TRIGGER POINT FORMATIONS

A *trigger point formation* is a palpably discrete nodule, capable of producing a distinct pattern of ***referred pain*** in a dermatome seemingly unrelated to its origin. Trigger point referred pain has been described as the "***great imitator***" of numerous conditions.

Trigger point formations may be responsible for conditions erroneously described as: headaches, migraine, toothache, jaw pain, sciatica, radiculitis, low back pain, vascular disease of the extremities, menstrual cramps, abdominal pain, earaches, tinnitus, sore throat, painful joints (arthritic pain), angina, neck pain, shoulder pain, trigeminal neuralgia, cervical dorsal outlet syndrome, pes plantis, or shin splints.

It is suggested by the author that a typical ***referred pain pattern*** associated with a particular *trigger point formation* is produced when the central nervous system confuses sensory impulses from the trigger point formation with sensory input from other areas of the body. The result is a pain that is real but not explained by routine physical examination of the locus of pain. This type of pain is amenable neither to the treatment of nerve roots supplying the dermatome housing the referred pain or of the dermatomes themselves. Muscle relaxants, tranquilizers, or most types of ***painkiller medication*** do ***not*** typically decrease trigger point referred pain.

Evidence suggests that the trigger point formation housed within a muscle is a deep, hyperalgesic, sustained, ***intrafusal fasciculation*** (spasm) involving a ***mega-muscle spindle***, composed of a high number of chain-fibers surrounding the nuclear bag fiber. These mega-muscle spindles seem to occur most commonly in muscles that perform ***joint stabilization*** a higher percentage of time than its ***prime mover*** function. These muscles are thought to be chiefly composed of ***extrafusal muscle fibers*** cytochemically classified as ***B-fibers*** (B-fibers are medium sized fibers with a variable glycogen content).

A *trigger point formation* may develop from ***muscle strain***, a draft across the overlying skin, ***extrafusal muscle spasm***, or a ***psychogenic neuromuscular mechanism***. The trigger point formation apparently produces the associated referred pain because the involved muscle spindle's ***sensory organs*** (flower-spray or ***secondary end organs***) have been provoked to generate a barrage of pain impulses, which are conducted to the spinal cord. There, they are fed into the interneuronal complex closely associated with sensory input from the ***dermatomes*** associated with the ***referred pain pattern***. The exact mechanism is still unexplained, but apparently the cerebrum is led to misinterpret the high intensity input coming from the muscle spindle as coming from the dermatome associated with the referred pain and not from the spindle (*trigger point formation*) itself.

It could be that the involved ***central nervous system*** (CNS) mechanisms receiving the sensory input from the trigger point formation are unable to appreciate contradictory information coming from the muscle (one spindle is saying the muscle is over-stressed while the muscle's other spindles are saying it is not over-stressed). Thus, it ascribes the pain impulses as coming from the ***interneuronally associated dermatome*** sensory grouping that is providing consistent sensory information. In other words, the CNS somehow concludes that since the pain is present and could not possibly be coming from the trigger point housing muscle (because of the overwhelming evidence provided by the muscle spindle majority to the contrary), it must be coming from an area of consistent sensory input, which is the ***interneuronally associated dermatome***. 1[1]

Trigger point formations may also occur in facial muscles, tendons and ligaments, which are without muscle spindles. The mechanisms that are responsible have not been clearly established or explained, but the neurological process responsible for the associated referred pain may be the same as that described above.

Although the *trigger point formation* itself does not directly produce any greater symptom than that of referred pain, if untreated (or improperly treated) the consequences may be grave, due to ***secondary developments***. If allowed to continue, the high frequency muscle spindle afferent impulses to the spinal cord may, over time, create ***abnormal motor responses*** from the lateral and anterior horn neurons, causing ***extrafusal muscle spasm*** and ***vasomotor changes*** that may cause new sources of pain. If unrelieved, this condition may gain momentum (with neuromuscular involvement) and

be very difficult to resolve, even if the causative trigger points are finally ameliorated. For example, trigger point formations housed in the upper trapezius and scalenus muscles may, through this developmental process, precipitate a ***shoulder-hand syndrome*** as muscle splinting and vascular changes progressively involve the whole upper extremity.

Physical evaluation of the trigger point has *historically* been based on the fact that a trigger point formation is a discrete, deep, hyperalgesic nodule that produces a distinctive pain pattern. The first step in evaluation is to listen to the patient's description of the pain pattern, so that it might be compared with known trigger point referred pain patterns. The next step is to confirm the presence of a *trigger point formation* by applying a firm discrete pressure into the site where the *trigger point formation* is typically located. If the trigger point is active, applied pressure into the point will provoke ***one*** of several responses. (1) It will immediately relieve the pain pattern. (2) It will relieve the pain pattern and cause excruciating pain at the site of the trigger point. (3) It will increase the intensity of pain pattern and cause an excruciating pain at the site of the trigger point. *No change* in the pain pattern indicates a negative response and the absence of an active trigger point at that site. Additionally, trigger points may *often* be determined through differential skin resistance (DSR) survey; i.e., the skin resistance directly over the trigger point may be comparatively high and typically encompass a four centimeter square area.[2]

Trigger points may occur as primary or secondary points. Secondary points generally occur within the referred pain pattern produced by a primary or initially occurring trigger point (the first to show up) and are thought to result from the vascular changes triggered by the initial referred pain pattern, as mentioned above. Primary and secondary trigger points may effectively combine their respective referred pain patterns (which may or may not overlap) to produce the total pain pattern described by the patient. Primary trigger points are generally responsible for the initial experience of the referred pain, and the secondary trigger points usually appear later, in muscles that lie inside the referred pain pattern produced by the primary trigger point.

A clear differentiation between primary and secondary formations is important and should be made. Direct pressure into a primary trigger point will more significantly affect the pain pattern than will the same pressure into a secondary trigger point. Like primary trigger points, secondary trigger points are hypersensitive areas, tender or painful on palpation, but direct pressure applied into them will not appreciably diminish or magnify the referred pain to any great extent. When it has been established which of the *trigger point formations* are primary, treatment efforts should be directed at relieving them. When primary *trigger point formations* have been relieved, secondary points may spontaneously disappear, especially if the muscles housing the secondary trigger points can be strengthened and toned through exercise or electrical stimulation.

Treatment

Clinical experience has suggested that treating the inflammatory process associated with a trigger point, just as any other soft tissue inflammation, may enhance the reduction of the trigger point. It has also been noted that adhesions are often found over and around both relatively acute and chronic trigger points and that soft tissue manipulation of these adhesions will often completely relieve the trigger point and its referred pain pattern.1[1],2[2]

Treatment of the **acute** *trigger point* referred pain involves lengthening the trigger-point-housing-muscle and inhibiting tonic intrafusal muscle contraction. To accomplish this, put the involved muscle on stretch and then ice pack (or ice massage) the trigger point site. Alternatively, the trigger point(s) can be electrically stimulated, brushed, or muscular antagonists vibrated to reduce it or them. Reducing **chronic** trigger point formations may depend on treating attendant inflammation over the trigger point as well as breaking any adhesions that have developed. Furthermore, toning the trigger point housing muscle through isometric and isotonic exercise may be necessary.

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[1] The opinions expressed above, as to the origin and the mechanisms responsible for the phenomena of the trigger point formation and referred pain patterns are not those of the primary investigator who has been most responsible for the study and exploration of trigger point referred pain, Janet G. Travell, M.D.

[2] Trigger points may be present without apparent inflammation, as measured by DSR survey.