TINNITUS OF MYOFASCIAL ORIGIN

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Abstract:
Tinnitus in some instances is due to a primary muscle disorder: myofascial dysfunction of the muscles of the head and neck. Tinnitus of this origin can be diminished in some cases, and completely abolished in others, by appropriate treatment; this includes trigger point deactivation, specific exercises, and treatment of the underlying causes of the muscle dysfunction.

Keywords: Tinnitus, Trigger Points, Intramuscular Injections, Bruxism, Otolaryngology, Masseter muscle, Myofascial Trigger Point Pain, Myofascial Pain Syndromes, Injection, Physical Therapy

INTRODUCTION

The muscles of the head and neck were neglected as a cause of medical problems until Dr. Janet Travell described myofascial dysfunction (MFD). The tenets of MFD are described in the classic Travell-Simons textbook, Myofascial Pain and Dysfunction: The Trigger Point Manual (1). MFD is a primary muscle disorder which is characterized by: a) taut bands (tight bands of contracted muscle fibers), trigger points (TrPs, exquisitely tender, discrete nodule within the taut bands), muscle shortening and muscle stiffening, with attendant limitation of range of motion, motor abnormalities, and disturbed motor function; b) sensory abnormalities, such as tenderness and referred symptoms; c) autonomic changes which are occasionally present, especially in longstanding cases (1,2,3,4,5,6).

PATHOGENESIS

MFD develops as a result of muscle abuse in individuals who have a poorly understood predisposition to this disorder. The muscle abuse takes the form of: a) overuse (e.g. sitting for many hours with poor posture, and other poor work ergonomics (7)); b) misuse (e.g. parafunctional chewing due to a dental problem); c) underuse; d) trauma (e.g. whiplash) (2,6). Chronic tightening of the upper shoulder muscles from mental stress also plays a role. Other factors which promote myofascial dysfunction are metabolic, endocrine, and nutritional abnormalities. (These factors along with the muscle abuse factors are known as precipitating and perpetuating factors. The muscle abuse types of precipitating and perpetuating factors may not be readily apparent) (1,6). There is a female: male incidence of MFD on the order of 3:1 to 9:1 (8).

Muscles involved with MFD can cause local or referred symptoms, or can incite secondary trigger points (termed satellite trigger points) which can in turn themselves
cause local or referred symptoms. Although pain is the best known symptom of this muscle disorder, MFD is responsible for a large number and a wide variety of symptoms, especially in the head and neck (1). These symptoms often masquerade as primary disorders of the ear, nose, and throat (2). One of these symptoms is tinnitus (1,9,10). The influence of the musculoskeletal system upon certain types of tinnitus is well recognized (1,9,10,11,12,13,14).

Tinnitus of myofascial origin may be unilateral or bilateral. There are no predictive audiometric patterns for MFD tinnitus. Patients with severe hearing loss may completely clear their tinnitus with MFD treatment, and tinnitus sufferers with a normal audiogram may have no improvement with MFD treatment, and vice versa.

Although there is no strong predictor of which patients will benefit from MFD treatment, the following factors are somewhat more predictive of a favorable outcome:

1. Tinnitus which has been present for a shorter period—e.g. under 2 years vs. 10-20 years.
2. Tinnitus which accompanies other myofascial ENT/head & neck symptoms.
3. Tinnitus which fluctuates in intensity synchronously with other myofascial ENT/head and neck symptoms.
4. Tinnitus which is more pronounced on the side ipsilateral to the more prominent accompanying myofascial symptoms.

**TREATMENT**

IN OUR EXPERIENCE tinnitus of myofascial origin CAN BE caused by TrPs in the masseter and sternocleidomastoid (SCM) muscles. Treatment of this is to inject the TrPs of the SCMs and the upper trapezius--this will release the TrPs of the SCMs and also usually release the TrPs of the masseters (1,15). (It is generally not necessary to inject the TrPs of the masseters). The precipitating and perpetuating factors of myofascial dysfunction of these muscles must be identified and discontinued: chronic poor posture with rounded shoulders and head forward position, which promotes upper trapezius and SCM TrPs, must be corrected. Head, neck, and upper shoulder muscular imbalances, and cervical spine mobility impairment, may also promote trigger points in these muscles; when present, these precipitating and perpetuating factors must be identified and corrected by appropriate physical therapeutic measures (16).

Clenching or dental grinding/bruxism, which promote masseter TrPs, should be treated with an occlusal splint (1). T.i.d. stretches of the head and neck muscles and masseters are also prescribed. Any nutritional, metabolic, or endocrine deficiencies are also corrected (1).

TrP deactivation can be done manually by local pressure termed Trigger Point Pressure Release (1,18). However the recommended method is to needle the trigger point (19,20). This can be done with injection of xylocaine (0.1cc—0.2cc), or without injection (dry
needling) (19,20). Both dry needling and xylocaine injections are equally effective (21). The addition of xylocaine tends to give less post-injection soreness of the upper trapezius and SCMs. There is no advantage to injecting other medications; injecting steroids are contraindicated because of possible tissue damage (necrosis) and muscle atrophy (1).

TrP needling should be done with surgical precision by a practitioner with a thorough three-dimensional knowledge of head and neck anatomy. TrP needling is a very safe procedure when done by a well trained and experienced practitioner. The senior author has performed tens of thousands of TrP injections without nerve, vessel, or other complications; post injection soreness and ecchymosis have been the only side effects.

Post-injection soreness (1) occurs in approximately half of the patients. This is most prominent after the first set of injections, and decreases rapidly with subsequent injections. Due to the possibility of subsequent post-injection soreness, it is best to treat TrPs of the SCM and upper trapezius on one side only on the initial visit, so as not to discourage the patient from subsequent injections. Bilateral TrP injections can be given on each visit after that.

TrPs are injected q. week until two criteria are met: absence of tinnitus for one week, and no TrPs are found at the end of one week. Then the next interval is q. 2 weeks until the same two criteria are met. Then the interval is q. 3 weeks until the two criteria are met. At the point where there are no TrPs at the three-week interval and there has been no tinnitus for 3 weeks, trigger point needling can be discontinued. After the 3 week tinnitus and trigger point-free interval, the patient is discharged and is instructed to continue with the occlusal splint, t.i.d. stretches, and avoiding the precipitating and perpetuating factors.

Muscles with MFD tendencies are prone to develop TrPs from sustained maximal shortening (1). An occlusal splint is helpful; it prevents maximal shortening of the masseters, and discourages clenching. There are as many different varieties of occlusal splints as there are practitioners prescribing the splints, with various types of splints purported to be the best splint for various ear, nose, throat, or dental maladies.

The occlusal splint used is a soft flat plane splint (8). We recommend one with a 3-mm thickness at the interface of the lower and upper teeth when in occlusion. It may fit over the upper or lower teeth; often the patient tolerates this better over the upper teeth. The splint should be worn two hours during the daylight hours and all night while asleep. Once the tinnitus is clear the patient can wear the splint during sleep only, and this should be done indefinitely.

Tinnitus is one of the more difficult symptoms in the head and neck to effectively treat; that is because tinnitus is often of multi-factorial causes; therefore, only that portion of tinnitus which is of MFD origin is amenable to improvement by MFD treatment. The exact mechanism of MFD causing tinnitus is unknown, BUT MAY RELATE TO SOMATOSENSORY PROJECTIONS FROM MUSCLES AND TENDONS OF THE HEAD AND NECK TO THE AUDITORY CNS (12).
Results of MFD treatment for tinnitus vary from no improvement to complete cessation of tinnitus. In our experience, of the patients who appear to have myofascial related tinnitus, approximately 25% have total and complete cessation of tinnitus, 25% experience decided improvement, and 50% have no improvement.

The only certain way to determine whether MFD treatment will be of benefit for tinnitus is to carry out a therapeutic trial. This consists of a minimum of 5 office visits for TrP injections of the SCMs and upper trapezius, and incorporating all the other parts of the treatment as discussed above. If there is no improvement in the tinnitus after the fifth treatment session, it is not likely that this therapy will be successful.

After TrP injections are completed and the patient is discharged with instructions, the duration of improvement/cessation of tinnitus, as with any other symptom of myofascial origin, is determined by how well and how long the underlying MFD is controlled. This is related to how resolutely the patient wears the occlusal splint, continues daily stretches, and continues to avoid the precipitating and perpetuating factors.

CONCLUSION

Current options for tinnitus relief include biofeedback, tinnitus masking, and benzodiazepines. MFD treatment with trigger point deactivation adds another important modality of tinnitus control, with dramatic complete cessation of tinnitus for some patients, and improvement of the tinnitus in others. Consideration should be given to offering this treatment to sufferers of tinnitus.

References


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17. Rocha CA; Sanchez TG: *Myofascial Trigger Points; Another Way of Modulating Tinnitus*. *Prog Brain Res*. 2007;166:209-14


**Recommended Reading**


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1. Dept. of Otolaryngology/Head and Neck Surgery, Eastern Virginia Medical School, Assistant Clinical Professor
2. Old Dominion University, College of Health Sciences, School of Physical Therapy, Adjunct Clinical Professor; Myopain Seminars, Faculty & Examiner
3. Eaton-Peabody Laboratory, Mass. Eye and Ear Infirmary
4. Dept. of Otolaryngology, Harvard Medical School