TEMPOROMANDIBULAR JOINT (TMJ) PAIN SYNDROME

The temporomandibular joint (TMJ) is a condylar, hinge-type joint. An articular disc provides the joint with congruent contours and lubrication while at the same time dividing it into superior and inferior compartments. Various ligaments provide static support and restraint. The temporomandibular (lateral) ligament restrains movement of the lower jaw and prevents compression behind the condyle. The condyle, disc and temporal bone are kept firmly opposed by the sphenomandibular and stylomandibular ligaments acting together. The condyle and its socket have fibrocartilaginous surfaces, and a thin loose synovial capsule encases the entire joint.

All TMJ motion occurs in the superior and inferior compartments. The two essential movements are rotation and gliding, and both are necessary for opening and closing the mouth. When opening the mouth, rotation takes place in the inferior compartment, and occurs from onset to midrange. Gliding (a translatory movement of the condyle and disc along the slope of the articular eminence) follows this in the superior compartment, from midrange to the end of range (mouth full open). Closing requires a reversal of this sequence. To be functional, the temporomandibular joints on either side of the jaw must perform the same movements simultaneously. The TMJ is also capable of lateral (side to side) mandibular movement and forward mandibular protrusion (chin thrust).

The list of muscles that provide the TMJ movement (or directly associated with it) is impressive and lengthy. It includes the temporalis, masseter, pterygoid externus, pterygoid internus, mylohyoid, geniohyoidcus, digastricus, stylohyoideus, sternohyoid thyrohyoid, sternothyroid, and omohyoid muscles. Together, and in coordination, they produce the TMJ motions associated with chewing, talking, non-verbal expression, and swallowing. They may also be responsible for producing a great deal of discomfort and pain if they are uncoordinated (the *TMJ Pain Syndrome*). The *TMJ Pain Syndrome* is characterized by limited TMJ range of motion and severe pain in and around the joint. Faulty occlusion of the teeth, caused by missing teeth, abnormal eruption, or dental caries, has been considered a causative or contributory factor by most authorities, while very little attention has been paid to the influence that the joint's combined musculature may have in contributing to the *TMJ Pain Syndrome*.

The motion of the TMJ is, for the most part, controlled by the combined and coordinated action of the pterygoid externus, pterygoid internus, digastric, suprahyoid, temporalis, and masseter muscles. The pterygoid externus and internus muscles work together to move the mandible, anteriorly and laterally, toward the contralateral side. The digastric and suprahyoid muscles combine to depress the mandible. The largest and strongest of the jaw muscles, the temporalis and masseter muscles, together elevate the mandible and approximate it with the maxilla.

If relative hypertonus or hypotonus develops in any one of these muscles or muscle groups, a neuromuscular imbalance may be produced which adversely affects normal function of the jaw. The dysfunction that is created by such imbalance varies with the number and nature of the muscles that are involved. For example, if the pterygoids on one side of the jaw are relatively hypertonic, the mandible may be forced forward and toward the contralateral side so that the anterior surfaces of the lower incisors are made to push upwards against the posterior surface of the upper incisors, especially on the contralateral side, and the stability of those upper incisors (in extreme cases) may be threatened within the maxilla. Likewise, if both the masseter and temporalis muscles on one side are hypertonic, compared to those muscles on the contralateral side, prolonged approximation of the homolateral TMJ may occur. This may have the consequence of producing abnormally high pressures between the articular surfaces of the joint, and the opposing molars and premolars on the homolateral side, effectively creating faulty occlusion. The eventual result may include excessive wearing and deterioration of the homolateral joint cartilage and the enamel surfaces of the involved teeth. In either of these scenarios, the most common symptom is the production of pain. Associated pain may occur on either side of the jaw, in any number of teeth, and be referred to various other areas of the face, head, or neck (anterior or posterior).

In the common *TMJ Pain Syndrome*, the limitation of mandibular movement is generally produced by unilateral hypertonus of one or more muscles or muscle groups, and bilateral hypertonus

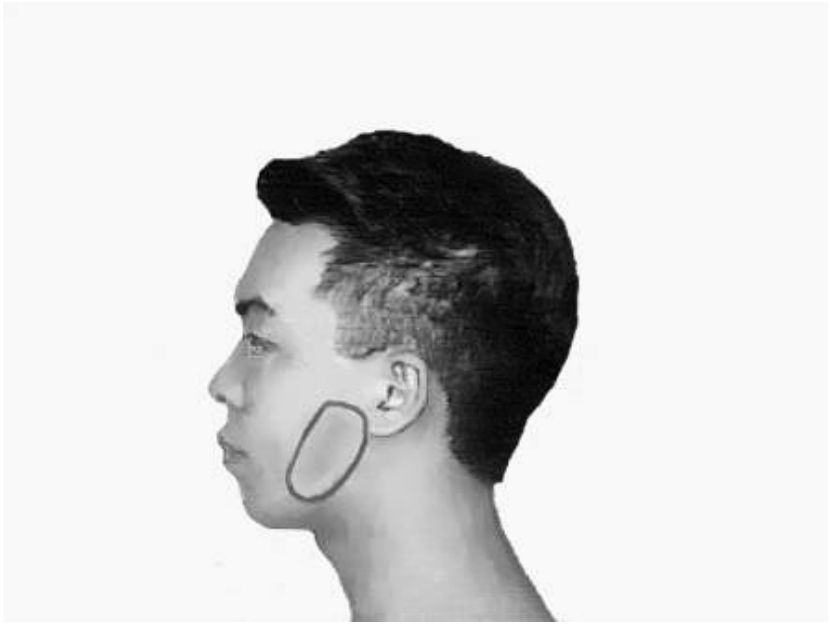
is rare. Historically, faulty occlusion of the teeth has been considered a causative agent in most cases of true *TMJ Pain Syndrome*. Emotional stress has been noted as a fairly common factor in producing or at least maintaining the syndrome, as reflected by habitual unilateral jaw clenching and nocturnal grinding of the teeth (bruxism). Inflammation, swelling and adhesions of the soft tissues associated with the upper chamber of the temporomandibular joint have been cited as being contributory to the limitations of lateral excursion of the mandible, while limitation of vertical excursion has been associated with inflammation, swelling and adhesions of the soft tissues associated with the lower chamber.

Any inflamed zones should be established through DSR survey of the TMJ and the pterygoid and masseter musculature.

Treatment

The chronic *TMJ Pain Syndrome* often has been noted to have a psychogenic origin and may not initially result from any initial malocclusion, joint disease, or joint deterioration. The associated symptomology generally arises from a neuromuscular imbalance of muscles that control the jaw. Effect treatment really depends on correcting this imbalance. The most efficient way of treating neuromuscular imbalance is with electromyometric feedback instrumentation, used in a program of neuromuscular reeducation that teaches the patient to establish and maintain a more normal neuromuscular balance of the TMJ musculature. This can be augmented by other forms of feedback that provide the patient with other means of controlling somatic responses to emotional stimuli. Specifically, galvanic skin resistance (GSR) feedback may be used to teach the patient not to over respond autonomically to emotional stimuli.

An acute *TMJ Pain Syndrome* may occur from over stretching or straining the musculature that articulates the temporal mandibular joint. Most commonly, the syndrome results from a strain or over-stretch of the masseter muscle, resulting in extreme pain that may seem to originate in the joint and then radiate along the jaw line. In such cases, a DSR survey may be useful in establishing any involved inflamed zones. A characteristic inflamed zone associated with the acute *TMJ Pain Syndrome* is illustrated below. Treatment should be directed at relieving any inflammation that may be present, as well as eliminating any contributing adhesions.



The high skin resistance pattern generally associated with the acute TMJ Pain Syndrome

Application:

- Manipulate the soft tissues in and around the inflamed zone to break any adhesions that may be present (such adhesion breaking should be extended to include the proximal portion of the sternocleidomastoideus on the involved sides, as well as over the temporalis and pterygoid areas) (refer to Soft Tissue Manipulation in Tight Areas).

- Preset the ultrasound unit a 3 (or 3.3) MHz, pulsed waveform, at 1.8 W/cm^2. Ultrasound the inflamed zone, utilizing an effective non-steroidal anti-inflammatory as a coupling agent, for six minutes. This procedure is designed to soften the adhesions that may be are present.

- Place a negative electrode over the inflamed zone and a positive electrode over the lower trapezius muscle on the same side. Preset an electrical stimulation unit to deliver a 7 Hz, wide-pulsed galvanic current and stimulate for 20 minutes. Adjust the amplitude of the stimulator to produce a visible contraction of the jaw musculature. Have the patient keep the mouth slightly open for the duration the stimulation.

- Utilizing a hand-held vibrator, gently vibrate the involved musculature for one-minute, and then the musculature on the opposing side for a minute. Have the patient keep the mouth half opened and relaxed throughout the stimulation period, and for the first five minutes following cessation of the stimulation.

The following treatment forms have also proven to be effective.

Variation:

- Preset the ultrasound unit a 3 (or 3.3) MHz, pulsed waveform, at 1.8 W/cm^2. Ultrasound the inflamed zone, utilizing an effective non-steroidal anti-inflammatory as a coupling agent, for six minutes. This procedure is designed to soften the adhesions that may be are present.

- Manipulate the tissues in and around the inflamed zone to eliminate any adhesions that may be present.

- Twenty minutes after the first ultrasound, preset the ultrasound unit to deliver a 3 (or 3.3) MHz, pulsed waveform, at 1.5 W/cm^2. Ultrasound the inflamed zone utilizing an effective non-steroidal anti-inflammatory as a coupling agent, for six minutes. This is performed to "cool off" the manipulated zone by effectively halting the production of prostaglandins by the stressed tissues.

- Apply mechanical vibration, delivered at 60 to 120 Hz, to the origin of the muscle(s) associated with the inflamed zone, for two minutes. Apply the vibration at a relatively high but tolerably comfortable level for the patient. This is performed to increase capillary circulation in the involved tissues.

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- Manipulate the tissues in and around the inflamed zone to eliminate any adhesions that may be present.

- Apply cold laser (with or without simultaneous electrical stimulation provided by the laser applicator) to the inflamed zone for approximately 6 minutes. This is performed to "cool off" the manipulated zone by effectively halting the production of prostaglandins (or facilitating enzyme destruction of **all** of the inflammatories being produced) by the stressed tissues.

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Trigger Points

The following trigger point formations may, singly or in combination, imitate or contribute to the pain associated with the *TMJ Pain Syndrome*: Masseter (deep), Masseter (superficial A), Masseter (superficial B), Masseter (superficial C), Temporalis (anterior), Temporalis (middle A), Temporalis (middle B), Temporalis (posterior supra auricular), Medial pterygoid, Lateral pterygoid, Anterior digastric, Posterior digastric, Suboccipital neck extensors, Occipitalis, Semispinalis capitis, Upper trapezius [A], Upper trapezius [B], Posterior cervical group, Splenius capitis [A], Sternocleidomastoideus (superficial fibers), Sternocleidomastoideus (deep fibers), Orbicularis oculi, Zygomaticus major, Platysma, Levator scapulae, Lower trapezius [A], Cervical multifidus (C4-C5).