sectional study using a mailed questionnaire (II), the prevalence of secondary otalgia, standardized according to age and gender, was 6%. In the interviews of the longitudinal study, the prevalence of secondary otalgia varied from 12% to 16%. The raw prevalence of recurrent secondary otalgia in Study sample 2 was in line with the prevalence of Study Sample 1 before standardization (9%). However, after standardization, the prevalence decreased to 6%. Although the methods of Study sample 1 and 2 were different, interview vs. mailed questionnaire, and the criteria were slightly different (in Study sample 1 no criterion of duration was used), the main reason for differences in distribution of secondary otalgia seems to be the standardization.

Studies concerning associations between aural symptoms and TMD have mainly been patient studies. We found four studies on the general population concerning the relationship between otalgia and TMD (Locker and Slade 1988, Agerberg and Bergenholz 1989, Agerberg and Inkapööl 1990, Conti et al. 1996). In these studies, the prevalence of otalgia varied between 4 and 18%, which is in line with results of the present studies. However, none of these studies reported the duration or frequency of otalgia.

In the anamnestic subgroups of secondary otalgia (II), no statistically significant differences were found in the distribution of men and women among the age groups. This disagrees with the study by Locker and Slade (1988), who found that women were more likely to report pain in the ears than men. The highest prevalence for recurrent secondary otalgia was found among the age group of 25-year-olds, which disagrees with the results of the study by Agerberg and Bergenholtz (1989). According to Agerberg and Bergenholtz (1989), the highest prevalence of otalgia was among the group of 65-year-olds in both genders. The disagreement between these studies may result from differences in study methods and selection of the subjects.

During the two-year follow-up (IV), prevalence of secondary otalgia varied from 12 to 16%, which is in accordance with the earlier patient studies reporting otalgia (Curtis 1980, Koskinen et al. 1980, Cooper and Cooper 1993, De Wijer 1995, Keersmaekers et al. 1996). The subjects with active TMD treatment need reported otalgia in 40 - 49%, while the subjects with no treatment need for temporomandibular disorders had otalgia only on the level of 2 - 4%. This result is in accordance with the study by Keersmaekers et al. (1996) showing that 40% of the patients with TMD reported otalgia.

6.1.2. Prevalence of tinnitus (II, IV)

In the SS2, the standardized prevalence of recurrent tinnitus (once a month or more often) was 15%. In the interviews of the longitudinal study, the prevalence of tinnitus varied from 12% to 17%, which is well in line with the prevalence in SS2. The prevalence of tinnitus in an adult population has varied between 10 to 32% (Leske 1981, Coles 1984, Axelsson and Ringdahl 1989, Pilgramm et al. 1999). In these studies, the prevalences of tinnitus were not standardized according to age or gender, and were not based on the occurrence of tinnitus. The frequency of unrestricted type and duration of tinnitus is about 30-38% (Coles 1984, Axelsson and Ringdahl 1989). In Study II, the prevalence of recurrent tinnitus (15%) is in line with the prevalence of spontaneous and prolonged tinnitus (10%, Coles 1984), and of tinnitus occurring often or always (15%, Axelsson and Ringdahl 1989). According to the earlier epidemiologic surveys on tinnitus, no single prevalence figure can be given (Coles 1984). Different definitions of tinnitus have led to diversity in prevalence figures. Consequently, comparing the prevalences of tinnitus across studies and nationalities is difficult. However, prevalences based on frequency are commonly used in the epidemiologic studies, e.g. recurring and chronic pain conditions. In the future, prevalences of aural symptoms based on occurrence could be one possibility to create comparable prevalence studies.

In Study II, the prevalence of tinnitus was quite even in men and women over 50 years of age: occasional tinnitus was, among men, 26% and, among women, 27%, while both genders reported recurrent tinnitus in 27%. Epidemiologic studies on tinnitus usually report women perceiving more tinnitus than men in all age groups (Leske 1981, Axelsson and Ringdahl 1987, Office of Population Censuses and Surveys 1983), or up to 50 years of age (Axelsson and Ringdahl 1989). In Study II, a similar gender difference was not seen. One reason for this could be that we used a questionnaire while the two earlier studies used an interview (Axelsson and Ringdahl 1987, OPCS 1983). Among subjects over 50 years of age, our study is not in accordance with the study by Axelsson and Ringdahl (1989), who reported that men more often reported tinnitus than women. In Study II, aural symptoms were more often seen in the younger age groups, with the age group of 25-year-olds showing the highest prevalence figures. This is in disagreement with the study by Davis (1992) reporting the maximum prevalence of tinnitus at 61 - 70 years of age among adults in Great Britain.

The prevalence of tinnitus varied from 12 to 17% in the follow-up Study IV, which is in line with the figures published earlier (Leske 1981, Coles 1984, Axelsson and Ringdahl 1989) and with the figures from Study II.

6.1.3. Prevalence of fullness of ears (II, IV)

In Study II, the prevalence of recurrent fullness of ears was 8% and occasional 28%, which are within the range of 6-62% reported by the studies concerning TMD patients (Costen 1934, Dolowitz et al. 1964, Bernstein et al. 1969, Brookes et al. 1980, Koskinen et al. 1980,). The disagreement between the studies mentioned above and Study II may result from differences in study methods (questionnaire vs. interview, inclusion criterion (none vs. duration of 6 months) and selection of subjects (adults vs. patients seeking treatment). The figures of fullness of ears in the follow-up study

6.2. VISITS TO PHYSICIAN

6.2.1. Anamnestic subgroups (II)

Among the anamnestic subgroups of all aural symptoms, the subjects with recurrent aural symptoms reported the highest number of visits to a physician during the preceding 12 months. This is in line with the study of OPCS Monitor (1983): 19% of the subjects who were bothered a great deal by tinnitus had visited a physician due to tinnitus during the preceding month. In Study II, visits to a physician due to tinnitus were not asked about. However, one in two of the subjects with occasional or recurrent tinnitus had visited a physician at least once during the preceding 12 months.

6.2.2. Otolgia with active TMD treatment need (IV)

The treatment-seeking of patients with TMD and aural symptoms is sometimes misleading: the subjects with active TMD treatment need reported a 10 times as high number of physician visits due to otalgia as the subjects with no TMD treatment need. Our experience is that treatment-seeking depends on the location of the referred pain. Moreover, if the cause of pain referral is not diagnosed, the result is more unnecessary visits to a physician (Glass and Glaros 1995). Also total costs usually increase because initial examinations are more expensive when provided by a general practitioner rather than by a dentist (Glass and Glaros 1995). Thus, after ruling out primary otalgia, the patients with secondary otalgia should be referred to a dentist with stomatognathic experience to rule out stomatognathic causes of aural symptoms. In this way, the number of unnecessary visits to a physician and unnecessary costs would be reduced.

6.3. CLINICAL FINDINGS

6.3.1. Clinical groups of secondary otalgia (III)

In Study III, the share of the CSD group was 35%, of TMD 20%, and of Combined CSD with TMD 30%. Visscher et al. (2000) reported that the percentage of patients with both CSD and TMD would possibly be lower using the dynamic/static tests in the examination of the cervical spine. In Study III, the clinical examination did not include dynamic or static tests, which, according to Visscher et al. (2000), could lead to increased overlapping of CSD with TMD. Moreover, the use of pain reporting without a withdrawal reflex as the criterion of cervical spine muscle and/or joint pain may also have led to increased overlapping. On the other hand, the overlapping of TMD with CSD was minimized because the diagnostic criteria of TMD were quite strict: only a withdrawal reflex together with pain reporting was recorded as a positive sign. This may result in diagnosing relatively fewer subjects with active TMD treatment need compared with the not so strict criteria of CSD.

6.3.2. Tenderness on palpation of stomatognathic muscles and TM joint (IV)

In Study IV, the subjects with aural symptoms had more often muscles tender to palpation and tenderness of the temporomandibular joint. This is in line with the results of Keersmaekers et al. (1996), and with the findings of McNeill (1993) and Okeson (1996), who report that the cause of otalgia in patients with secondary TMD is referred pain from the masticatory muscles or TM joints. This is also in line with the study by Rubinstein (1993) reporting that the number of subjects with pain at palpation of stomatognathic muscles proved to be significantly higher in a population with tinnitus than in population without tinnitus.

6.3.3. Active TMD treatment need (IV)

Men and women in the active TMD treatment need subgroup reported aural symptoms with equal frequencies (IV). This is surprising because, throughout the follow-up, women more often reported aural symptoms than men. It seems that active TMD treatment need equalizes the difference between men and women in reporting aural symptoms. This can also mean that, men and women are equally symptomatic in the active TMD treatment need subgroup. This is controversial because, according to previous studies, women have head and neck region pain more often than men (LeResche 2000).

The share of subjects with active treatment need for TMD reporting any of the aural symptoms was quite high, 60-73%, during the two-year follow-up. The highest frequency of aural symptoms was found in subjects with otalgia, 40-49%, which is in line with the TMD patient studies (Koskinen et al. 1980 (47%), Friction et al. 1985

(42%), Cooper and Cooper 1993 (46%), Keersmaekers et al. 1996 (42%)). In subjects with otalgia, also the occurrence of tinnitus was more than double (31-49%) compared to the frequency reported in the whole study sample (12-17%). The frequency of tinnitus in the active TMD treatment need subgroup is also in line with TMD patient studies reporting tinnitus (Gelb et al. 1967 (40%), Bernstein et al. 1969 (42%), Cooper et al. 1986 (36%), Bush 1987 (33%), Cooper and Cooper 1993 (53%), Keersmaekers et al. 1996 (36%)).

6.4. CLINICAL ASSOCIATIONS OF SECONDARY OTALGIA

6.4.1. General health, pain and sleep problems (I, III)

The subjects in Study sample 1 were as healthy as Finns in general (I). According to Aromaa (1989), cardiovascular diseases are the most frequent general disease in Finnish adults (24%), followed by general arthrosis (10%), diabetes (6%) and pulmonary diseases (5%). The reported prevalences of general disease in SS1 were in line with the study by Aromaa et al. (1989): cardiovascular diseases 19%, general arthrosis 3%, diabetes 4% and pulmonary diseases 7%.

The reported prevalences of recurrent neck pain were in line with the studies by Aromaa et al. (1989), Takala et al. (1982) and Mäkelä et al. (1991). The prevalence of recurrent headache in the present study was lower than that reported by Sillanpää (1983) and Honkasalo et al. (1995). The prevalence of recurrent shoulder pain in the present study was higher than reported by Takala et al. (1982) and Mäkelä et al. (1991). However, in Swedish studies by Westerling and Jonsson (1980) and Ekberg et al. (1995), prevalences of shoulder symptoms were much more in line with our study.

In the present study, the subjects with otalgia reported sleep problems more often than those without. According to the study by Fricton et al. (1985), sleep problems are associated with myofascial pain in TMD patients. Consequently, sleep problems may also increase the intensity, duration, frequency or handicap of secondary otalgia. In Study I, 58% of subjects reported some sleeping problem, which is higher than the 35% in the study by the National Institute of Mental Health (1984). However, it is in line with the figures of TMD patient studies by Fricton et al. (1985), 42%, and Harness et al. (1990), 61%. It seems obvious that, in patients with secondary otalgia, the amount and quality of sleep should also be evaluated and treated along with other symptoms.

In Study I, the prevalence figures for bruxism were based on interview and not on clinical examination. Our figures may underestimate the prevalence of bruxism because bruxists are not always aware of the parafunction. This underestimation does not, however, necessarily affect the revealed association between secondary otalgia and bruxism. The total prevalence of bruxism in our study is almost the same as in the study by Goulet et al. (1995), 31% and 26%, respectively.

6.4.2. Symptoms in the NF group compared with the clinical groups (III)

The subjects with secondary otalgia but without CSD or TMD (15%) reported the same level of intensity and impact of otalgia and psychological distress as the other subjects with secondary otalgia but less often head and neck region pain and sleep-related symptoms. A better coping ability may explain the absent signs or symptoms of CSD or absent or only mild signs or symptoms of TMD in the NF group.

6.5. PSYCHOMETRIC ASSOCIATIONS

6.5.1. Symptoms of Stress Inventory (I)

In Study I, a total of 39% of subjects with otalgia had a total stress symptom score of 120 or higher, which is in line with the frequency of 32-49% in a follow-up study of an adult population by Kuttilla M. (1998). Elevated level of stress could explain why individuals with an elevated level of stress symptoms experience otalgia more often than those with a lower stress level.

6.5.2. Brief Symptom Inventory (III)

In the analyses of symptoms of psychological distress, the subjects in the NF group did not differ in reporting psychological distress compared with the others. This can be a result of a too small sample or of coping well with secondary otalgia occurring once a month or more often. When compared with the results of an American study reporting data of non-patients ((Derogatis and Melisaratos 1983)), the means of the dimensions and indices were higher in the present study. This can indicate differences between the two cultures in expressing psychological distress.

6.6. EXPLANATORY MODELS OF AURAL SYMPTOMS (I, III)

In Study I, the explanatory model of secondary otalgia in the whole study sample comprised active need for TMD treatment, a total stress symptom score of more than 120, and bruxism. The final model is in accordance with the studies by Kuttilla et al. (1998) and common clinical experience. This means that TMD treatment need, elevated stress level and bruxism independently contribute to secondary otalgia. Furthermore, if the cause of otalgia is not found in a clinical otological examination, referral to a dentist with experience in stomatognathic diagnostics is recommended to rule out or to treat possible TMD and bruxism. Stress management is also recommended. In women with secondary otalgia, the final model included elevated

frequency of stress symptoms, active need for TMD treatment, and 55-year-old age group. In men with secondary otalgia, recurrent neck pain was the only variable in the final model. Consequently, diagnosing secondary otalgia may require a different approach in men and in women.

In Study II, the final model of recurrent aural symptoms included other aural symptoms, temporomandibular disorder (TMD) pain, headache and shoulder ache, and the age groups 25- 55-year-olds. According to clinical experience, head and neck region pains were expected to be one of the significant predictors of aural symptoms, while the co-existence of otalgia with tinnitus or fullness of ears was unexpected. This result can mean that further consultations would help to diagnose the underlying disorders of secondary otalgia, and the patients with secondary aural symptoms may be referred for adequate treatment even before the head and neck region pains force them to visit a physician.

6.7. OCCLUSAL SPLINT TREATMENT (V)

6.7.1. Intensity and improvement of secondary otalgia

The treatment with a stabilization splint showed a positive outcome in both secondary otalgia and TMD treatment need. With regard to otalgia, the decrease was significant within the groups but not between the groups. This may be due to the relatively low intensity of the secondary otalgia at baseline. In our opinion, the improvement in otalgia in Study V may indicate that pain in the auricular area is often referred pain from the temporomandibular joint and associated structures.

6.7.2. Active TMD treatment need

The positive outcome of subjects treated with a stabilization splint indicates that the use of a stabilization splint is beneficial with regard to secondary otalgia and active TMD treatment need. This is in line with the study by Keersmakers et al. (1996) although Keersmakers et al. studied TMD patients seeking treatment and Study V investigated subjects with secondary otalgia and an active TMD treatment need.

7. CONCLUSIONS

1. Younger subjects more often report recurrent secondary otalgia, tinnitus and fullness of ears than older ones.
2. The subjects with secondary otalgia have a lowered general health, problems of sleeping, or pain in the head and neck region. Clinical findings and symptoms of cervical spine and temporomandibular disorders can explain secondary otalgia in 85% of the subjects. The final models of the aural symptoms assessed by a questionnaire, included other aural symptoms, temporomandibular disorder pain, headache and shoulder ache, and the age groups from 25- to 55-year-olds. Women and men may need a different approach in diagnostics of secondary otalgia because, in men, the final model included only neck pain while, in women, the final model included active TMD treatment need, elevated stress level and age of 55 years explain secondary otalgia.
3. Secondary otalgia can be alleviated with the use of stabilization splint. Also the need for an active treatment of temporomandibular disorders decreased during the ten-week therapy with a stabilization splint.
4. Recurrent aural symptoms are associated with frequent visits to a physician. The subjects with secondary otalgia and active TMD treatment need frequently visit a physician due to otalgia.

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A handwritten signature in black ink, appearing to read "Pertti Kurki". The signature is written in a cursive, flowing style with a long horizontal stroke at the end.

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11. APPENDIX

11.1 QUESTIONNAIRES

The questionnaire on secondary otalgia and seeking treatment (IV)

1. How many times have you visited a physician because of otalgia?
2. Have you, because of otalgia, had massage, physiotherapy, neural pathway massage?
3. Have you visited a dentist to get stomatognathic treatment because of otalgia?
4. Have you, because of otalgia, had occlusal adjustment, occlusal appliance, your prosthesis fixed?
5. Have you, because of otalgia, got a prescription for ear drops, pain killers, antibiotics, Triptyl, Saroten, Doxal or Limbitrol, Somadril comp, Baklofen, Norflex, Robaxin or Dolan

QUESTIONS ABOUT AURAL SYMPTOMS

The mailed questionnaire on secondary otalgia (II, III and V).

1. How often have you perceived aural pain during the preceding six months?
2. How intense has your aural pain been at its worst?
3. Has aural pain decreased your performance in daily activities and work?
4. Has aural pain made you feel blue?
5. Have you had tinnitus in the ear or in the head, e.g. buzzing, whistling, wheezing or hissing without any external source of the sound?
6. How intense has your tinnitus been at its worst?
7. Has tinnitus decreased your performance in daily activities and work?
8. Has tinnitus made you feel blue?
9. How often have you had fullness of ears?
10. How intense has your fullness of ears been at its worst?
11. Has fullness of ears decreased your performance in daily activities and work?
12. How many times altogether have you visited a physician during the last year? (take into account all visits despite different causes of visits and specialties of physicians)
13. Have you had headache?
14. Have you had neck ache?
15. Have you had shoulder ache?
16. Have you had at least once during the preceding six months pain or ache in the masticatory muscles or temporomandibular joint in front of or inside of the ear without any diagnosed infection?
17. Have you had, during the preceding 10 years, an accident or trauma to the head, neck or skull?

The questionnaire on secondary otalgia, sleeping and chronic pain (II)

The following questions 1. – 3. apply to the preceding month.

1. How intense has your strongest aural pain been? Mark a cross on the line below at the most appropriate place. (VAS 0-100mm)
2. How intense has your aural pain on average been? Mark a cross on the line below at the most appropriate place. (VAS 0-100mm)
3. How big is the biggest trouble that your aural pain has caused? Mark a cross on the line below at the most appropriate place. (VAS 0-100mm)

The following questions 4. – 5. apply to the preceding six months.

4. How much has your aural pain, on average, disturbed your concentration?
5. How much has aural pain disturbed your sleeping?

Please, circle only the most appropriate alternative in each question.

6. Have you perceived otalgia ? in the right ear, in the left ear, in both ears
7. In addition to otalgia, have you perceived any kind of other pain that has been going on for at least six months and has been located, e.g. in back, in the stomach, in the neck, in the extremities or in the head?
8. How many nights during a week do you not get to sleep?
9. How many times a week at most do you awake up from sleep?
10. Do you wake up in the morning with headache?
11. Do you wake up in the morning with otalgia?
12. Do you wake up in the morning with neck pain?
13. Do you wake up in the night with otalgia?
14. Do you wake up in the night with neck pain?
15. How many hours do you sleep on average in the night?
16. Do you usually wake up feeling satisfied?
17. Do you usually wake up feeling powerful?
18. Are you usually alert during the day?
19. Do you take sleeping pills to get to sleep?
20. Do you sleep in the day time?
21. Do you wake up in the small hours?
22. Is your sleep restless?
23. Are you very tired during the day?

Symptoms of Stress inventory (I)

This questionnaire is designed to measure the different ways people respond to stressful situations. In the book are sets of questions dealing with various physical, psychological and behavioural responses. We are particularly interested in the *frequency* with which you may have experienced these stress related symptoms *during the past month*. **Please circle the most appropriate response to each question!** (0=no, 1=seldom, 2=sometimes, 3=often, 4= very often).

Sometimes people under stress experience a variety of physical responses. During the preceding month have you been bothered by:

1. Flushing of your face	0	1	2	3	4
2. Sweating excessively even in cold weather.....	0	1	2	3	4
3. Severe itching.....	0	1	2	3	4
4. Skin rashes.....	0	1	2	3	4
5. Breaking out in cold sweats.....	0	1	2	3	4
6. Cold hands or feet.....	0	1	2	3	4
7. Hot or cold spells.....	0	1	2	3	4

Have you noticed any of the following symptoms when not exercising:

8. Pains in your heart or chest.....	0	1	2	3	4
9. Thumping of your heart.....	0	1	2	3	4
10. Rapid or racing heart beat.....	0	1	2	3	4
11. Irregular heart beats.....	0	1	2	3	4
12. Rapid breathing.....	0	1	2	3	4
13. Difficult breathing.....	0	1	2	3	4

Have you experienced:

14. A dry mouth.....	0	1	2	3	4
15. Having to clear your throat often.....	0	1	2	3	4
16. A choking lump in your throat.....	0	1	2	3	4
17. Hoarseness.....	0	1	2	3	4
18. Nasal stuffiness.....	0	1	2	3	4
19. Colds.....	0	1	2	3	4
20. Colds with complications (e.g. bronchitis).....	0	1	2	3	4
21. Increased asthma attacks.....	0	1	2	3	4

Have you experienced:

22. Spells of severe dizziness.....	0	1	2	3	4
23. Feeling faint.....	0	1	2	3	4
24. Blurring of your vision.....	0	1	2	3	4
25. Migraine headaches.....	0	1	2	3	4
26. Tension headaches.....	0	1	2	3	4
27. Sinus headaches.....	0	1	2	3	4
28. Increased seizures (convulsions).....	0	1	2	3	4

Have you been bothered by:

29. Indigestion.....	0	1	2	3	4
30. Nausea.....	0	1	2	3	4
31. Severe pains in your stomach.....	0	1	2	3	4
32. Increased appetite.....	0	1	2	3	4
33. Poor appetite.....	0	1	2	3	4
34. Loose bowel movements or diarrhoea.....	0	1	2	3	4
35. Heartburn.....	0	1	2	3	4
36. Constipation.....	0	1	2	3	4

Muscle tension is a common way of experiencing stress. Have you noticed excessive tension, stiffness, soreness or cramping of the muscles in your:

37. Neck.....	0	1	2	3	4
38. Jaw.....	0	1	2	3	4
39. Forehead.....	0	1	2	3	4
40. Eyes.....	0	1	2	3	4
41. Back.....	0	1	2	3	4
42. Shoulders.....	0	1	2	3	4
43. Hands or arms.....	0	1	2	3	4
44. Legs.....	0	1	2	3	4
45. Abdomen or stomach.....	0	1	2	3	4

In your day-to-day activities, have you noticed symptoms of anxiety or restlessness, such as:

46. Fidgeting with your hands.....	0	1	2	3	4
47. Pacing.....	0	1	2	3	4
48. Chewing your lips.....	0	1	2	3	4
49. Difficulty sitting still.....	0	1	2	3	4
50. Increased eating.....	0	1	2	3	4
51. Increased smoking.....	0	1	2	3	4
52. Biting your nails.....	0	1	2	3	4
53. Having to urinate frequently.....	0	1	2	3	4
54. Having to get up at night to urinate.....	0	1	2	3	4
55. Difficulty in falling asleep.....	0	1	2	3	4
56. Difficulty in staying asleep at night.....	0	1	2	3	4
57. Early morning awakening.....	0	1	2	3	4
58. Changes in your sexual relationship.....	0	1	2	3	4

Have you noticed

59. Worrying about your health.....	0	1	2	3	4
60. Stuttering or stammering.....	0	1	2	3	4
61. Shaking or trembling.....	0	1	2	3	4
62. Being keyed up and jittery.....	0	1	2	3	4
63. Feeling weak and faint.....	0	1	2	3	4
64. Frightening dreams.....	0	1	2	3	4
65. Being uneasy and apprehensive.....	0	1	2	3	4

Stress is often accompanied by a variety of emotions. During the preceding month have you felt:

66. Alone and sad.....	0	1	2	3	4
67. Unhappy and depressed.....	0	1	2	3	4
68. Like crying easily.....	0	1	2	3	4
69. Like life is entirely hopeless.....	0	1	2	3	4
70. That you wished you were dead.....	0	1	2	3	4
71. That worrying gets you down.....	0	1	2	3	4

Does it seem:

72. That little things get on your nerves.....	0	1	2	3	4
73. You are easily annoyed and irritated.....	0	1	2	3	4
74. When you feel angry, you act angrily toward most everything.	0	1	2	3	4
75. Angry thoughts about an irritating event keep bothering you...	0	1	2	3	4
76. You become mad or angry easily.....	0	1	2	3	4
77. Your anger is so great that you want to strike something.....	0	1	2	3	4

78. You let little annoyances build up until you just explode.....	0	1	2	3	4
79. You become so upset that you hit something.....	0	1	2	3	4
In your day-to-day living do you find:					
80. Working tires you out pletely.....	0	1	2	3	4
81. Severe pains and aches make it difficult for you to work	0	1	2	3	4
82. You get up tired and exhausted in the morning even with your usual amount of sleep	0	1	2	3	4
83. You suffer from severe nervous exhaustion.....	0	1	2	3	4
84. You get nervous and shaky when approached by a superior.....	0	1	2	3	4
85. Your thinking gets completely mixed up when you have to do things quickly.....	0	1	2	3	4
86. You become so afraid you can't move.....	0	1	2	3	4
87. You must do things very slowly to do them without mistakes..	0	1	2	3	4
88. You get directions and orders wrong.....	0	1	2	3	4
89. Unable to keep thoughts from running through your mind	0	1	2	3	4
90. Fearful of strangers and/or strange places make you afraid	0	1	2	3	4
91. Sudden noises make you jump or shake.....	0	1	2	3	4
92. Frightening thoughts keep coming back.....	0	1	2	3	4
93. You become suddenly frightened for no good reason.....	0	1	2	3	4
94. You have difficulty in concentrating.....	0	1	2	3	4

The Brief Symptom Inventory (III)

The following questions consider problems and symptoms that bother people every now and then. We are interested in how much these problems and symptoms have bothered you during the last month. Please circle the most appropriate response to each question. (0=Not at all, 1=A little, 2=Some what, 3=Quite a lot, 4=Very much).

1	Nervousness or shakiness inside	0	1	2	3	4
2	Faintness or dizziness	0	1	2	3	4
3	The idea that someone else can control your thoughts	0	1	2	3	4
4	Feeling others are to blame for most of your troubles	0	1	2	3	4
5	Trouble remembering things	0	1	2	3	4
6	Feeling easily annoyed or irritated	0	1	2	3	4
7	Pains in heart or chest	0	1	2	3	4
8	Feeling afraid in open spaces	0	1	2	3	4
9	Thoughts of ending your life	0	1	2	3	4
10	Feeling that most people can not be trusted	0	1	2	3	4
11	Poor appetite	0	1	2	3	4
12	Suddenly scared for no reason	0	1	2	3	4
13	Temper outbursts that you could not control	0	1	2	3	4
14	Feeling blocked in getting things done	0	1	2	3	4
15	Feeling lonely	0	1	2	3	4
16	Feeling blue	0	1	2	3	4
17	Feeling no interest in things	0	1	2	3	4
18	Feeling fearful	0	1	2	3	4
19	Your feelings being easily hurt	0	1	2	3	4
20	Feeling that people are unfriendly or dislike you	0	1	2	3	4
21	Nausea or upset stomach	0	1	2	3	4
22	Feeling inferior to others	0	1	2	3	4
23	Feeling that you are being watched or talked about by others	0	1	2	3	4
24	Difficulty getting to sleep	0	1	2	3	4
25	Having to check and double-check what you do	0	1	2	3	4
26	Difficulty making decisions	0	1	2	3	4
27	Feeling afraid to travel on buses, subways or trains	0	1	2	3	4
28	Trouble getting your breath	0	1	2	3	4
29	Hot or cold spells	0	1	2	3	4
30	Having to avoid certain things, places or activities because they frighten you	0	1	2	3	4
31	Your mind going blank	0	1	2	3	4
32	Numbness or tingling in parts of your body	0	1	2	3	4
33	Feeling hopeless about the future	0	1	2	3	4
34	Trouble concentrating	0	1	2	3	4
35	Feeling weak in parts of your body	0	1	2	3	4
36	Feeling tense or keyed up	0	1	2	3	4
37	Thought about death or dying	0	1	2	3	4
38	Having urges to beat, injure or harm someone	0	1	2	3	4
39	That you wake up early in the morning and can't get back to sleep	0	1	2	3	4
40	Having urges to break or smash things	0	1	2	3	4
41	Feeling very self-conscious with others	0	1	2	3	4
42	Feeling uneasy in crowds	0	1	2	3	4
43	Attacks of terror or panic	0	1	2	3	4

44	Getting into frequent arguments	0	1	2	3	4
45	Feeling nervous when you are alone	0	1	2	3	4
46	Others not giving you proper credit for your achievements	0	1	2	3	4
47	Feeling lonely even when you are with people	0	1	2	3	4
48	Feeling so restless you can not sit still	0	1	2	3	4
49	Feelings of worthlessness	0	1	2	3	4
50	Feeling that people will take advantage of you if you let them	0	1	2	3	4
51	The idea that you should be punished for your sins	0	1	2	3	4
52	Never feeling close to another person	0	1	2	3	4
53	The idea that something is wrong with your mind	0	1	2	3	4

11.2. INTERVIEW

The questions of the standardized stomatognathic interview (I-V)

- 1) Have you had ear symptoms during the last month not associated with infection or otitis?
 - a) earache
 - b) tinnitus or hum
 - c) fullness (or fluid) in the ears
 - d) no ear symptoms

- 2) Have you noticed keeping your teeth tightly together in any situations other than eating?
 - a) yes
 - b) no

- 3) Have you had headache?
 - a) yes
 - b) no

- 4) Have you had pain
 - 1) in the back?
 - a) yes
 - b) no
 - 2) in the neck
 - a) yes
 - b) no
 - 3) in the shoulder
 - a) yes
 - b) no
 - 4) no pain?

- 5) Have you heard sounds from the TMJ during mandibular movements?
 - a) yes
 - b) no

- 6) Have you had any of the following symptoms of TMD
 - 1) tiredness or stiffness of the mandibular
 - a) yes
 - b) no
 - 2) intermittent locking of the mandibular
 - a) yes
 - b) no
 - 3) continuous difficulties in opening the mouth
 - a) yes
 - b) no
 - 4) luxation of the mandible
 - a) yes
 - b) no
 - 5) pain on wide opening of mouth
 - a) yes
 - b) no
 - 6) none of the symptoms mentioned above

- 7) Have you had pain during mastication?
 - a) yes
 - b) no

- 8) Have you had globus not associated with major sensations or feelings?
 - a) yes
 - b) no

- 9) Have you had difficulties in swallowing?
a) often or all the time?
b) seldom (less than once a month)
c) no
- 10) Have you had hoarseness or voicelessness not associated with a common cold?
a) yes b) no
- 11) Have you noticed keeping your teeth tightly together in situations others than eating?
a) yes b) no
- 12) Have you had trauma to the jaws?
a) yes b) no
- 13) Have you had facial pain?
a) yes b) no
- 14) How intense is your facial pain at the moment on a scale 0 - 10?
(0=no facial pain, 10=the worst imaginable facial pain)
- 15) How intense has your facial pain been at its worst during the preceding month on a scale 0- 10?
(0=no facial pain, 10=the worst imaginable facial pain)
- 16) How intense has your facial pain been on average during the preceding month on a scale 0 -1 0?
(0=no facial pain, 10=the worst imaginable facial pain)
- 17) Have you had lingual pain?
a) yes b) no
- 18) Have you had dizziness?
a) yes b) no
- 19) Have you had stomatognathic treatment earlier?
a) orthopedic appliance
b) occlusal treatment
c) ongoing stomatognathic treatment
d) no
- 20) How many years ago did you have stomatognathic treatment?
- 21) Do you yourself think that you need stomatognathic treatment because of
1) stomatognathic problems a) yes b) no
2) otalgia a) yes b) no
3) both a) yes b) no
4) no treatment need

11.3. Signs and symptoms of tmd in the tmd treatment classifications (III, IV)

Distribution of subjects with clinical findings and symptoms of TMD at the baseline among the TMD treatment need subgroups at the baseline in Studies I and IV (n=411).

Clinical findings of TMD	TMD treatment need subgroup					
	Active		Passive		No need	
	%	n	%	n	%	n
Total of subjects	100	30	100	191	100	190
Tenderness on palpation						
masticatory muscles	87	26	62	118	15	29
TMJ	63	20	14	26	1	2
TMJ clicking	50	15	31	59	20	38
TMJ crepitation	37	11	31	58	16	30
Painful jaw movements	53	16	20	38	5	9
Symptoms of TMD						
Total of subjects	100	30	100	191	100	190
Tiredness or stiffness						
of the mandible	30	9	14	27	3	5
Intermittent locking						
of the mandible	23	7	11	21	4	7
Continuous difficulties						
in opening the mouth	10	3	3	6	0	0
Pains on §wide opening						
of the mouth	20	6	4	8	1	1
Pain in TMJ or muscles in front of						
the ear (during mastication)	37	11	12	22	3	5

Distribution of subjects with symptoms and clinical findings of TMD among the active TMD treatment need subgroup and the Not active subgroup in the SS1 (n=91)

Clinical findings of TMD	TMD treatment need subgroup			
	Active		Not active	
	%	n	%	n
Total of subjects	100	44	100	47
Tenderness on palpation				
masticatory muscles	87	26	62	18
TMJ	67	20	13	25
TMJ clicking	50	15	31	59
TMJ crepitation	37	11	30	58
Painful jaw movements	53	16	20	38

Symptoms of TMD	Active		Not Active	
	%	n	%	n
	Total of subjects	100	44	100
Tiredness or stiffness				
of the mandible	11	5	6	3
Intermittent locking				
of the mandible	7	3	2	1
Continuous difficulties in				
opening the mouth	2	1	0	0
Pains on wide opening				
of the mouth	9	4	2	1
Pain in TMJ or muscles in front of				
the ear (during mastication)	27	12	4	2