

Atypical Earache Otomandibular Symptoms

With a basic understanding of ear pain-related pathologies and referral patterns, a physician can provide a quick screening examination for atypical earache by palpating the jaw joint, muscles of mastication, and cervical muscles.

By Keith A. Yount, DDS, FAGD

Introduction

There is a close relationship between disorders of the jaw/neck and symptoms of the ear. In one study of 344 patients, 60% of the patients with temporomandibular joint disorder (TMD) had ear symptoms.¹ In another study of 400 patients, 42% had ear symptoms.² 21 of 28 patients had relief of tinnitus following orofacial pain therapy.³ The prevalence of non-otologic aural symptoms or referred otalgia in TMD patients varies from 3.5 to 42%.⁴ The prevalence of tinnitus is 15% in the general population. With TMD patients, this percentage escalates from 33% to 76%.⁵ The prevalence of vertigo in the general population is 5% and in the TMD population this percentage escalates from 40% to 70%.⁶ The fullness in the ear and minor hearing loss have not have been adequately studied. Referred otalgia may account for as much as 50% of all ear pain complaints.⁷ Disc displacement was found to be present in the ipsilateral joint in all 53 patients with unilateral tinnitus.⁸ In a clinical case report, a 38-year old female with confirmed TMD underwent an arthroscopic procedure; upon awakening she had ear pain, saline in her middle ear, and hearing loss without ear canal perforation.⁹

An understanding of the relationship between the jaw/neck pathologies and ear requires reviewing today's anatomical, neuromuscular, and central sensitization science. The anatomical concepts relate the direct connection of the jaw joint and trigeminal structures to the ear. The neuromuscular concept relates referral patterns of specific muscles of the trigeminal and cervical systems to the ear. Central sensitization incorporates how several different sources of pain (undiagnosed pain, 'piggy-back' pains, frequent recurrent pains, and chronic pains) induce anatomical, neurochemical, and physiological changes to the nervous system. The chronic pain that evolves from these mechanisms changes the visual diagnostic world of acute pain to the "bio-detective" world of orofacial pain and may confound diagnoses.

Differential Diagnosis

The atypical earache patient's differential diagnostic list should include articular disc disorders (TMJ/TMD), myofascial pain dysfunction (chewing muscle pain), cervical muscle dysfunction, or cancer. Even though the ear is the source of most ear pain, the atypical earache is a fairly common adult occurrence in medical practices. Atypical earaches commonly are in the chronic pain arena with multiple causes and often involving central sensitization. If the physician had some way of knowing it was only a muscle problem the patient could be referred to a dentist for therapy. If the diagnosis is unknown at the time of referral, it may be a better choice to refer to an orofacial pain specialist trained in all the atypical ear pains. The atypical ear-ache poses a management problem. All too often, after ruling out the ear as the source of the pain—an important step in the diagnostic journey—the patient is given no direction about how to complete this diagnostic journey. Some patients report that they have been told that they are "just a stressed out female" since the pain is aggravated by stress. Occasionally, a physician might just pass it off as TMD without an exam due to the statistics of occurrence. Some refer the patient to an ENT for a

second opinion on the ear as the source of the pain. In a busy practice where the typical doctor has about 12.5 minutes with the patient, it is difficult to correctly diagnose or refer the patient to the appropriate specialist.

The following clinical case illustrates the risks in not pursuing a differential diagnosis for an atypical earache:

A patient complained of moderate, deep aching pain in her left ear with one or more of following symptoms: tinnitus, vertigo, fullness in the ear, or minor hearing loss. The patient had little or no inflammation of the tympanic membrane. She complained that chewing, yawning, opening wide, and stress aggravated the pain. Since the pain seemed not to be associated with the ear, the attending physician made a casual comment that it might be TMD. However, a referral to an orofacial pain specialist was not made. The patient, feeling that her complaint was a benign problem, went home instead of seeking the appropriate consultation. The left ear pain worsened and she finally went to an orofacial pain specialist. Months later, this pain was diagnosed as a cancer in the Pterygoid fossa.

Pathophysiology

The anatomical relationship between the jaw/neck and ear is seen by examining the skull and reviewing the close proximity of the two adjacent structures. The “wiring of the skull” is much more complex and convoluted than in the rest of the body, which makes it difficult for the brain to discriminate between the ear and jaw joint. The jaw joint is separated by a thin window of bone to the mid-brain and a thick bone to the adjacent ear canal.¹⁰ The retrodiscal tissues of the jaw joint attach up and down the ear bone with the blood and nervous tissues in the lower and middle tissues. The upper compartment of retrodiscal tissue attaches elastin from the tympanic plate bone to the meniscus (disc) of the superior head of the Lateral Pterygoid. The elastin retracts the disc from the 2 o’clock position on opening back to the 11 o’clock position on closure. Also, the elastin resists condylar dislocation or excessive translation. This elastin opposes the eccentric pull of the superior head of the Lateral Pterygoid in front of the disc to continually keep the disc directly interposed between the two convex surfaces of bone at maximum vector of force. In 1983, Dr. Parker Mahan found that the Superior Lateral Pterygoid muscle contracts on closing¹¹ as the condyle moves posteriorly. The disc sits in between the convex surfaces in a bow tie configuration, when anatomically correct. The disc is connected to the condyle by two-bucket handle ligaments on the lateral and medial sides of the joint. The medial aspect of the joint is associated with the Medial Disc Ligament, the Anterior Malleolar Ligament, and the Sphenomandibular Ligament. The medial aspect of the condyle has no capsule, but is a blending of different tissues.¹²

The second way the jaw joint and ear are connected is through a common nerve distribution, the Auriculotemporal Nerve. This nerve supplies both the lateral surface of the meatus of the ear and all the innervation of the jaw joint.¹⁰ This wiring commonality is one of the ways pain from the joint may be referred to the ear. In the world of acute pain, the site of the pain is the source of the pain, but in the chronic pain world this is not always true. Whether it is known exactly how this referral happens scientifically, it does happen clinically—especially with chronic pain pathologies.

Dissection research by Rees, Pintos, and Loughner discovered the direct connection from the ear to the jaw joint by revealing an embryonic ligament that in approximately 6% of individuals is large and mobile enough to put tension on the middle ear structures. In 1954, Dr. Rees described the discomalleolar ligament (DML) that arises from the tendon of the Lateral Pterygoid and passes superiorly through the petrotympanic fissure and attaches to the malleus. In two additional studies, the DML was not observed to

be a viable ligament in these dissections. Some studies show it attaches to the wall of the petrotympanic fissure or dissipates with development; and therefore is not a viable link. Pintos' dissection¹³ in 1962, suggests the anterior Malleolar ligament (AML) is a viable ligament that could explain the inter-connected symptoms of the ear and jaw joint. He reported that this ligament (AML) attaches to the anterior process and neck malleus and passes inferiorly through the petrotympanic fissure to join the retrodiscal tissues of the medial aspect of the joint joining the Sphenomandibular ligament. Since that time, the ligament existence has been confirmed by additional researchers. In 1989, Loughner, Larkin, and Mahan expanded Pintos' dissection research findings.¹⁴ In seventeen of the specimens in the Loughner dissection, the DML joined the AML attaching to the neck of the malleus. In three of the specimens, even pulling on the SML caused movement on both the AML and malleus. This is a concern for any surgical procedure that requires mandibular distraction in the jaw joint allowing tension on the SML, AML, and then on the malleus, causing potential hearing loss. The mandible is lowered up to 10 mm for jaw joint surgery. This movement would be enough to rip the malleus from the tympanic membrane. This helps explain the significant statistical relationship between tinnitus and displaced discs (articular disc disorder). The disc of the jaw joint assembly displaces at the medial pole in an anterior and medial direction, putting tension on the medial and retrodiscal tissues, including the medial ligament and the AML as it enters the pterotympanic fissure. If the AML were mobile enough, it would put enough tension on the malleus to cause the tinnitus or vertigo. Figures 1 and 2 illustrate the pterotympanic fissure and the malleolar ligament interconnections.



Figure 1. Pterotympanic fissure: anterior Malleolar artery and vein, chorda tympana nerve, and anterior Malleolar and disco Malleolar ligament run through this fissure. Photo by Keith A. Yount, DDS, FAGD.



Figure 2. Anterior Malleolar Ligament attaching to head of Malleolus. From Loughner BA, Larkin LH, & Mahan PE.¹⁴ Reprinted with permission.

Trigger Points

Muscular problems of the head and neck may refer pain to distant sites under certain conditions. To understand this phenomenon, research must be reviewed that has been conducted over the past 25 years. The muscle spindle is innervated by the sympathetic system.¹⁵ In a series of physiologic studies in 1985, Passatore & Gassi placed an EMG electrode into the muscle spindle, showing a significant increase in activity when sympathetic stimulation is provided. This response is not blocked by curare (skeletal neuromuscular blocker), but is blocked by phentolamine (sympathetic alpha blocker).¹⁶ Hubbard used an EMG guided needle into a muscle spindle to record spontaneous activity in trigger points, but even at a distance of 1 mm there was no activity.¹⁷ It is now a known fact that the sympathetic system does exert control over the muscle spindle and accounts for tension in muscles when over-stimulated by stress, anxiety, or emotional upset. Over time, and with repeated over-stimulation, the spindle swells, becomes tender, damaged, and a source of ectopic electrical activity.¹⁸ This hidden source of recurrent, persistent, and ectopic pain signal threatens to add confusion to any physician's diagnostic differentiation. These muscle spindles have a tendency to refer pain to distant sites, making the diagnosis more difficult. Surprisingly, most patients are rarely aware of the existence of these trigger points in the muscles until they are pointed out by a physical therapist, an orofacial pain specialist,¹⁸ or even during a massage. During a massage, the woman may accuse the masseuse of too much pressure, but in reality a tender spot was touched in the muscle with the same pressure. Trigger points are established from an array of predisposing and precipitating factors including poor sleep, poor nutrition, lack of cardiovascular conditioning, bad posture, cervical parafunction, clenching/grinding teeth, poor bite, stimulants, muscle tension, an up-regulated sympathetic system, and an array of many other factors.¹⁹

Because there are multiple causes, trigger points are impossible to manage without using a multi-disciplinary approach. The presence of a trigger point in one part of the muscle makes the muscle prone to additional trigger points in other areas of the muscle.¹⁸ Trigger points lower pain thresholds, intensify muscle pain, increase muscle fatigue, fatigue the muscle faster, functionally shorten the muscle, and increase muscle weakness.¹⁸ One of the most effective techniques in locating a trigger point is to find the taut band (it feels like a banjo string) and palpate the length of the band until finding the trigger point.¹⁸ The trigger point location will be confirmed by the patient's jump sign. These damaged muscle spindles seem to be the key to the referred pain from a muscle to a distant site. Muscle referral patterns have been worked out by Dr. Janet Travell in her book, *MPD and Trigger Point Manual, Volume 1*.¹⁸ The basis for this referral mechanism is the damaged area of muscle, commonly called the trigger point, and its interconnection with the sympathetic and skeletal system.

Pain Referral Patterns

The jaw joint refers to the ear more often than any other structure. The TMJ is separated from the ear by millimeters of bone. It is so close to the ear it is easy to understand how pain from the joint is confused with ear pain by the brain especially with all its head and neck wiring. The second way the jaw joint can refer pain to the ear is through the common nerve distribution of the Auriculotemporal Nerve that supplies the eardrum and all of the jaw joint structures. The third way is the direct connection from the medial ligament to the AML to the malleus. This helps explain the strong tinnitus and vertigo component common to patients with articular disc disorders. If one finds no inflammation in the eardrum, you should consider screening for jaw joint problems should be considered. These structures may be checked out like any orthopedic problem. Any painful joint or muscle tissue is easily identified by palpating or functionally moving the jaw structures. The lateral pole of the jaw will be significantly tender to

palpation, especially when disc displacements and arthritic changes affect the jaw joint.²⁰ When the patient reports that the ear pain is aggravated by chewing, yawning, or opening wide, it provides clues as to the source of the pain. A further clue is that the dull moderate ache is episodic and does not intensify with time. The pain will be episodic for the TMJ and not progressive, as it would be with an ear infection. Additional clues to a TMJ problem, is a history of restricted opening or deviation on movement. Also there may be a presence or history of jaw clicking on function. There are many ways to differentiate the jaw joint from the ear, but one should choose a set of tests that can be performed in 5 minutes to be able to refer with confidence to an orofacial pain specialist. In the diagnostic world, pathologies rarely present with this much diagnostic data. The presentations of a real earache and the atypical earache may be so similar that a clinical trial of antibiotics may be necessary to differentiate between the two. If the earache is not responsive to the anti-biotic, then a referral to an orofacial pain specialist may be the right choice. On the other hand, if all indications continue to point to the ear itself, then a referral to ENT may be the best next step.

The referral of pain from a muscle to the ear can be from any one of four muscles, two trigeminal and two cervical. The trigeminal muscles are the deep Masseter and the Lateral Pterygoid. The cervical muscles are the SCM and the deep posterior cervical muscles (little devils of the atlas/axis complex). Of all the muscles, the Deep Masseter is the most likely structure to refer pain to the ear¹⁸ from a muscle group.

Deep Masseter Muscle

The Deep Masseter is part of the power closing muscles and assists at retruding the mandible. It lies at a 45-degree angle to the vertical plane from the zygomatic arch to the posterior border of mandible. The Superficial Masseter is in the vertical plane over the Deep Masseter from the zygomatic arch to the lower border of mandible. Over-stimulation of this muscle occurs in patients that tooth brace, clench, object bite, avoid a high posterior crown or filling, or have a Class II bite. The muscle can be palpated just below the jaw joint, along the border of the posterior mandible. To palpate the Deep Masseter, the physician has the patient clench and relax, feeling the bulging Superficial Masseter with a finger. The Deep Masseter lies in the lower part of the triangle between the bulge of Superficial Masseter on clench, the posterior border of the mandible, and the lateral pole of the jaw joint. In a patient with a significantly tender Deep Masseter, it increases the suspicion that the differential diagnosis for the atypical earache should include Deep Masseter referral of pain. The aggravators to this type of pain will be chewing, talking, yawning, stress, opening wide, and clenching. The pain descriptors are dull, ache, and moderate. The pain does not respond to antibiotics or any ear therapy, but does partially respond to muscle relaxants and NSAIDS. The pain is episodic in nature. An ear pain would not be challenged by chewing gum. In this case, the ear cartilage may be tender for either joint or Deep Masseter pathology due to diffusion of inflammatory chemicals to the surrounding area. See Figures 3 & 4 for illustration of Deep Masseter fibers and referral pattern.



Figure 3. Deep Masseter: fibers run at 45 degree angle to vertical as compared to superficial Masseter fibers are more vertical. From Travell JG and Simons DG.¹⁸ Reprinted with permission.

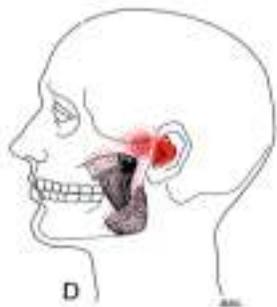


Figure 4. Referral pattern: Deep Masseter: source pain is DM and site pain is ear. From Travell JG and Simons DG.¹⁸ Reprinted with permission.

Lateral Pterygoid Muscles

The Lateral Pterygoid muscles manage the position of the disc or meniscus between the condyle and eminence during function. It will refer pain to the ear,¹⁸ the maxillary sinus, or retro-orbital. The Lateral Pterygoid is comprised of an upper belly and a lower belly. The lower belly contracts on opening, protrusion, or movement to the opposite side. The upper belly contracts on closing to stabilize the disc against the posterior pull of the elastin and to keep the disc in place between the convex bones at the major force zone. When clenching, both bellies contract at the same time along with the power closure muscles which not the muscles' normal mode of operation nor function. This results in isometric contraction of muscles, excessive lactic acid production with no lymphatic pump, and inflammation. The excessive pull on the joint structures can cause microtearing of ligaments and connective tissue and the inflammation can create pain in tissues adjacent to the ear bone.

Sternocleidomastoid (SCM) Muscle

The cervical muscles are less likely to refer pain to the ear, but they can. In the absence of tender jaw muscles or a tender lateral condylar pole and jaw function not aggravating the pain, the physician might consider the possibility that cervical muscle referral might be part of the differential diagnosis for the atypical earache. One of the cervical muscles Janet Travell found to refer pain to the ear¹⁸ is the Sternocleidomastoid (SCM). As compared to the deep cervical muscle of the atlas/axis complex, the Sternocleidomastoid, or SCM, is easy to palpate.¹² It lies just below the skin at the anterior lateral border of the neck. It attaches to the Mastoid process just behind the ear and to the sternum and clavicle as two different bodies. This attachment to the bone behind the ear structure may be one of its mechanisms for referral to the ear. It is a major muscle for head function and balance. If the SCM is tender to palpation and the pain is aggravated by head and neck movement, SCM may be considered as a component of the differential diagnosis. The pain may be described as a moderate, dull, or ache. Referred pain from the SCM occurs with poor posture, carrying a heavy backpack, heavy computer bags, tension in the cervical muscles, up-regulation of the sympathetic system, holding the phone on the shoulder, poor work station layout, arthritis of the cervical facet joints, and injury. The SCM has been associated with dizziness and is responsible for balance. In the absence of tender jaw muscles and lateral pole of joint and pain not

aggravated by jaw function, cervical muscles are considered a possibility for a cause of the ear pain. In the presence of a tender SCM and head function aggravating the pain, the SCM muscle would be suspect as the source of the ear pain. Figure 8 illustrates the referral pattern of the Sternal Cleido Mastoid neck positioning muscle which is used in forward head posture and balance.



Figure 5. The source of the pain is SCM with the perceived site of the pain posterior to the ear. From Travell JG and Simons DG.¹⁸ Reprinted with permission.

Deep Posterior Cervical Muscles

The little devils or sub-occipital muscles are the other cervical muscles that refer pain to the ear.¹⁸ The suboccipital muscles attach to the atlas/axis complex (C_1 & C_2) in the deep, third level of neck muscles. The names of these muscles are Rectis Capitis Superior, Rectis Capitis Inferior, Inferior Oblique, and Superior Oblique. Patients with this type of pain referral usually have a history of motor vehicle collisions (MVC's) or cervical traumas associated with the onset of ear pain. These patients also report that head and neck movements or posturing can aggravate the ear pain. The pain can be provoked by poor head and neck posture and by provoking the deep muscles, creating ear pain or significant other pain. The report that the pain worsens as the day progresses can be a clue indicating that cervical muscles may be a strong candidate for the source of this pain. These cervical muscles cannot be directly palpated and must be provoked to create the tension on the muscle that may indicate its involvement. Unlike acute pain where the source and site of pain are the same, when it comes to chronic pain, there is no rule that limits the number of structures that can refer pain to same site. In fact, it is not unusual for the jaw joint and the Deep Masseter to refer pain to the ear at same time. Fig 6 & 7 illustrate the location of potential pain sources in the suboccipitals and the subsequent referred pain sites.



Figure 6. Sub-occipital muscles at the base of the skull and neck. Several names for these muscle (Little Devils, atlas and axis muscles, deep cervical muscles, 3 layer of posterior neck muscles, suboccipital muscles; specific names (Rectus Capitus Superior and Inferior, Superior oblique and Inferior Oblique). Illustration after [Netter et al. 19xx](#).



Figure 7. True source of the pain is suboccipitals with the perceived site of pain at the ear. From Travell JG and Simons DG.¹⁸ Reprinted with permission.

Another mechanism for referred pain is the elastin tension on the ear bone from the pull of the superior head of the Lateral Pterygoid against the disc to which the elastin attaches. An additional way the Lateral Pterygoid can create pain in the ear is by its attachment to an embryonic ligament, the AML, putting tension on the malleus. In this case, the ear pain is usually associated with dizziness or tinnitus. The Lateral Pterygoid muscle cannot be palpated; thus, the only way to test this muscle is by provoking the muscle to work. If the muscle is inflamed and is provoked, it will feel tightness, tension, pulling, or discomfort. However, provocation of a muscle is never as accurate as direct palpation. In this case, the ear pain will be non-progressive, dull, and moderate compared to acute ear pain.

Chronic Pain Progression

The more difficult a problem is to diagnose—with multiple causes or aggravators or weaker referral connections and communications—the more chance it has of remaining for many months or even years. The longer the duration and the more severe the pain levels, the greater the chance that the pain will alter the biochemistry, anatomy, and function of the pain reporting system. The greater the severity and the longer duration of pain, and the more emotionally distressed the patient, the greater the impact will be on hormonal function.²¹ It has been observed that the intensity of pain from a physical injury relates to the attention given at the time.²² Chronic pain has lasting effects on the pain sensing and reporting systems. Constant or recurrent pain induces a lowering of the threshold for pain by the pain receptors.²³ The pain system also activates the silent nociceptors by initiating “wind up.”²⁴ These receptors do not normally react to pain stimulation, but when activated by long term pain, they are brought into action. The pain system reacts to smaller and less intense stimuli. Under normal conditions the pain reporting fibers (C-fibers) report a pain stimulus from a pain receptor. Over time, the pain reporting fibers (C-fibers) begin to develop alpha adrenergic receptors²⁵ which respond to adrenalin, the stress molecule. The pain also induces mass cells to enhance production of NGF²⁶ enhancing the sprouting and growth of nerves. This in turn stimulates the sympathetic nerves to sprout in lamina 3 of the dorsal horn and attach to the pain reporting fibers in lamina 1.²⁷ A stressor can now initiate the same pain response that a pain stimulus can elicit. The purpose of the adaptive response to pain is to limit activity to allow healing, but when the pain system is over-stimulated, the pain is ignored, the pain is undiagnosed, the pain has significant meaning, or the pain is so severe the adaptive response is not as effective. A compounding aspect is the difficulty in limiting activity in the head region due to its importance. The nervous system adapts to multiple, constant,

or recurrent pain impulses, becoming part of the pain syndrome. The longer a pain is present and the more severe the pain, the more changes manifest in the pain reporting system.

Conclusion

The majority of earaches are associated with ear structures; yet one may find ear pain with a tympanic membrane that is normal, or one in which antibiotics do not reduce the pain. The physician may refer the patient for an ENT evaluation and find that the patient returns after no inflammation is found in the tympanic membrane. The usual medical practice sees mostly acute ear pain in which the site and the source are same. The atypical ear pain patient presents a chronic pain in which the pain impulse may be coming from one or multiple sources and is enhanced by the pain reporting system, but is referring to the ear. When there is no inflammation in the eardrum, one is tempted to dismiss the patient's concerns. When lack of time limits the number of diagnostic tests, the physician may provide a diagnosis of exclusion (if it's not the ear, it must be TMJ or psychosomatic/stress-induced), or take the pharmaceutical route. Such misdiagnosis may lead to a hyper-active pain reporting system, a dependence on drugs, patient frustration, or perhaps even legal implications for one's practice.

Expanding the differential list of pathologies to include these sources of atypical earaches can help the practitioner determine the best course of diagnostics to define the problem. By referring for an orofacial consultation, the practitioner can rule out these sources of ear pain and help the patient finally get to the root of the problem—yet, because there is misconception and confusion involving the connection of ear pain to the jaw and neck structures, an orofacial pain specialist is typically the 5th to 7th doctor seen for atypical earaches. With an understanding of these ear pain-related pathologies, a physician can provide a quick screening examination by palpating the jaw joint, muscles of mastication, and cervical muscles and then referring as needed. One of these referral sources is an orofacial pain or trigeminal orthopedic specialist. It is important to note that “TMD patients” respond well to conservative treatment.² and TMD is significantly correlated to aural health.¹

About the Author

Keith A. Yount, DDS, FAGD, is in practice at Raleigh Facial Pain Services in Raleigh, North Carolina. A diplomate of the American Board of Orofacial Pain, Dr. Yount received his DDS from the University of North Carolina, Chapel Hill. In addition to lecturing on chronic pain, he also has written articles on this subject for several publications. Dr. Yount can be reached at 4505 Fairmeadow Lane, Ste 207, Raleigh, NC 27607.

References

1. Lam David K, Lawrence HP, and Tenenbaum HC. Aural symptoms in TMD patients attending a craniofacial pain unit. *Journal Orofacial Pain* 2001; 15: 146-157.
2. Keersmaekers K, Boer JA, and Berghe D. Otalgia in Patients with TMD. *Journal Prosthetic Dentistry* 1996; 75:72-76.
3. Bernstein JM & Mohl ND. Tmd masquerading as disease of the ear, nose, throat. *Tr Am Pcad Ophth & Otol* Nov-Dec 1969; 73:1208-1217.
4. Ciancaglini R., Loreti P, and Radaelli G. Ear, nose, throat symptoms in patients with TMD: The association of symptoms according to severity of arthropathy. *J orofacial Pain* 1994; 8:293-297.
5. Rubinstein B. Tinnitus & craniomandibular disorders: Is there a link? *Swed Dent J* 1993; 95 (spo):1-46.

6. Parker WS & Chole RA. Tinnitus & temporomandibular pain-dysfunction disorders. *Clin Otolaryngol Allied Sci* 1994; 19:370-380.
7. Yanagisawa K and Keton JF. Referred Otagia. *Am J Otolaryngology* 1992;13(6):323-327.
8. Ren YF and Isberg A. Tinnitus in Patients with Temporomandibular Joint Internal Derangement. *J Cranil* 1995; 13 (2);75-80.
9. Schickinger B, Gstoettner W, Cerny C, & Kornfehl J. Variant petrotympanic fissure as possible cause an otologic complication during TMJ arthroscopy. In *J Oral Max Surg* 1998; 27:17-19.
10. Moore KL. *Clinically Oriented Anatomy*, 2nd edition. Williams & Wilkins. 1985. page a-914, b-918.
11. Mahan, PE. Superior and inferior bellies of the lateral pterygoid muscle EMG activity at basic jaw positions. *J Prosthetic Dent*, 50:710, 1983.
12. Mahan PE & Alling CC *Facial Pain*, 3 edition. Lea & Febiger 1991; page a-201, b-148.
13. Pinto OF. A new structure related to the TMJ and middle ear. *J Pros Dent* 1962; 12 (1): 95-103.
14. Loughner BA, Larkin LH, & Mahan PE. Discomalleolar and anterior Malleolar ligaments: Possible causes of middle ear damage during TMJ surgery. *OOO* 1989;68:14-22.
15. Barker D & Banks R. *The muscle spindle*. New York: McGraw-Hill. 1986:309-411.
16. Passatore M, Grassi C, & Filippi G. Sympathetic induced deeloment of tension in jaw muscles: the possible contraction of intrafusal muscle fibers. *Pfugers Arch* 1985; 405: 297-304.
17. Hubbard DH & Berkoff GM. Myofasical Trigger Points Show Spontaneous Needle EMG Activity. *Spine* 1993;18(13): 1803-1807.
18. Travell JG & Simons DG. *Myofascial Pain and Dysfunction, The Trigger Point Manual, Volume 1*. Williams & Williams. 1983, pages a-46,b-52,c-54 & 56,d-62,e-204, f-220, g-260, h-322)
19. Friction JR & Arancio DD. *Interdisciplinary Mahnagement of Myofasical Pain of the Masticatory Muscles*, Ch 27. *Orofacial Pain And TMD*. Friction and Dubner. Raen Press, New York.
20. Dawson PE. *Evaluation Diagnosis, & Treatment of Occlusal Problems*, 2nd edition. C Mosby Co. 1989 page 96-98.
21. Lipton JA & Marbach JJ. Components of the response to pain and variables influencing the response in three groups of facial pain patients. *Pain* 1983;16:343-359.
22. Sirois DA. Transient Injury Leading to Persistent Pain. Presentation at AAOP National Meeting 1999.
23. Mense S & Schaible HG. *General Pain Physiology*. Ch 8. *Applied Anatomy, Physiology, Psychology, & Pharmacology*.
24. Moskowitz MA. The neuro-biology of vascular head pain. *Ann Neurol* 1984;16:157-168.
25. Lou LP & Wiesenfeld Z. Effects morphine on prevention of spinal cord hyperexcitability following injury. *Pain* 1994;58:245-252.
26. Leon A, Buriani A, Toso D, Fabris M, Romanello S, & Aloe L. Mast cells synthesize, store, and release NGF. *Neurobiology* 1994;91:3739-3743.
27. Kim NJ, Na HS, Man HJ, Park KA, & Kang BS. Sprouting sympathetic nerve fibers into dorsal root ganglion following peripheral nerve injury. *Neuro Lett* 1995;199:87-90.