

**DYSPONESIS: AN IMPORTANT FACTOR IN PTSD**

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## DYSPONESIS: AN IMPORTANT FACTOR IN PTSD

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George Whatmore (Smith, 1987), introduced a new word, "dysponesis" which he defined as the misuse of energy. His basic focus was on the misuse of motor energy; preparatory tension prior to movement, chronic muscle contraction as a bracing against injury and the recruitment of whole muscle groups in cases of excessive hypervigilance. He noted that dysponesis was an important neurophysiological factor in functional disorders.

This paper examines dysponesis in chronic post-traumatic stress conditions resulting from childhood trauma. We specifically look at the etiology of trauma induced dysponesis, the destructive effects of this condition, the incorporation of these effects in dissociation using the manipulation of conditioned stimuli and conditioned responses derived from the unconditioned stimuli and responses comprising the early experiences of trauma. We give particular attention to the interaction between cognitions, affect and behavior, both internal micro behavior and external operant behavior.

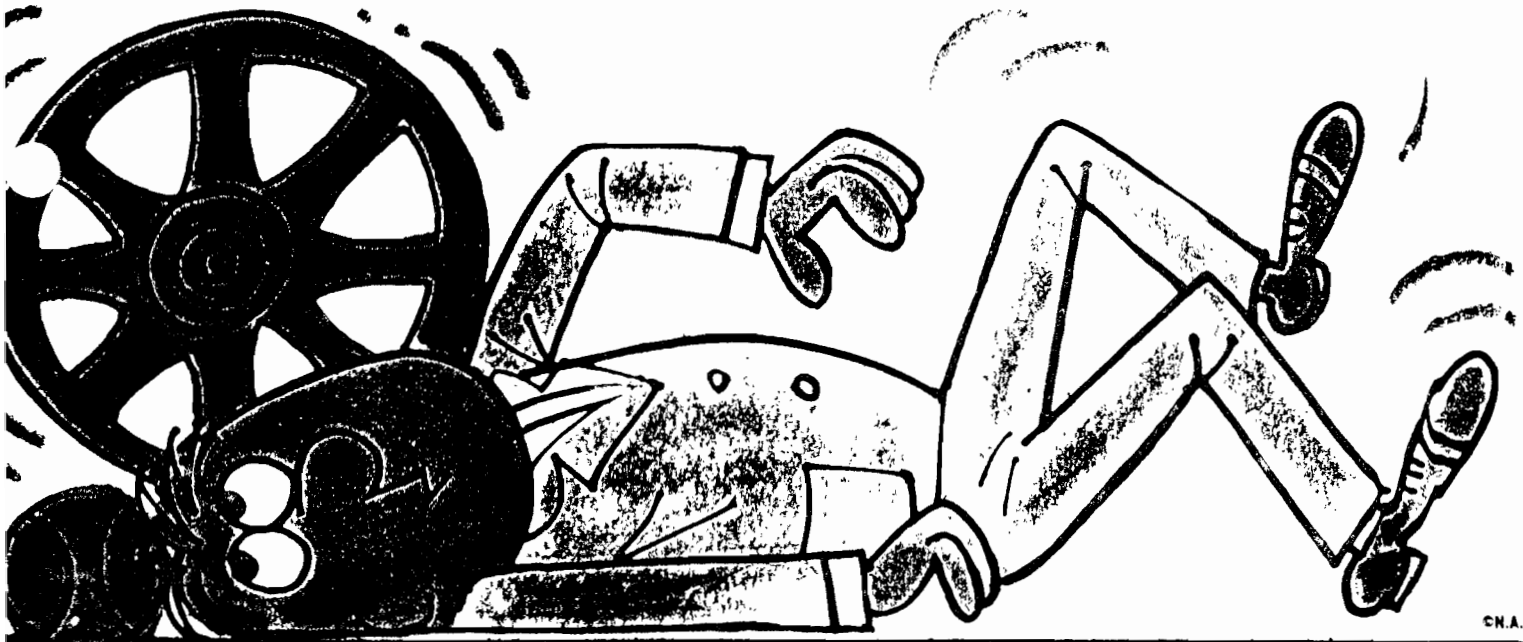
Our basic thesis is that chronic dysponesis is physically painful and is retraumatizing which gives credence to the idea that chronic PTSD is a somatopsychic condition rather than a psychosomatic one. Smith (1987) indicated that chronic retraumatization greatly undermined the capacity of the neocortex to conceive and direct rational responses to both threatening and non-threatening situations. The cortex is reduced to ratifying sub-cortically mediated behavior that is dysponetic and dissociative. This continuation in dysponetic dissociation makes it virtually impossible for rational neocortical ability to return. Thus we feel that the behavioral and affecting components of dissociation are as powerful or more powerful than the cognitive aspects and that cognitive modification is not sufficient for treatment.

The physical pain of dysponetic responding and emotional distress must be addressed to bring about sufficient soothing, comfort, and control, so that cognitive modification becomes possible. The following section provides detailed information on the myofascial mechanisms of dysponesis necessary for developing an adequate treatment protocol.

# DYSPONESIS

KEEP YOUR **EYE** ON THE **BALL**  
YOUR **SHOULDER** TO THE **WHEEL**  
YOUR **EAR** TO THE **GROUND**

now try to work in that position!



## MECHANISMS OF DYSPONESIS

The actual mechanism by which conditioned emotional stimuli produce their effect is a physical response, the flexor withdrawal reflex, described by Wilson (Smith, 1987). When an area of the body is in pain, this reflex acts to increase the muscle tension around, and diminish the flow of the blood to the pain site. This serves to "splint" the injured area and constrict the blood vessels flowing to it thus preventing hemorrhage. This response protects a child by withdrawing the painful area and keeping it from further movement.

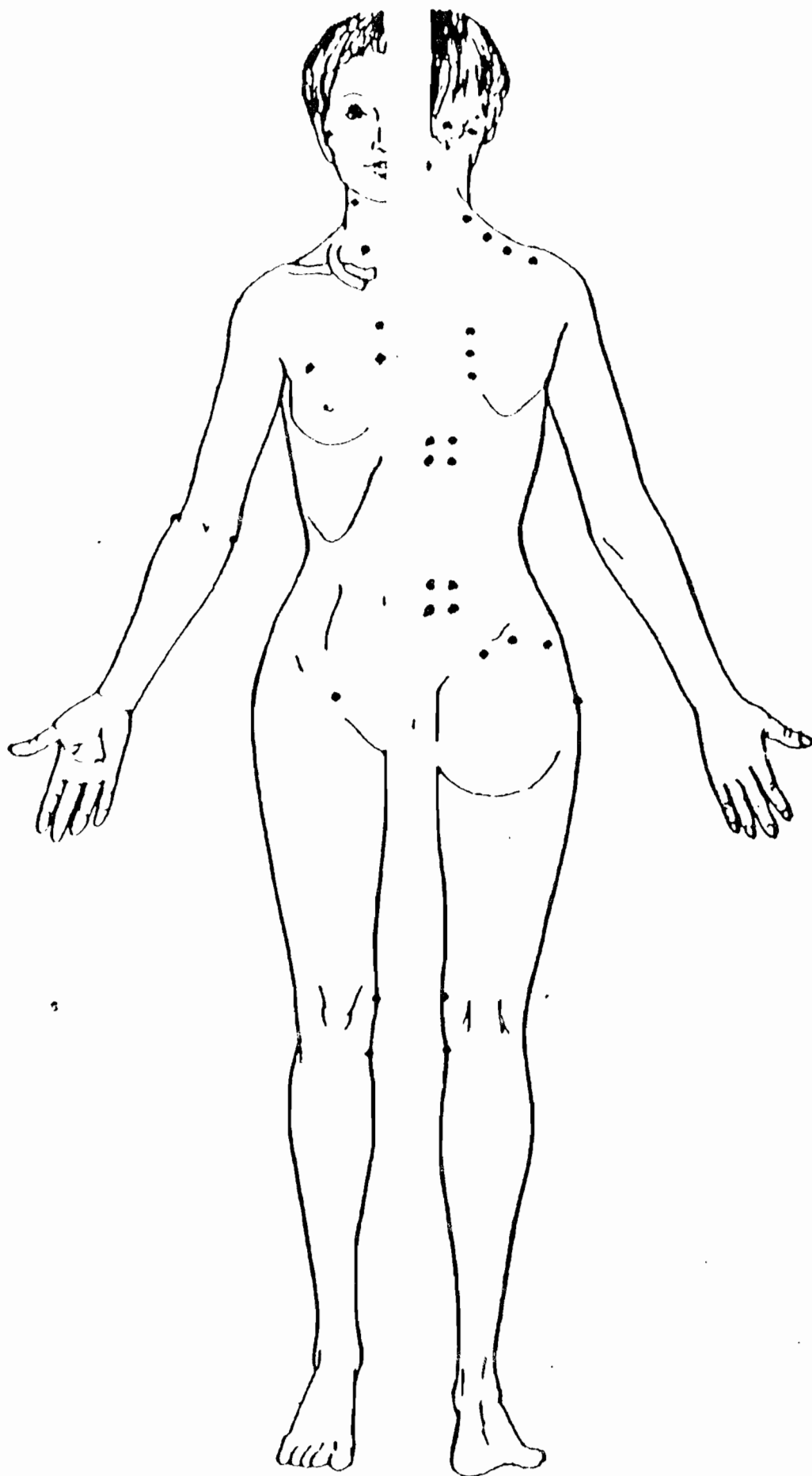
Wilson notes that when a pattern of muscle contraction becomes well established, small, very painful knots of muscle (trigger points) begin to form in muscles in and around the pain sites as well as in muscle groups that "splint" the injured area. These painful trigger areas tend to accumulate muscle contraction waste products since wastes cannot be pumped back out through constantly constricted muscles. Thus a cycle of pain eliciting reflex spasm with sequelae and the resulting trigger points produce pain to again initiate the muscle contraction.

This cycle is repeatedly reinforced by the experience of abuse combined with strong prohibition of expression of the natural (unconditioned) responses to the abuse, ie. crying, protest, fight, flight, etc. As a result the muscles of expression become chronically contracted to inhibit observable responses. This mechanism therefore, is an integral part of the dissociative process.

The traumatized child learns to contract (clench) muscles of the face, throat, diaphragm, arms, legs and pelvis to control all expressions. This chronically contracted state becomes the norm and the basis for "body memories." The concept of "body memory" infers that nociceptive mechanisms are not activated, when in fact they are. The myofascial trigger points and ischemia in chronically contracted muscles evoke characteristic referred pain and autonomic phenomena. This chronic activation becomes an integral part of the dissociative pattern in which somatic symptoms become a primary stimulus of emotional and cognitive upheaval by way of the somatosensory nervous system. Conversely, cognitive upheaval, visual images and auditory manifestations of emerging traumatic memories trigger maximal autonomic arousal which in turn activates trigger points and splinting of the related myofascial components.

Once this trigger point state has occurred, symptoms associated with specific trigger points can be activated by stress, temperature changes, fatigue, body position, nutrition, poor sleep, etc. In addition to these naturally occurring conditions, these factors can be deliberately induced by perpetrators of abuse to shape specific behavioral responses. Primary Fibromyalgia, a myofascial pain syndrome, is an example of a condition involving wide spread trigger points and is a common disorder found in survivors of childhood trauma.

# PRIMARY FIBROMYALGIA

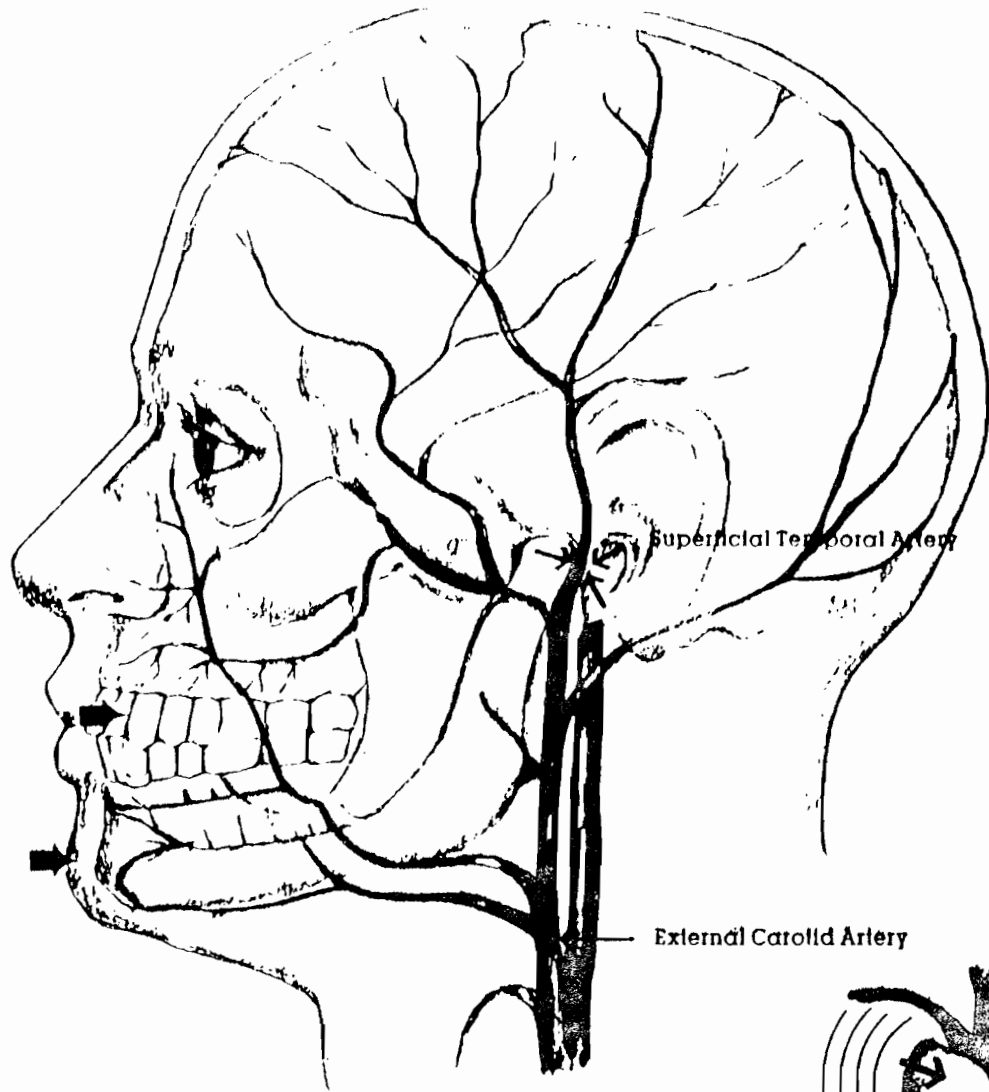


Further examples of the way in which "clenched" muscles of expression generate somatic symptoms mislabeled as "body memories" are:

1. **Temporomandibular joint disorders** - chronic clenching; pain, facial and cervical muscular contraction and biomechanical joint dysfunction combine to elicit a wide range of symptoms. Posterior joint position can cause inflammation of the joint capsule and irritation of the nerve supply. In addition, compression of the blood vessels and trigeminal nerve can occur.
2. **Sternocleidomastoid muscle** is the site of many chronic trigger points. These trigger points, besides causing severe pain, can also cause visual disturbances, dizziness, nausea and unsteadiness when walking.
3. **Scalenus and pectoralis minor** when contracted can entrap the blood vessels and nerves coming from the neck going down the arm. Elevation of the arm relieves scalene entrapment but increases compression by the pectoralis minor. When the arm is at the side scalene entrapment worsens.
4. **Sternocleidomastoid - sternalis - rectus abdominus** have a relationship to one another which can trigger symptoms from one to another. The sternalis trigger points can cause coughing while rectus trigger points can cause intestinal symptoms. This relationship is profoundly affected by bracing the diaphragm.
5. **Diaphragm splinting/bracing** limits respiration and acts to hold down emotional responses. Breathing requires use of accessory muscles when diaphragm movement is limited.
6. **The trapezius muscle** is frequently contracted causing pain referral patterns and can also trigger nausea.

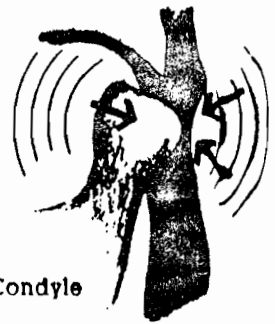
# Arteries of the Head

## POSTERIOR CAPSULITIS

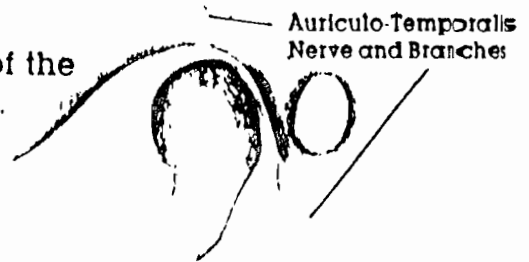


Retruded Condyles reduces the circulation to the TMJs and head.

Retruded Condyle



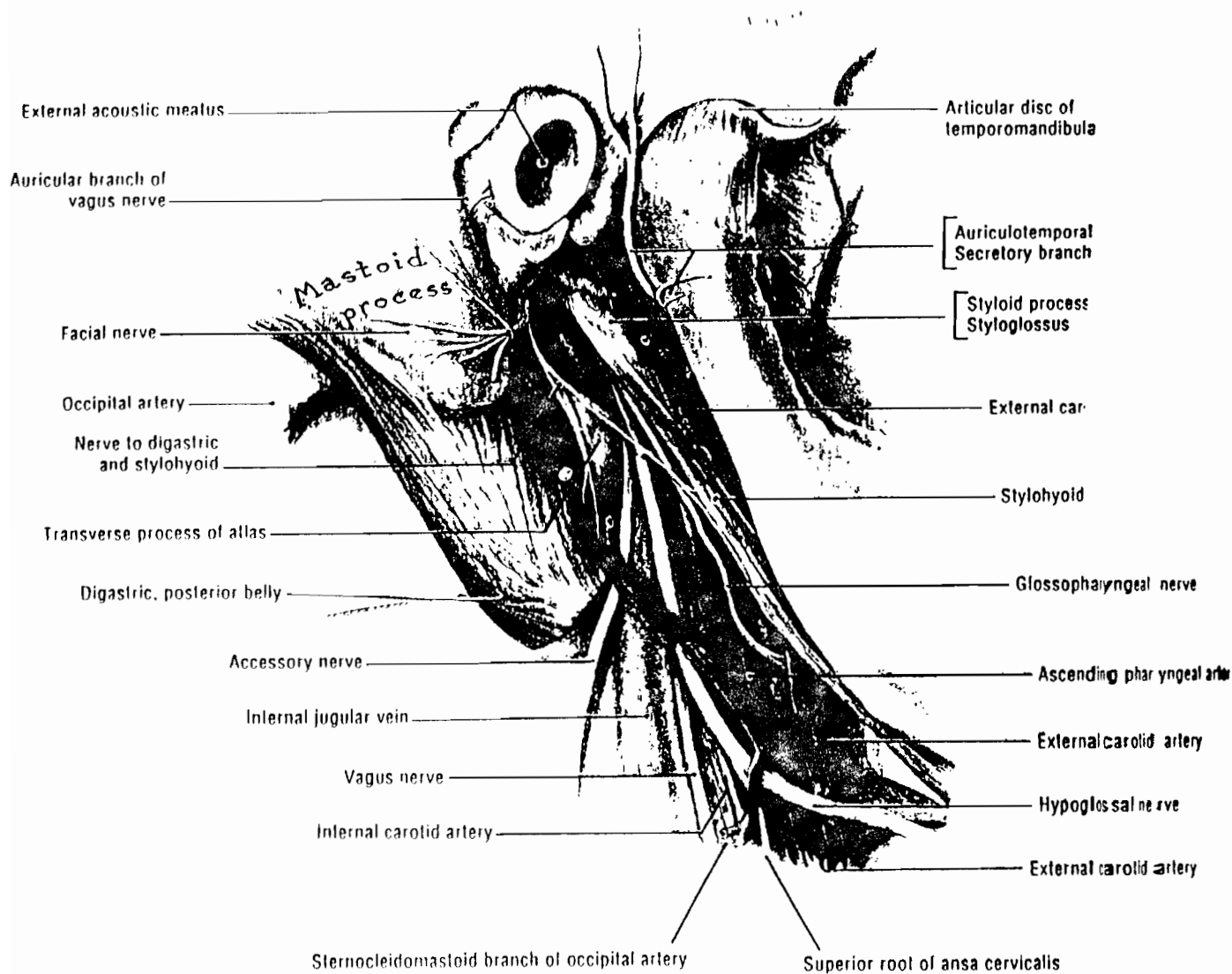
Retruded Condyles compress the nerves of the Temporomandibular Joint.



*John W. Witzig, D.O.S.*

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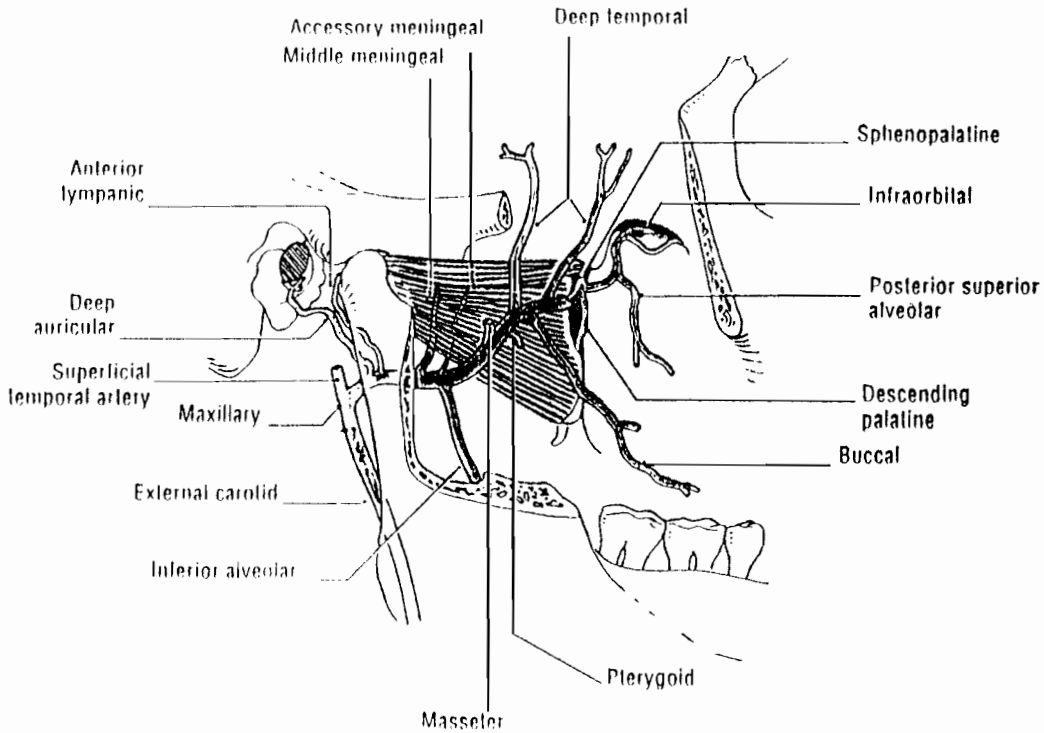
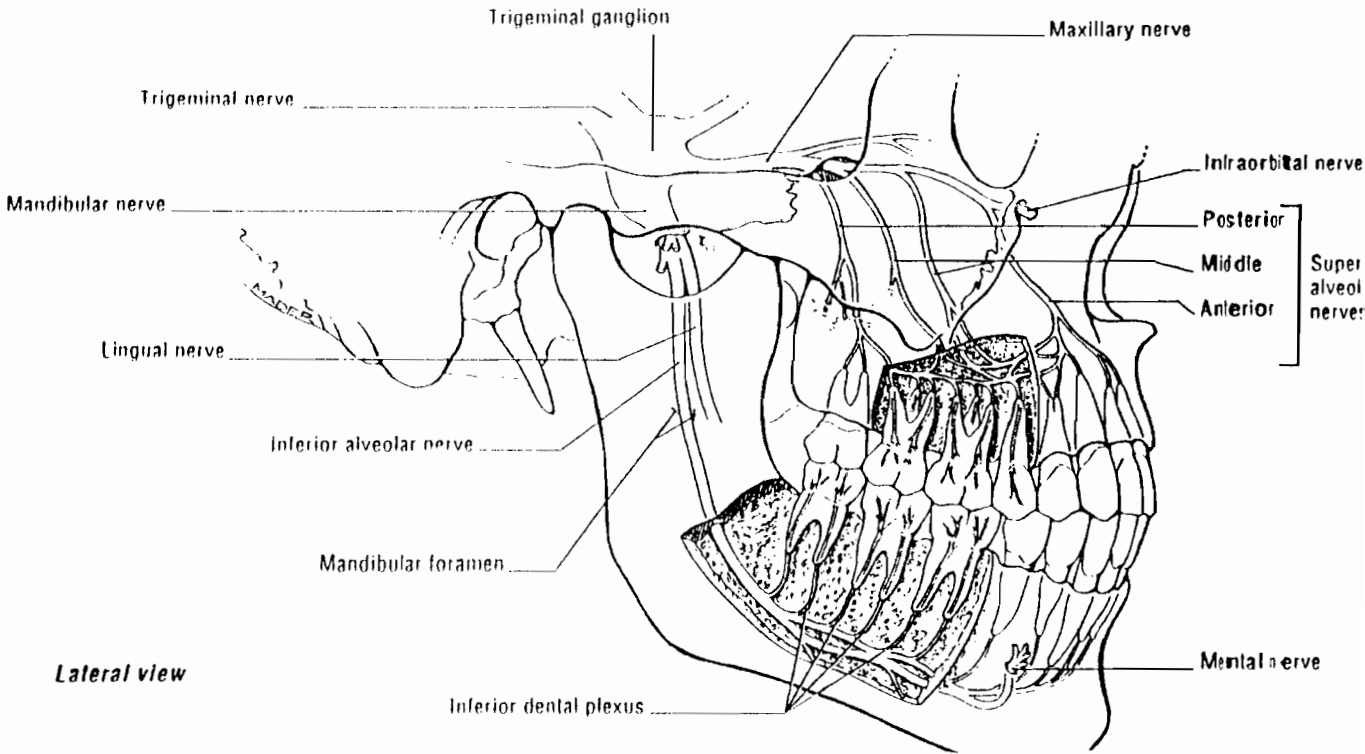
# NERVE AND CIRCULATORY COMPRESSION FROM CHRONIC MUSCLE TENSION (DETAIL 1)



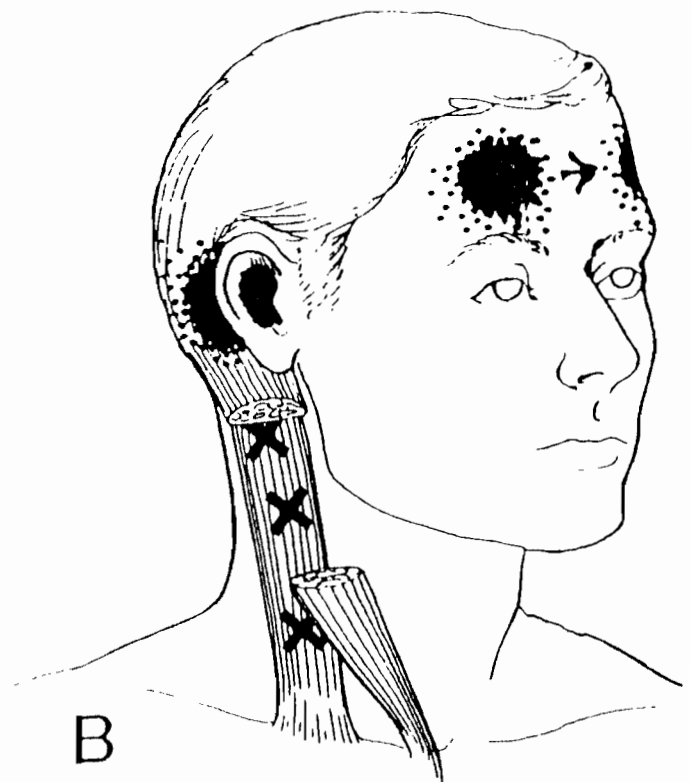
**Figure 7-73.** Dissection of the structures deep to the parotid bed. The facial nerve, the posterior belly of the digastric, and the nerve to it are retracted



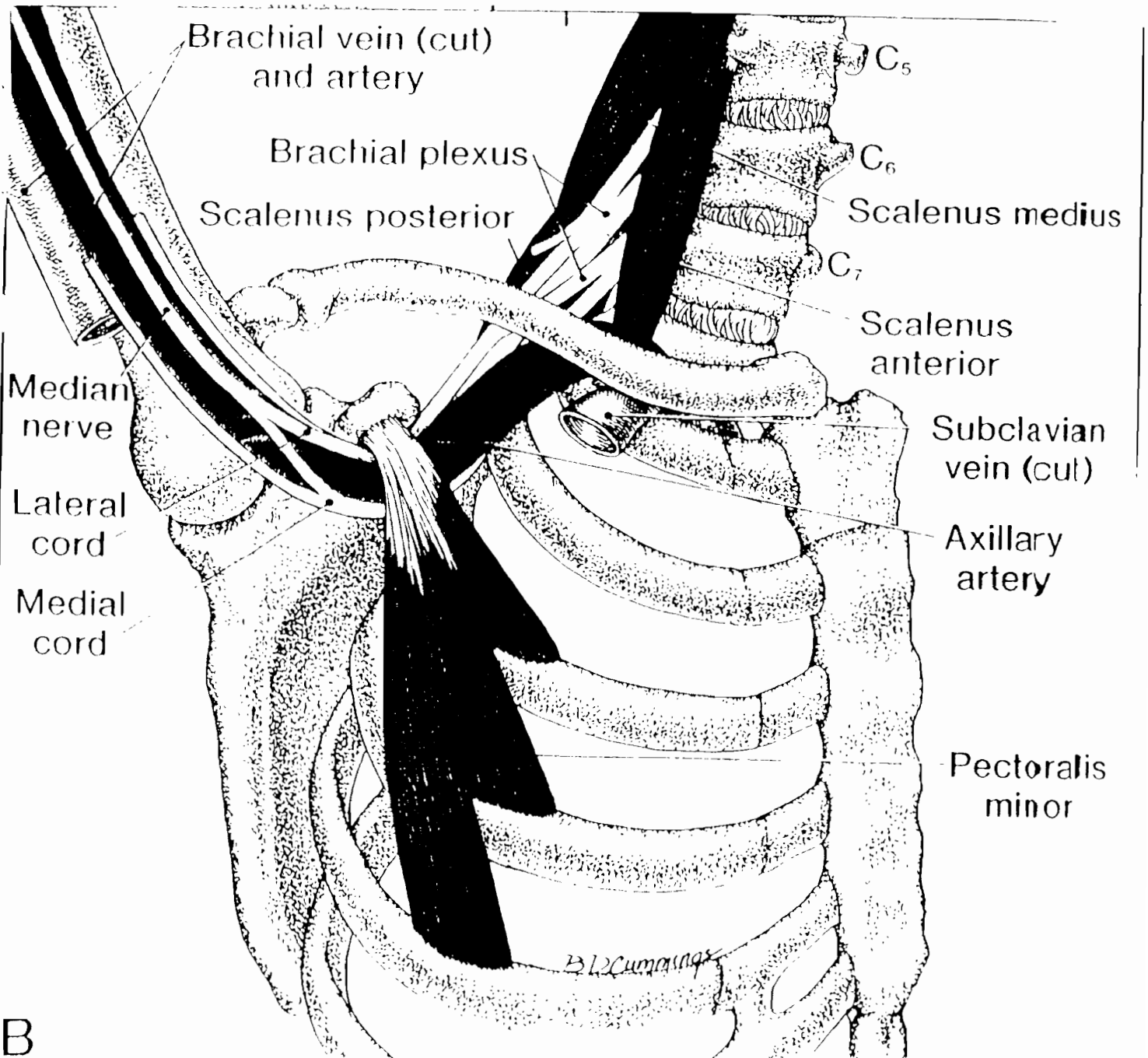
# NERVE AND CIRCULATORY COMPRESSION FROM CHRONIC MUSCLE TENSION (DETAIL 2)



# REFERRED PAIN PATTERN SCM TRIGGER POINTS

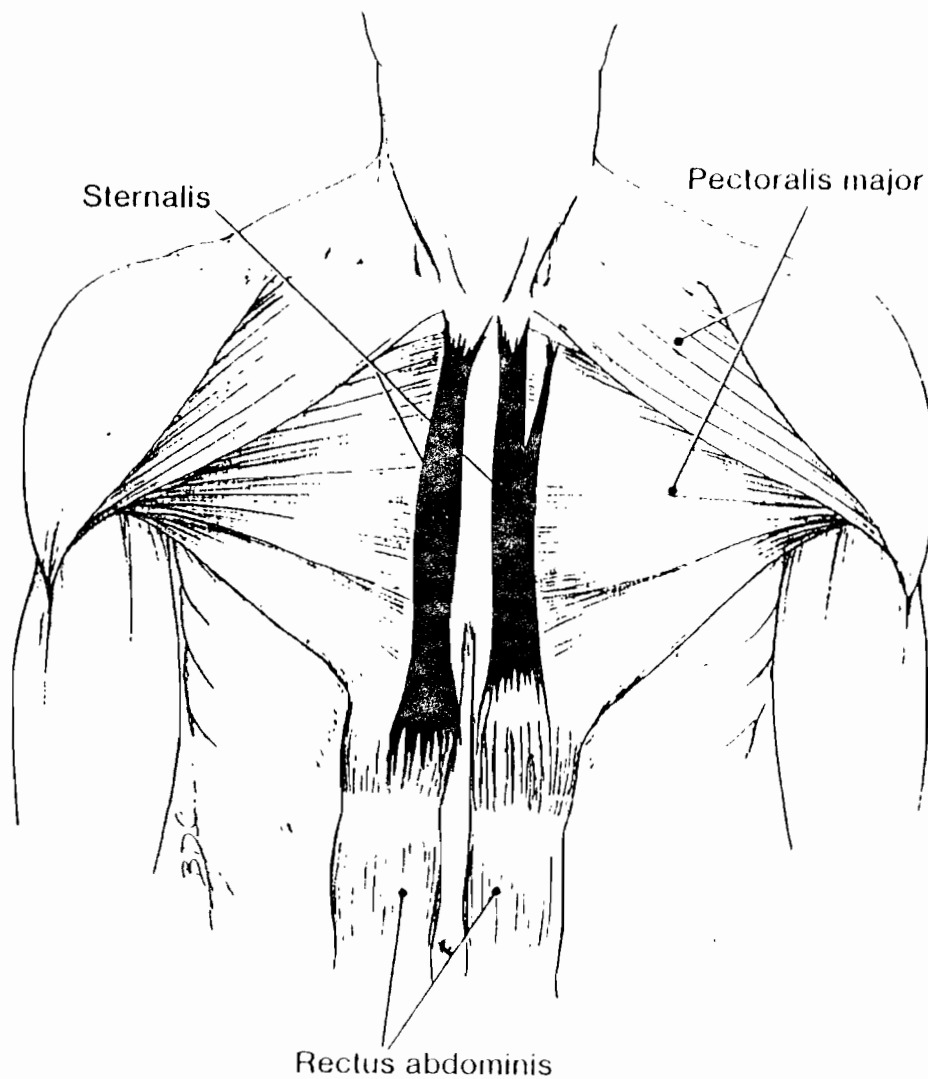


# ENTRAPPED NERVES AND BLOOD VESSELS BY CONTRACTED MUSCLES

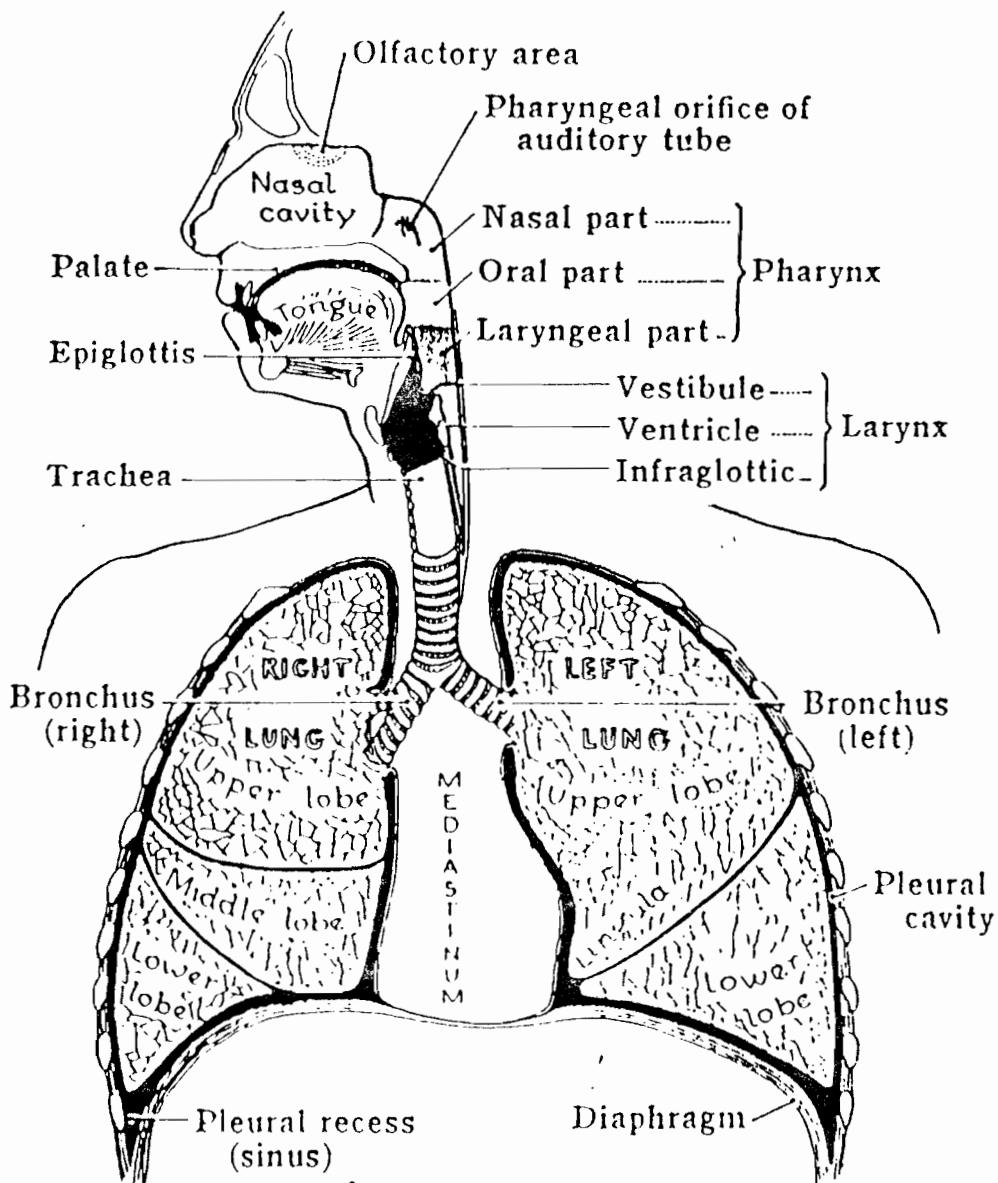


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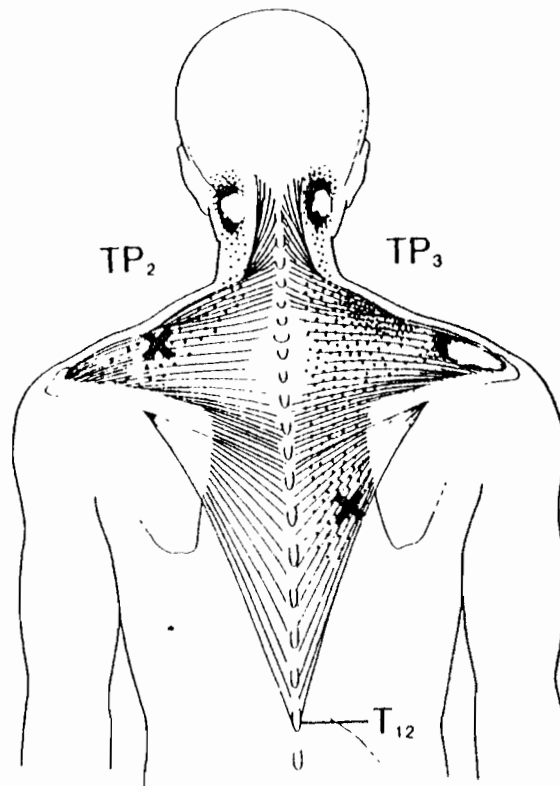
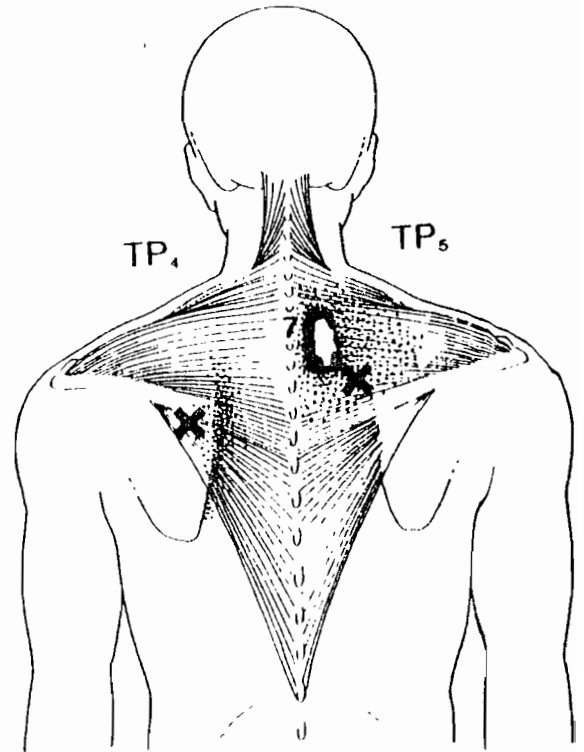
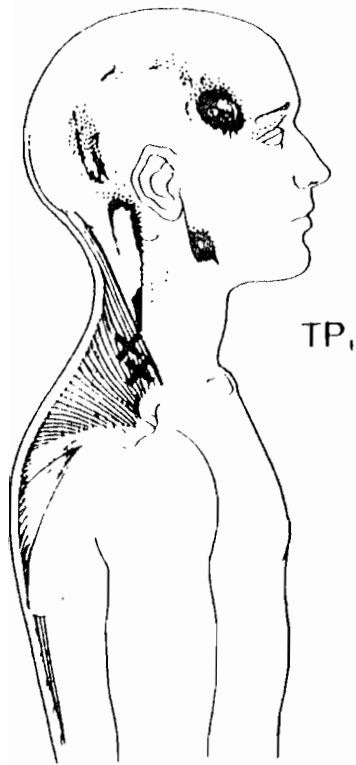
# NORMAL ARRANGEMENT OF MUSCLE GROUPS



# BREATHING PROBLEMS AND THE DIAPHRAGM



# PRIMARY TRIGGER POINTS AND REFERRED PAIN PATTERN OF TRAPEZIUS MUSCLE



### IMPLICATION FOR TREATING

A unified approach in the treatment of the behavioral (physiological - somatic), emotional and cognitive aspects of PTSD requires a considerable amount of future research. However, certain important points can be noted. Therapists monitoring muscle activity and autonomic responses report a concomitant flair up in myofascitus in the uncovering of traumatic issues in patients with PTSD/MPD. Extremely elevated levels of muscle activity can be demonstrated via Electromyogram feedback monitoring as stressful material emerges. In addition, direct examination of the muscles

involved in the pain referral pattern at the time provides objective evidence of myofascial symptoms. These symptoms when evaluated outside the psychiatric arena are treated with medication, trigger point injection, biofeedback, and physical therapy. Hopefully, this same opportunity for physical assessment and treatment will be afforded the psychiatric patient.

MRS:KT:dd  
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## References

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- Smith, M. R. (1987). Post-Traumatic Stress and the Loss of Ontological Security. Chicago: Proceedings of the 4th International Conference on MPD and Dissociation.