

**THE INVOLVEMENT OF THE STYLOID PROCESS  
IN HEAD AND NECK PAIN - A PRELIMINARY STUDY**

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## A STUDY OF THE STYLOID PROCESS AND HEAD AND NECK PAIN

### ABSTRACT

The styloid process and associated structures have been implicated in a variety of craniomandibular dysfunctions and pain complaints. Treatment directed at this area can result in a dramatic reduction in referred symptoms, somatic pain and autonomic signs as well as an increase in mandibular range of motion. In the past, an elongation of the styloid process was considered necessary for pain and dysfunction symptoms to arise from this area. The patients in this study did not have elongated styloid processes, yet had orofacial pain and dysfunction symptoms seemingly referred from this area. An injection of local anaesthetic and corticosteroid in the area of the styloid process in these patients statistically significantly reduced lateral head pain and marginally improved maximum jaw opening. The data suggest that in at least some patients with craniomandibular disorders, some form of inflammatory lesion may exist in the vicinity of the styloid process. It is hypothesised that an inflammatory process, induced by trauma, is present at the styloid enthesis and that this process can be reversed by an injection of local anaesthetic and corticosteroid.

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## ABSTRACT

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N.A. indicates the patients that could not be contacted for their one year follow up.

\*One patient was undergoing a course of orthodontic therapy that restricted the mandibular opening measurement.

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## 1. INTRODUCTION

In the clinical examination of patients with facial pain and mandibular dysfunction and/or pain in the cervical region, investigation of the styloid process is rarely included. Consequently, styloid process pathology is usually overlooked as a possible source of symptom referral. However, there are a range of symptoms that have been associated with an involvement of the styloid process and its associated structures. These symptoms are summarized in Table 1.

Most of the reports concerning styloid process initiated pain have been case reports of patients with elongated styloid processes, or Eagle's syndrome, without long-term follow up or standardised evaluation methods. However, elongated styloid processes are a relatively rare occurrence. Even more rare are elongated styloid processes that have become symptomatic (Harma 1967; Correll et al 1979). Therefore, it is of interest that patients with orofacial pain and dysfunction, yet without evidence of lengthened styloid processes, list symptoms that are very similar to many of the symptoms described as emanating from an elongated styloid process.

It is the purpose of this study to determine if a non-elongated (ie normal) styloid process can act as a source of pain. If so, then why does a "normal" styloid process become painful and can it be held responsible for some of the symptoms of patients with orofacial pain complaints? The hypothesis of a trauma-induced, occult, soft tissue injury at the styloid process that can result in an inflammatory condition capable of referring symptoms to the ipsilateral head and neck areas will be presented.

<u>DISCIPLINE</u>	<u>DIFF. DIAGNOSIS</u>	<u>REFERENCE</u>	<u>POSSIBLE PATH.MECH.</u>	<u>TREATMENT</u>
<u>ENT</u>				
	<b>DYSPHAGIA</b>	BUTLER and TARSITANO (1969) CHANDLER (1978) EAGLE (1948,1949,1958) FIELDING and COLLINS (1976) FROMMER (1974) KAHL and HARTMEIN (1990) LEIGHTON et al(1991) LINDEMAN (1985) McCORKELL (1985) STEINMAN (1968,1970) OZAWA et al (1995) RUSSELL (1977)	ELONGATION ELONGATION  ELONGATION  ELONGATION FRACTURE INSERTION TENDINOSIS ELONGATION ELONGATION	RESECTION RESECTION  EXCISION  RESECTION EXCISION INJECTION RESECTION
	<b>DYSPHONIA</b>	SCHMIDT (1951) STEINMANN (1968,1970)	ELONGATION INSERTION TENDINOSIS	RESECTION INJECTION
	<b>PHARYNGEAL PAIN</b>	BOEDTS(1978)		
	<b>GLOSSITIS</b>	BUTLER and TARSITANO (1969) EAGLE (1948,1949,1958)	ELONGATION ELONGATION	RESECTION RESECTION
	<b>REFERRED OTALGIA</b>	MURTLY et al(1990) SIVERS and JOHNSON (1985) CHIEN et al (1991) STEINMANN (1968, 1970) WOOLERY (1990) KRESPI et al (1981)	ELONGATION ELONGATION ELONGATION INSERTION TENDINOSIS ELONGATION	RESECTION RESECTION RESECTION INJECTION RESECTION
	<b>TONSILLITIS</b>	HARMA (1967) GASIOREK (1989)	ELONGATION	
	<b>EAR PAIN</b>	EAGLE (1948,1949,1958) FIELDING and COLLINS (1976) LINDERMAN (1985) RUSSELL (1977) SHANKLAND (1987)  STEINMANN(1968,1970)	ELONGATION ELONGATION ELONGATION ELONGATION INERTION INJURY  INSERTION TENDINOSIS	RESECTION RESECTION RESECTION  INJECTION, THERMOLYSIS INJECTION
<u>DENTISTRY</u>				
	<b>ATYPICAL FACIAL PAIN</b>	CHIEN et al (1991)  BALASUBRAMANIAN (1964) INOVAY and KATONA (1992) KRESPI (1981)  RIEDIGER and EHRENFELD (1989) SCHROEDER (1991) WINKLER et al (1981) ZOHAR et al (1986) NISHIHARA et al (1986)	ELONGATION  OSSIFICATION  ELONGATION TENDINOSIS  FRACTURE ELONGATION ELONGATION ELONGATION	RESECTION    RESECTION, INJECTION  EXCISION RESECTION RESECTION RESECTION
	<b>HEADACHES</b>	CHIEN et al (1991) DOLAN et al (1984) EAGLE(1948,1949,1958) ITOH et al (1986) KELLY (1975) LENGELE and DHEM (1980) SHANKLAND (1987)	ELONGATION ELONGATION ELONGATION ELONGATION ELONGATION ELONGATION INSERTION INJURY	RESECTION RESECTION RESECTION RESECTION  RESECTION INJECTION THERMOLYSIS
	<b>DENTAL PAIN</b>	SATALOFF and PRICE (1984) EAGLE (AS ABOVE)	LIGAMENT INJURY ELONGATION	RESECTION

<b>TMJ PAIN</b>	EAGLE (AS ABOVE) DOLAN et al (1984) ERNEST (1986) CHRISTIANSEN et al (1975) GOSSMAN and TARSITANO (1977) KEGLEVICH and GERE (1990) SCHROEDER (1991) SHANKLAND (1987)	ELONGATION ELONGATION TENDON INJURY	RESECTION RESECTION INJECTION
	WONG et al (1995) OZAWA et al (1995)	FRACTURE INSERTION INJURY  TENDON INJURY ELONGATION	EXCISION INJECTION THERMOLYSIS INJECTION RESECTION
<b>NEUROLOGY</b>			
<b>NEURALGIA</b>	CHRISTIANSEN et al (1975) DVORSKI (1986) EAGLE (AS ABOVE) LOESER and CARDWELL (1942) NISHIHARA et al (1986) SATALOFF and PRICE (1984) MONTALBETTI et al (1995) OZAWA et al (1995)	ELONGATION	RESECTION
		LIGAMENT DISTENSION ELONGATION ELONGATION	RESECTION RESECTION
<b>CAROTIDYDYNIA</b>	EAGLE (AS ABOVE) LOCH et al (1990) SHENOI (1972) MONTALBETTI et al (1995)	ELONGATION VASCULAR IMPINGEMENT  ELONGATION	RESECTION  RESECTION
<b>CERVICAL DYSFUNCTION</b>	HAAS et al (1991)	ELONGATION	RESECTION
	KRESPI et al (1981)  McCORKELL (1985) SIVERS and JOHNSON (1985) STEINMANN (1968,1970)	ELONGATION TENDINOSIS  FRACTURE ELONGATION INSERTION TENDINOSIS	RESECTION INJECTION EXCISION RESECTION INJECTION
<b>VERTIGO</b>	CORRELL et al (1979) DOLAN et al (1984)	MINERALISED LIGAMENT ELONGATION	RESECTION

TABLE.1

Brief, not exhaustive account of published studies on styloid process related pain.

## 2. ANATOMY

### 2.1 Structure, Position and Form:

The styloid process is attached to the inferior aspect of the petrous part of the temporal bone, immediately below the tympanic membrane and immediately behind the tympanic plate which shields its attachment. It lies just behind the pharyngeal wall in the area of the palatine fossa between the internal and external carotid arteries. In close proximity is the glossopharyngeal nerve lying in the posterolateral wall of the tonsillar fossa. The facial nerve emerges from the stylomastoid foramen that is slightly posterolateral to the base of the styloid process (Boedts, 1978, Dolan et al, 1984).

Medial to the process is the internal jugular vein together with the accessory, hypoglossal, vagus and glossopharyngeal nerves. The internal carotid artery with the sympathetic chain is also medial to the styloid process (Fig. 1).

A styloid process of normal length is commonly regarded as 2.5 cm long (Fig. 2B). The styloid process is considered elongated if it is greater than 3 cm in length. An elongated styloid process can usually be palpated in the tonsillar fossa and can be confirmed from an OPG X-ray (Fig. 2A).

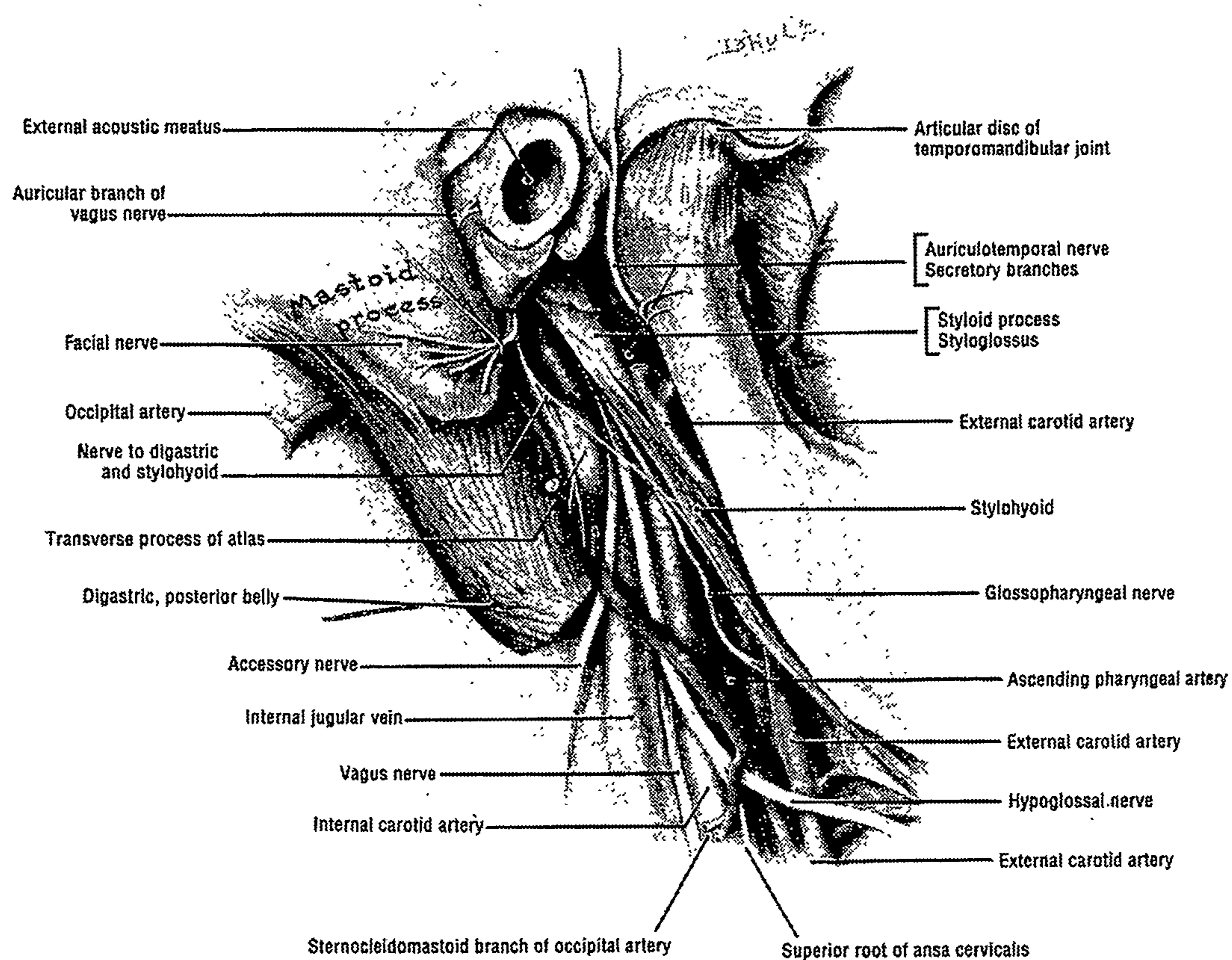


Fig. 1 Dissection of the structures deep to the parotid bed. The facial nerve and the posterior belly of the digastric muscle have been retracted to show the structures surrounding the styloid process. (from Moore, 1992)

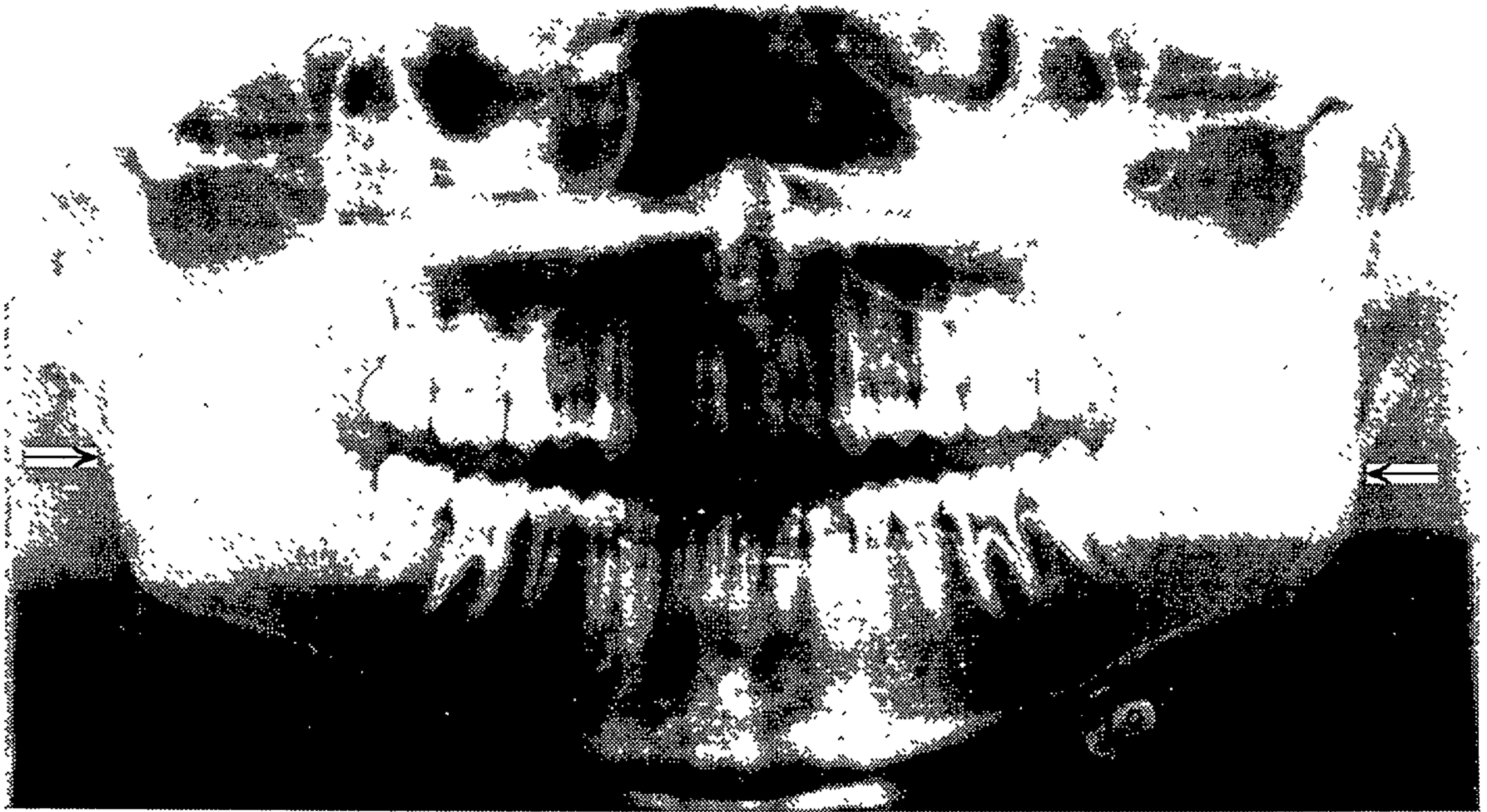


Fig. 2A



Fig. 2B

Fig. 2 Orthopantomograms (OPG's) showing elongated (arrows, A) and normal length (arrows, B) styloid processes.

## 2.2 Anatomic connections:

The following muscles have their origins at the styloid process:

1. The stylopharyngeus muscle originates on the medial side of the root of the styloid process and inserts into the superior and posterior borders of the thyroid cartilage.

Some fibres mingle with those of the constrictors of the pharynx. Its functions include raising and dilating the pharynx. The stylopharyngeus is innervated by the glossopharyngeal nerve.

2. The styloglossus muscle originates from the anterior border of the styloid process and inserts into the sides of the tongue. Its fibers spread and mingle with the palatoglossus and hyoglossus muscles. This muscle retracts the tongue with the aid of the anterior fibres of the genioglossus and elevates the tongue with the aid of the palatoglossus. It is innervated by the hypoglossal nerve.

3. The stylohyoid muscle originates from the posterior border of the styloid process near its base and inserts into the body of the hyoid bone at the junction with the greater horn. It elevates the hyoid bone and the base of the tongue. The stylohyoid muscle is innervated by the stylohyoid branch of the facial nerve.

The following ligaments have their origins at the styloid process:

1. The stylohyoid ligament inserts into the lesser cornu of the hyoid bone.
2. The stylomandibular ligament inserts into the angle of the mandible.
3. The stylopharyngeus ligament inserts into the posterior pharyngeal wall aponeurosis.

Generally, the muscles attach superiorly to the styloid process and the ligaments attach inferiorly to the process. The anatomic sites of attachment for all the soft tissues are called entheses. At these loci, the collagenous extra-osseous fibres are continuous with the Sharpey's fibres of the bone itself. The enthesis complex (ie periosteum, ligaments and muscles) is a richly innervated area. This feature together with its vulnerability to inflammatory disorders, makes the enthesis an interesting area to clinicians. Of interest is the possibility of referral arising because of central convergence of somatosensory input from afferent fibres in the many different nerves supplying the area. The muscles and ligaments from the styloid process are supplied by branches of the hypoglossal, glossopharyngeal and facial nerves. The area surrounding the styloid process is supplied by the third cervical nerve. The overlapping sites of termination of trigeminal and cervical nerves (e.g. Fig. 3) provides a possible neural basis for an injury at the styloid process to refer pain to structures supplied by the trigeminal nerve. A styloid process injury would trigger an afferent nerve barrage directed at the dorsal horn and the trigeminal spinal nucleus and could influence central neurones also in receipt of cervical spinal afferents. The question of peripheral and central sensitisation will be discussed in the section on neurophysiological aspects of chronic pain.

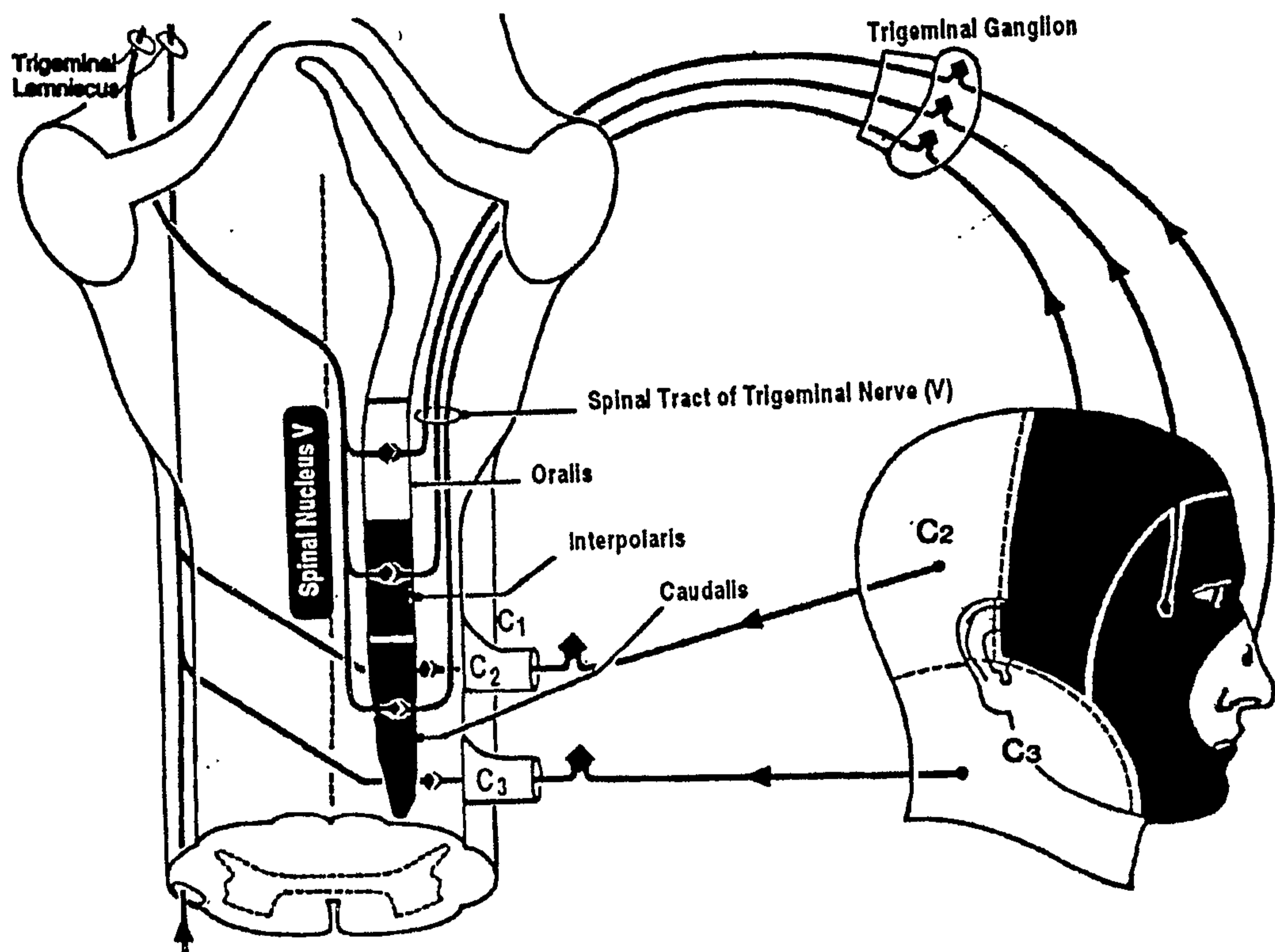


Fig. 3 Proposed Trigemincervical interactions at the spinal cord level. (diagram courtesy of Dr. Samuel Quek, lecturer at the TMJ/Orofacial Pain Center UMD-New Jersey Dental School)

### 3. EMBRYOLOGY:

Embryologically, the styloid process belongs to the hyoid system and is first formed in cartilage (Steinman, 1970). Reichert's cartilage, which is derived from the second branchial arch, in conjunction with the third branchial arch, develops downward and medially towards the contra-lateral cartilage and gives rise to four centers which form the styloid and hyoid processes. The first three arches of this system undergo ossification.

The tympanohyal process is calcified at birth but is not yet attached to the temporal bone. The attachment is believed to take place during the first year of life. Dwight (1907) found that the tympanohyal process may not protrude at all from the surface of the temporal bone yet at other times it may protrude considerably. Often, the connection of the tympanohyal process with the temporal bone remains comparatively long in a cartilagenous state, sometimes until adult age (Steinman, 1970). The apparent variability of this growth center can account for the large numbers of morphological differences noted. It may even account for the high incidence of completely absent styloid processes found in one study (Anson and McVay, 1971).

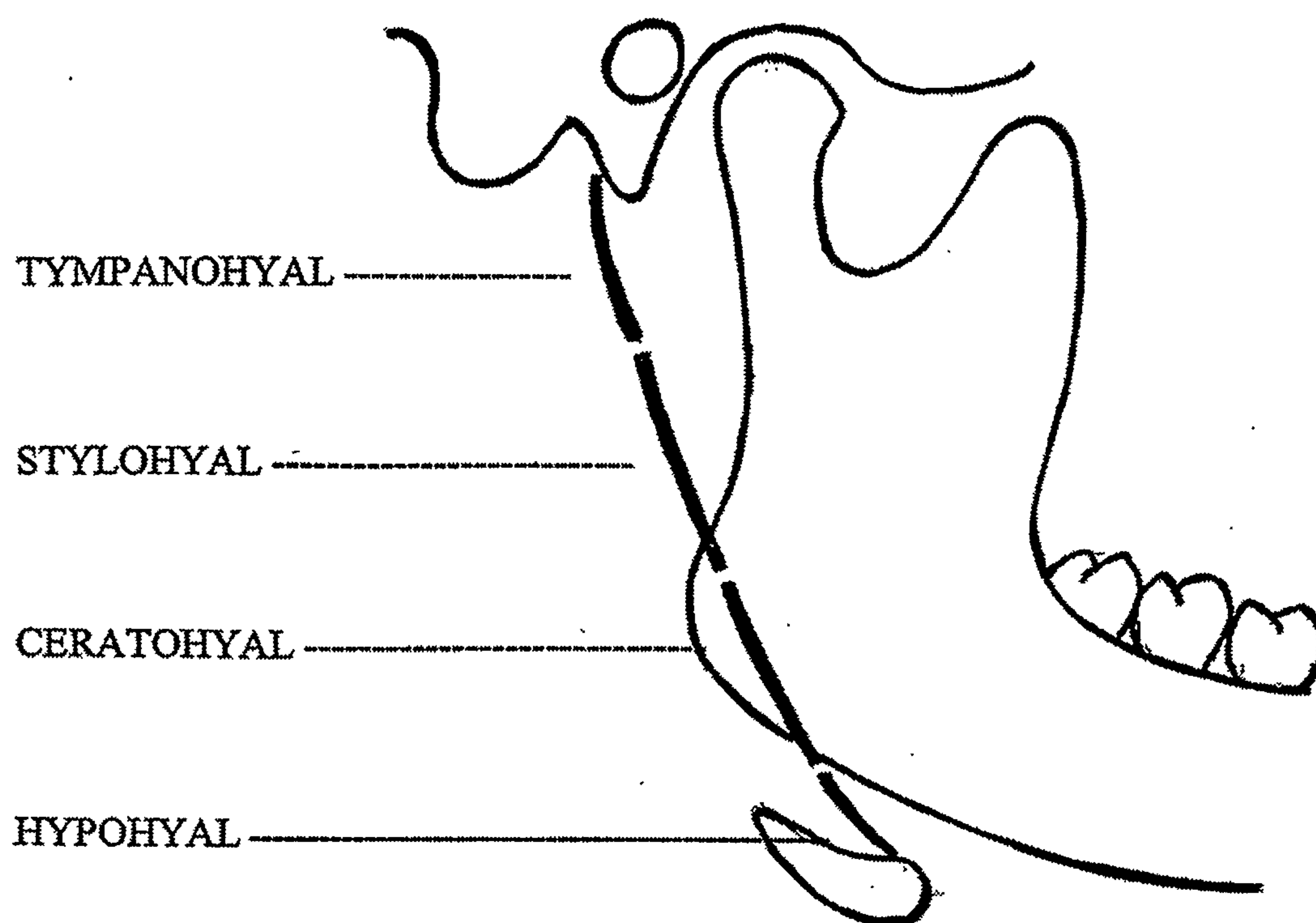


Fig. 4 Embryological derivation of the styloid process apparatus.

The styloid process develops from the first two segments, known respectively from above as the tympanohyal process and stylohyal process (O'Carroll, 1984).

(see Fig. 4)

The stylohyal process appears after birth and calcifies slowly during the first years of life. The styloid process mainly originates in this center. The process shows several points of ossification and at 5-8 years of age this ossification process is normally quite advanced (Boedts 1978; Marcucci, 1959).

Both tympanohyal and stylohyal processes usually fuse at puberty, although sometimes fusion can be delayed or may not occur at all. In most cases they undergo ossification to produce a long styloid process. When only the tympanohyal process ossifies, a short styloid process exists (Lengele and Dhem 1980). The ceratohyal (Fig. 4) changes in the interuterine stage into the stylohyoid ligament (Marcucci 1959) and the hypohyal (Fig. 4) gives rise to the lesser cornu of the hyoid bone (Dwight 1907). Interestingly, Reichert's cartilage of the second branchial arch, the progenitor of the styloid and hyoid processes, also gives rise to the stapes located in the middle ear (Ernest, 1986) indicating a possible embryological link between symptoms originating in the styloid process area and the ear.

#### 4. NEUROPHYSIOLOGICAL BASIS OF OROFACIAL PAIN:

Pain is part of normal physiology providing an individual with a warning system against tissue injury and disease. Somatic pain is due to the activation of nociceptor afferent nerve fibres whose activity is transmitted to the central nervous system and may result in reflex responses and the sensation of pain.

##### 4.1 Peripheral Mechanisms:

Sensory nerve fibres may be classified as A- and C-fibres.

Thin unmyelinated nerve fibres are termed C-fibres. They vary in diameter between 0.2 and 1  $\mu\text{m}$ , and have conduction velocities between 0.25 and 2.0 m/s. The majority of C-fibres may be involved in pain and nociception, although activity in other C-fibres results in non-painful sensations (i.e. warm, itch). C-fibre mediated pain has a diffuse and dull quality. Myelinated nerve fibres range in diameter from 2-20  $\mu\text{m}$  and conduct at 12-120 m/s. Among these myelinated fibres are a group ranging up to 6  $\mu\text{m}$  called A-delta fibres, and are involved in nociception and pain (and cold) sensations in the first instance. Of all the sensory nerves there seem to be more C-fibres than A-delta fibres.

Primarily, mechanoreceptive stimuli and also low intensity electrical stimuli, activate larger myelinated fibres termed A-beta fibres that evoke non-painful sensations (touch, pressure, vibration) from muscles, skin and joints. A-beta stimuli may provoke pain in inflammatory pain states.

Two types of receptors (nociceptors) for noxious stimuli include high-threshold mechanoreceptors (HTM's) that signal via A-delta axons, and polymodal nociceptor units signalling via C axons. The HTM's respond to moderate to severe pressure and are slowly adapting. Their receptive fields consist of multiple points over a large area with overlap between fibres. Some of these receptors transmit via A-beta fibres.

Polymodal nociceptors respond to heat and chemical injury as well as firm pressure and are slowly adapting. Their receptive fields are single small zones. With repeated stimulation, polymodal nociceptors reduce their threshold and increase their activity, as part of the phenomenon of peripheral sensitisation.

Abrahams et al (1982) noted the presence of a number of mechanoreceptor units responding to moderate pressure, stretch and contraction and signalling via A-delta fibres at muscle-tendon junctions.

The type of information signalled by a particular sensory fibre depends on its threshold and speed of adaptation. A peripheral stimulus activates many afferent fibres and the resultant sensation perceived by the intact organism depends on the interaction between the sensory input at various relay levels of the input pathway to higher cognitive levels of the brain. Inhibitory and excitatory interactions occur between the sensory inputs at each relay level. The sensation of pain then becomes a decoding of many afferent inputs at the highest level of the ascending nociceptive system.

The central terminations of most afferent fibres extend over a wider area than the group of cells they normally excite (Wall and Werman 1976). As far as A-delta fibres in the dorsal horn, these normally "silent" connections are inhibited probably from peptide containing cells in the substantia gelatinosa of the dorsal horn. C-fibre afferent impulses may control this inhibition. Injury, inflammation and C-fibre activation (i.e. chemical activation) could result in the reduction of such inhibition and an expansion of the receptive fields of the relevant myelinated fibres. C-fibre input is perhaps the requirement for the initiation and maintenance of secondary hyperalgesia.

#### 4.2 Central Mechanisms:

The spinal cord is divided into cervical, thoracic, lumbar and sacral parts. Emerging from the cord, the ventral and dorsal roots join together distally to the dorsal root ganglion to form a mixed spinal nerve consisting of afferent as well as efferent nerve fibres (see Fig. 5).

The spinal cord is composed of white matter (mainly nerve bundles) and grey matter (mainly cell bodies). The white matter is divided into three bilateral columns or funiculi. Of these, the dorsal column forms one of the major ascending pathways for sensory information. The lateral columns contain both ascending sensory pathways and descending control systems. A laminar organisation of the spinal grey matter was proposed by Rexed in 1952.

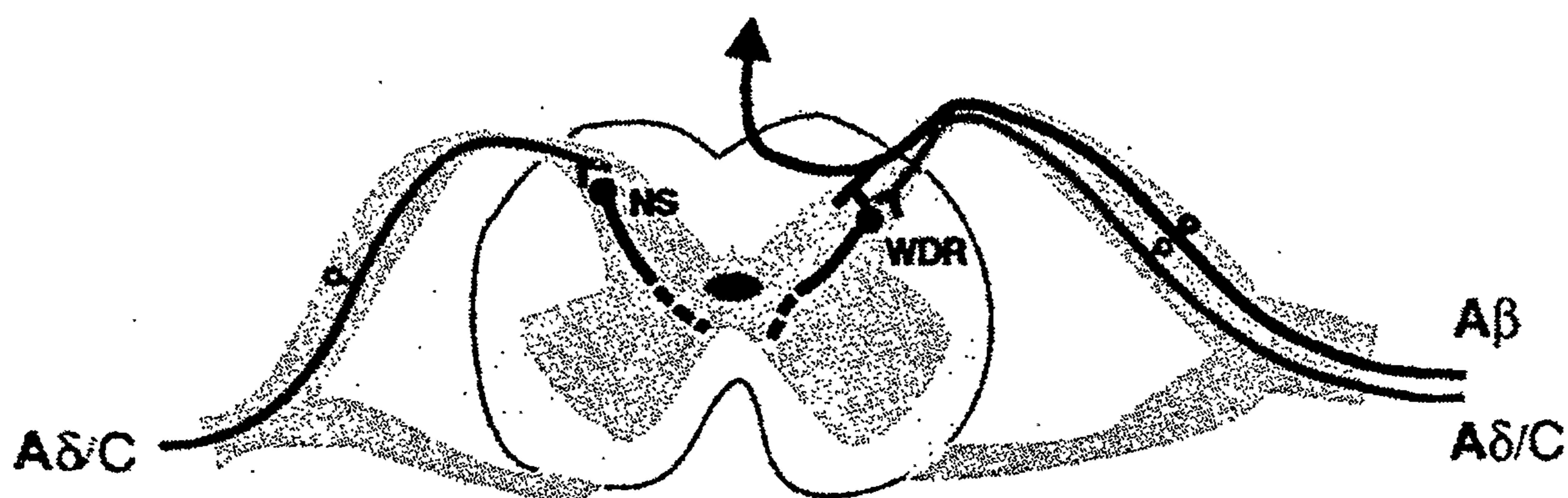


Fig. 5 Anatomical features of the spinal cord and the peripheral nerves. Adapted from Ekblom and Rydh-Rinder (in Rawal) (1998)

Sensory nerves supplying orofacial tissues carry nociceptive information through the trigeminal ganglion. They then enter the brain stem and ascend or descend in the trigeminal tract before entering the trigeminal sensory nuclear complex. The latter is subdivided into the main sensory nucleus and the spinal tract nucleus. The latter nucleus consists of three subnuclei, oralis, interpolaris and caudalis. Many of the neurones in the trigeminal sensory nuclear complex or adjacent to the complex have connections with the cranial nerve motor nuclei and may serve as reflex interneurons in the reflex responses to orofacial pain stimuli.

Nociceptive afferent fibres synapse onto second order neurons in the dorsal horn of the spinal cord or the trigeminal sensory nuclear complex in the medulla and pons. C polymodal nociceptive afferents synapse exclusively in lamina I, II (substantia gelatinosa) and V of the dorsal horn. A-delta nociceptors also terminate in the lamina I and II but penetrate deeper to end in lamina V and X. Nucleus caudalis of the trigeminal sensory nuclear complex is often referred to as the medullary dorsal horn (Sessle 1987) and acts as the principle brain-stem relay site of orofacial nociceptive information. Subnucleus caudalis and the spinal cord dorsal horn have very similar functional and morphological features. The more rostral parts of the trigeminal sensory nuclear complex, especially the main sensory nucleus and subnucleus oralis of the spinal tract nucleus, are seen to be major relay sites of tactile information. (Sessle 1987)

After the first synapse in the dorsal horn or subnucleus caudalis, the second-order neurone transmits activity to supraspinal areas via specific pathways. Most fibres conveying nociceptive information project via pathways in the ventrolateral white matter (spinothalamic tract) of the spinal cord, or the trigeminothalamic tract in the case of the trigeminal system. Most second-order neurones then cross to the contralateral side and ascend to the thalamus. The spinothalamic tract is one of the most important pathways in nociceptive transmission. It projects directly to the ventroposterior lateral nucleus in the thalamus. Third-order neurons then project to different areas in the somatosensory cerebral cortex and to the limbic forebrain (Chapman and Turner 1986) (see Fig. 6).

Second-order somatosensory neurons have been classified into three main groups: nociceptive specific (NS), wide dynamic range (WDR) and low threshold mechanoreceptive (LTM) neurones. NS neurons respond exclusively to noxious stimuli and receive A-delta and/or C fibre input. They possess only a small receptive field. WDR neurons are excited by both noxious and non-noxious stimuli (i.e. mechanical, thermal and noxious or chemical) and are thought to receive input from both large and small diameter myelinated A fibres as well as unmyelinated C fibres. They exist in high concentrations in lamina V and have large receptive fields. Under certain conditions, NS neurons appear to be able to reversibly change their properties so that they resemble WDR neurones. LTM neurons respond to light touch and this input is conveyed by large myelinated A fibres. The final output of second-order neurones is influenced by the convergence of many afferent inputs.

Extensive convergence from afferents in skin, mucosa, viscera, the temporomandibular joints, the jaw, face, tongue and jaw muscles, tooth pulp and cervical afferents, underlies the spread and referral of pain frequently seen in many orofacial pain conditions (Sessle 1987). Subnucleus caudalis neurones that project out of the nucleus to various sites are primarily found in lamina I, III-VI. Lamina II (substantia gelatinosa) receives low threshold peripheral afferents, nociceptor afferents and noradrenaline and serotonin containing terminals from higher centres. The descending somatosensory modulation of nociceptive transmission is especially apparent at the level of second-order neurones in substantia gelatinosa thereby forming the neural basis of the gate control theory proposed by Melzack and Wall (1965). Large fibre transmitted counter-irritants such as TENS, ultrasound and other modalities produce inhibitory effects on the nociceptive system. The transmitters involved in the descending control system at the spinal cord level are primarily serotonin and noradrenaline. Subnucleus caudalis neurones then project to the posterior thalamus, cerebellum and the periaqueductal gray, pons, spinal cord and the rostral region of the trigeminal brainstem complex.

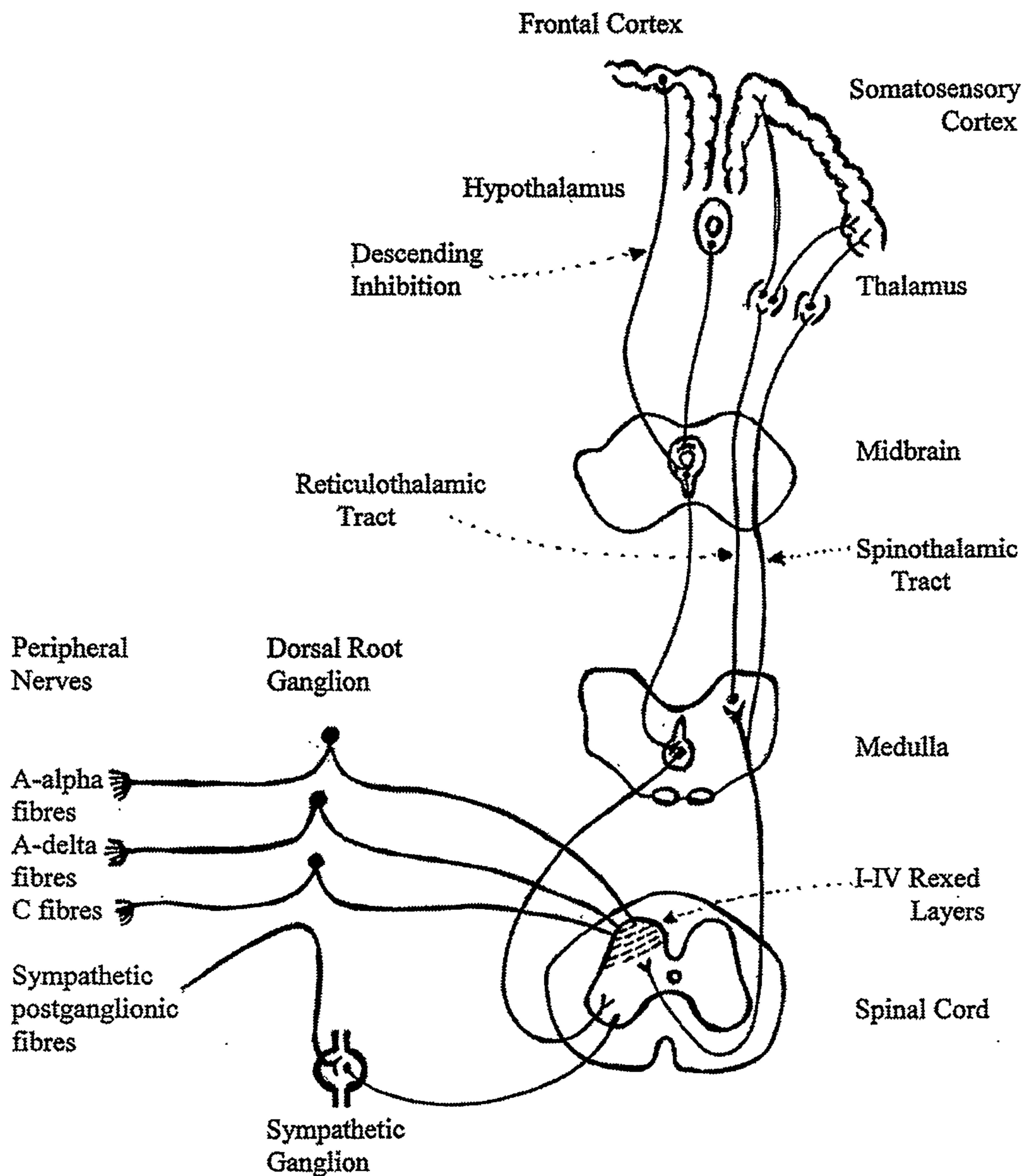


Fig. 6 Transmission of nociceptive signals from the periphery via the spinothalamic tract to the cortex. Pain modulation is depicted as a descending influence from the cortex and the hypothalamus.

The synaptic endings of primary afferents in the dorsal horn contain various types of vesicles and excitatory neurotransmitters. There are three classes of neurotransmitter, peptides, amino acids and the nitric oxide gas molecule. Following their release, these neurotransmitters take part in the inflammatory process (Fig. 7).

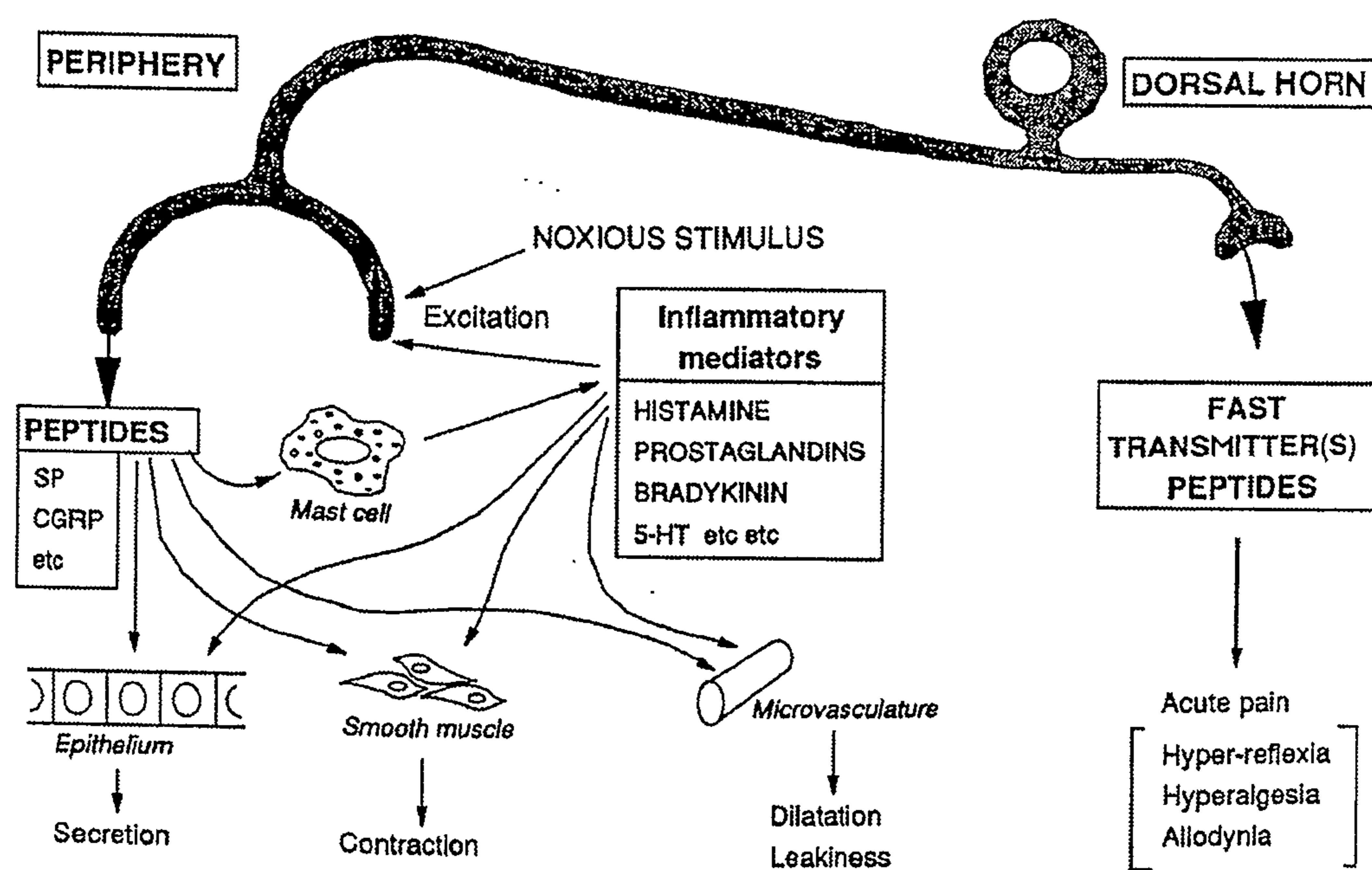


Fig.7 Afferent and efferent aspects of nociception. An afferent fibre is showing activation by injury. Following injury, the afferent fibre itself releases peptides which stimulate various inflammatory cells to release pro-inflammatory substances.

(diagram courtesy of Dr. Samuel Quek, lecturer at the TMJ/Orofacial Pain Center UMD-New Jersey Dental School)

There are many types of neurotransmitters in the central terminals of primary afferents. The most important of these are the excitatory amino acids such as glutamate, aspartate, substance P (SP), somatostatin, cholecystinin (CCK), calcitonin gene related peptide (CGRP) and dynorphin, which are found in the dorsal root ganglion (DRG).

Primary afferents contain more than one neurotransmitter in their terminals. It has been shown that some pre-synaptic terminals of second order neurones may contain multiple neurotransmitters also (Raj 1997).

SP is present in 10-20% of spinal sensory neurones. It has been found within neuronal terminals of sensory neurones (for example A-delta and C fibres) located in lamina I and II of the dorsal horn. SP has an excitatory effect on dorsal horn neurones that is gradual in onset and prolonged. SP facilitates the response of cells activated by noxious cutaneous stimuli. Other neurotransmitters such as glutamate and CCK appear to be less selective and produce excitatory and facilitatory effects on neurones that respond to a wide variety of stimuli. It has been shown that several neurones in the peripheral and autonomic as well as the central nervous system contain and secrete more than one biologically active substance. It is possible that both substance P and a fast acting neurotransmitter may be secreted by the same neurones in the dorsal horn.

Other neurotransmitters in the dorsal horn neurones are involved in pain processing. Opioid peptides and receptors in the dorsal horn mediate nociceptive impulse transmission directly. Substance P release from sensory and dorsal horn neurones in response to noxious stimuli is blocked by exogenous and endogenous opioids. Neurotransmitters are also involved in descending control of spinal pain transmission. Neurones in the periaqueductal gray matter and nucleus raphe magnus modulate pain transmission at the dorsal horn. These neurones are thought to be opioid sensitive.

Glutamate and aspartate have been found at excitatory synapses throughout the central nervous system (Kahn 1991). Serotonin is the major neurotransmitter associated with terminal projections of nucleus raphe magnus. Direct intrathecal serotonin results in a reduction in response to noxious stimuli. Two major systems in the CNS are responsible for modulation and transmission of pain impulses:

- a) neurones located in the dorsal horn of the spinal cord contain endogenous opioids which block the transmission of painful stimuli from peripheral afferents,
- b) descending modulatory system inhibits transmission at neurones in the dorsal horn. One of the primary neurotransmitters in this system is serotonin.

Nitric oxide may be of importance in neuronal signalling. Nitric oxide has also been discussed as a neuromodulator at the dorsal horn under pathological conditions (Garthwaite 1991). Bradykinin activates afferent C-fibres and is an important mediator of peripheral inflammation. Bradykinin also induces nitric oxide production.

Nociceptive transmission in the spinal cord includes ventral horn motoneurons evoking spinal reflexes as important protective mechanisms (Fig. 8). Taken as a whole, the spinal cord neurons, like the peripheral neurons, exhibit a dynamic plasticity which gives rise to a broad range of responses.

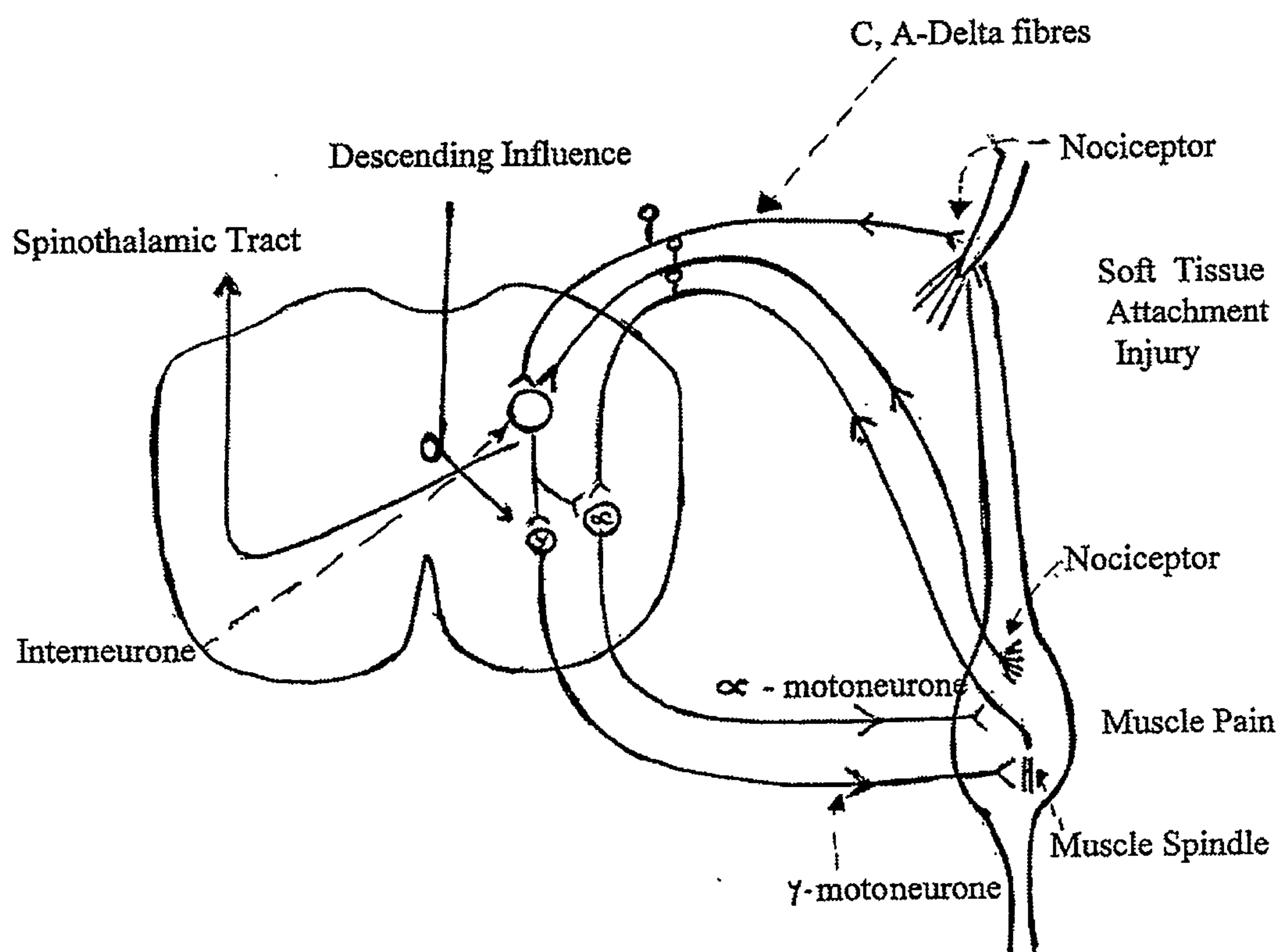


Fig. 8 A proposed myofascial reflex as an explanation of muscle splinting and spasm in patients with attachment injuries.

### 4.3 Chronic Pain

Tissue damage results in the increased sensitivity of nociceptors at the site of injury. This is termed peripheral sensitisation. The nociceptors exhibit spontaneous activity, lowered thresholds and increased responsiveness to suprathreshold stimuli. The increased nociceptor activity produces an increased neuronal barrage to the central nervous system (CNS) resulting in central sensitisation that contributes to secondary hyperalgesia and spontaneous pain.

Nerve damage at the site of trauma can result in neuroma formation or axonal sprouting. Neuromas emit spontaneous activity and are sensitive to mechanical, thermal and chemical stimuli (Wall and Gutnick 1974). Spontaneous neural activity also originates from the cell bodies of damaged nerves located in the dorsal root ganglia (Wall and Devor 1983). This increased activity results in hyperexcitability of the CNS that is believed to contribute to secondary hyperalgesia and spontaneous pain.

Therefore, both tissue and nerve injury at the site of trauma can result in prolonged changes in the nervous system and peripheral and central sensitisation.

It is generally accepted that nociceptive pain produced by tissue injury is significantly influenced by peripheral inflammatory changes (Coderre et al 1993). Increased peripheral barrage produced by inflammation results in an enlargement of receptive fields, increased excitability in response to thermal and mechanical stimuli (Dubner 1997). This increase in neuronal activity may be perceived as hyperalgesia. The hyperexcitability of dorsal horn neurones appears to involve excitation at receptor sites activated by excitatory amino acids such as, glutamate and aspartate, that act at NMDA receptor sites (Dubner and Basbaum 1994).

The release of excitatory amino acids and their effects on dorsal horn neurones appears to be enhanced by neuropeptides, substance P, CGRP and dynorphin. NMDA receptor antagonists significantly attenuate the hyperalgesia induced by inflammation.

It appears that persistent pain is not only initiated but maintained by peripheral nerve activity associated with the injury (Gracely et al 1992).

Melzak (1979) proposed that a healthy balance between noxious and non-noxious stimuli in a healthy state sets the mechanisms controlling afferent input to both the spinal cord, brain stem and higher levels. Persistent pain may disturb this balance and may "set" the nervous system in a new pattern. Perhaps reactivating a healthy balance of sensory inputs is all that is needed therapeutically (i.e. treating the source of the pain).

The nervous system cannot be regarded as a rigidly wired electrical circuit.

Neuroplastic changes occur in central neurones that result in changes to receptive fields as well as the threshold and duration of neuronal responses following chronic nociceptive stimulation. Activation of C-fibres is important to initiate these plastic changes. The established plastic changes may persist for long periods and may be re-elicited by local triggering factors. Experiments have shown that peripherally applied anaesthetic can reverse the plastic changes recruited by a pain experience, returning the neurones to their previous "neutral" state (Reynolds and Hutchings 1948), or "healthy balance" (Melzak 1979).

### **Nociceptive Pain and Central Plasticity**

Primary hyperalgesia is classed as increased sensitivity to noxious stimuli at the site of injury. This effect is mediated by peripheral mechanisms (neurogenic inflammation). Secondary hyperalgesia is an increase in sensitivity extending beyond the site of injury and is related to central hyperactivity or sensitisation. This secondary hyperalgesia is also characterised by allodynic-type pain, where pain is experienced following light touch that would not normally be associated with pain.

Such referred pain spreads to areas which generally do not share the same dermatomes (Lewis 1942). It is clinically well known that pain from deep somatic structures may be referred to distant structures. Furthermore, referred pain has been found to spread specifically to sites of previous injury (Henry and Montuschi 1978). Of interest here is that in some studies (Noordenbos and Wall 1981), a peripheral trigger provided the input required to activate central neural structures subserving the memory of a past injury thereby resulting in pain. Activity in C-fibres, post-injury, induces an enlargement of the receptive fields of second-order nociceptive specific neurons, and also permits the dorsal horn neurons to start to respond to non-noxious stimuli.

Cutaneous (Woolf 1983) and deep (Woolf and McMahon 1985) tissue injury, as well as noxious electrical stimuli of cutaneous and muscle afferent nerves (Wall and Woolf 1984) also produce an increase in excitability of ipsilateral and contralateral flexor efferent nerves in response to noxious mechanical stimuli. Since the increased excitability in the contralateral flexor efferent nerve is maintained after blocking inputs by local anaesthetic, this suggests central not peripheral changes underlie this effect. As a result, peripheral injury can produce central changes that are maintained after the injury input is removed.

Peripheral nociceptor sensitisation and central neuroplasticity resulting from chronic nociceptor discharge would be a succinct "vicious cycle" explanation (see Fig. 9 from Mense 1990). This figure depicts a chronic, soft tissue lesion (A) responsible for a constant barrage of inflammatory mediators as well as a constant barrage of nociceptive afferent impulses. This initiates a cycle resulting in muscle ischaemia which contributes a greater number of afferent impulses and vasoactive chemicals. As a central response to this soft tissue lesion, muscle splinting is initiated in an attempt to immobilise the injured area, and (B) contributes to the local ischaemia. This leads to a lack of ATP and a failure of the calcium pump with contracture. The resultant compression of blood vessels enhances the ischaemia. After sustained activation of nociceptive afferents, some form of nociceptor sensitisation is expected. This may be the aetiology behind latent and active trigger points, assuming the site of the lesion remains undiagnosed and, therefore, untreated. The latent trigger point becomes the result of a chronic lesion. The active trigger point can then be seen as a heightened response, following normally innocuous stimuli of an already sensitised nociceptor site.



Only one chemical condition, low extra-cellular pH, is known to provide sustained excitatory drive to nociceptors (Steen et al 1992; Issberner et al 1996). Although the inflammatory exudate can considerably enhance the number and activity of nociceptors, tissue pH levels are sufficient to maintain nociceptor discharge (Steen 1995). This condition is regularly found in inflamed tissue and in muscle tissue working under ischaemic conditions. In both cases there is an imbalance between increased blood flow and even more increased cellular metabolism resulting in lactic acid accumulation (Reeh and Steen 1996).

Reeh and Sauer (1996) claim that inflamed tissue remains free of pain as long as it is allowed to rest. As soon as the tissue is forced to work, increases in the temperature and proton concentration may result in pain by sensitisation of the nociceptors induced by inflammatory mediators. Chronicity, therefore, may be a matter of permanent nociceptor sensitisation due to the permanent presence of inflammatory mediators. This supports the vicious cycle theory.

With regard to the styloid process attachment sites, it is hypothesised that they are perhaps predisposed to a chronic pain scenario since they are subjected to constant tensile forces from the normal functions of the attaching structures. For instance, the stylopharyngeus, styloglossus and stylohyoid structures are active during a variety of movements, for example, swallowing, chewing and talking. An injury at this site may also be subjected to considerable parafunctional forces in a bruxing patient. This may occur, for example, during marked excursive movements of the mandible when bruxing, and such movements may place large tensile forces on the stylomandibular ligament attachments. These tensile forces could constitute a continuing source of irritation to the site of injury. If so, then there is the possibility of a persistent presence of inflammatory mediators sufficient to induce and maintain a sensitized nociceptor site. This may also lead to neuroplastic changes at the dorsal horn level.

The potential effects of a cyclic response are illustrated in Fig.10

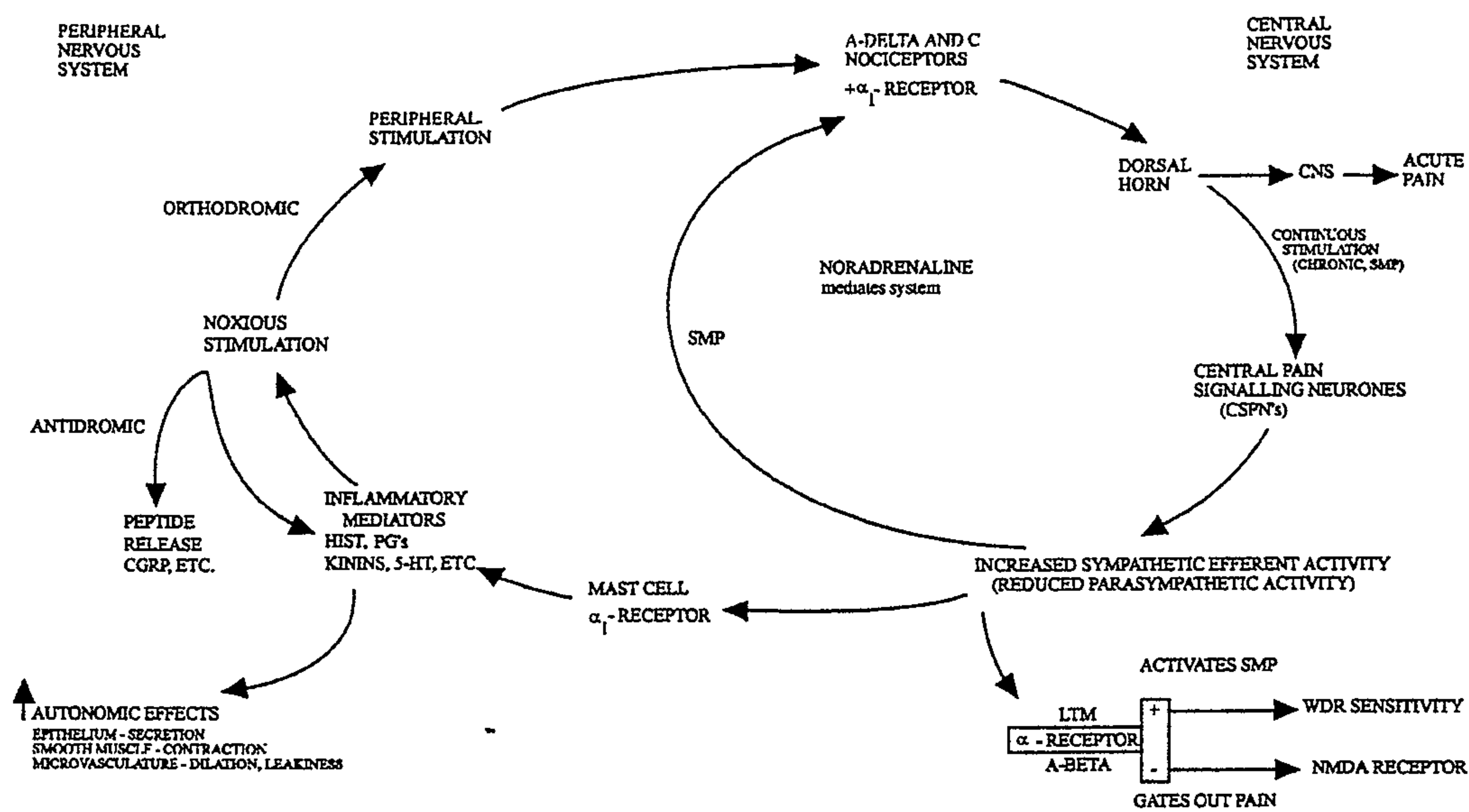


Fig. 10 This diagram illustrates the potential peripheral and central consequences of a peripheral injury. The "noxious stimulation" term can be substituted with "styloid process lesion" and indicates a styloid process soft tissue injury. (Diagram courtesy of Dr. Samuel Quek, lecturer at the TMD/Orofacial Pain Center UMD-New Jersey Dental School)

## Central Sensitisation

Following noxious stimuli, there is sensitisation of neurons in the dorsal horn of the spinal cord and other areas of the somatosensory pathway. This sensitisation is reflected by increased spontaneous activity, reduced thresholds or increased responsiveness to afferent inputs, prolonged after-discharge to repeated stimuli and an expansion of peripheral receptive fields of dorsal horn neurons.

Repeated C-fibre afferent stimulation following injury, sequentially raises dorsal horn activity resulting in prolonged discharge (Mendell 1966). Tissue injury also produces an expansion of the receptive fields of dorsal horn neurons. Receptive field expansion has also been observed in the trigeminal brainstem neurons following chemical stimulation of deep craniofacial afferents (Hu et al., 1992).

## Neurochemical Mediators of Noxious Stimulus-Induced Plasticity

A trauma-induced, soft-tissue attachment injury at the styloid process presumably would exhibit local inflammatory changes such as oedema, hyperaemia and infiltration by polymorphonuclear leukocytes.

Approximately 80% of the peptides produced in the cell bodies of afferents travel towards the periphery. This efferent pool of peptides may be released in response to nociceptor activation. Following their release, these peptides take part in the inflammatory process. They participate in the signs of inflammation such as redness, heat, swelling and pain. Once released, they may stimulate various inflammatory cells to release pro-inflammatory substances thereby establishing a vicious cycle response (Fig. 10). Recognising the potential for nociceptor sensitisation, perhaps more important in this context is the potential transition of acute to chronic pain if the inflammatory sourced peripheral nociception is not diagnosed and treated early and effectively.

It appears that C-fibre neuropeptides are involved in triggering CNS plasticity following injury or noxious stimulation. Various substances produced during inflammation can also directly activate nociceptor afferents, sensitise them and/or result in the release of proinflammatory substances from them. Noxious stimulation or peripheral inflammation causes the release of substance P, neurokinin A, somastatin, calcitonin gene-related peptide (CGRP) and galanin in the dorsal horn. Such peripheral inflammatory conditions also are responsible for the release of histamine, serotonin, bradykinin, prostaglandins and leukotrienes. Some of these substances can increase the sensitivity of nociceptors and nociceptor afferents towards other pain producing substances or stimuli (ie mechanical, thermal and chemical). This is an important feature considering C and A-delta nociceptors are found in particularly high concentrations at attachment sites/entheses (Ball 1970; Harvey 1987), and may be present in high concentrations at the styloid process attachment sites.

Noxious stimulation or peripheral inflammation causes the release of glutamate and aspartate in the dorsal horn implicating these excitatory amino acids in injury-induced neuroplasticity. Noxious stimulation also results in an increase of intracellular calcium in nociceptive neurons. Repetitive C-fibre stimulation produces "wind up" activity at the dorsal horn level. This can be mimicked by the application of glutamate at NMDA receptor sites. Substance P, neurokinin A, dynorphin or CGRP have been found to enhance the release of glutamate and aspartate from the dorsal horn (Kangrga and Randie 1990). Mense (1997) found that during experimental myositis, substance P and glutamate were the most likely substances to increase the excitability of dorsal horn neurones.

It appears that persistent pain is not only initiated but is maintained by residual peripheral nerve activity associated with injury (Gracely et al 1992). Several studies have also shown that central sensitisation is increased and is more prolonged after deep tissue injury of the muscle or viscera (Dubner 1997). Inflammation of deep tissues also induces enhanced excitability of dorsal horn cells. Twenty percent of these dorsal horn cells with deep input were exclusively driven by nociceptors located in deep tissues such as tendons and ligaments (Hoheisel and Mense 1990). These neuroplastic changes of the spinal cord may be a prelude to chronic pain.

## 5. FUNCTIONAL INFLUENCES ON THE STYLOID PROCESS

Although the average normal length of the process is approximately 1 inch or 2.5 cm., this length can be different on either side in the same person. Studies (Camarda 1989) have shown that the styloid process has a rapid growth period until the age of 30 years after which it slows considerably. Some studies have stated styloid process growth then accelerates somewhat at age 50 years. The reasons for these growth observations have not been clarified. To cloud this issue further, Lengele and Dhem (1988) reported no correlation between age and the length of the styloid process. It seems that the only certainty is the variability of the styloid process dimension.

It has been theorized that the source of elongated styloid processes may be continual growth at the cartilagenous base or a calcification of the stylohyoid or stylomandibular ligaments. Camarda (1989) considered the term "calcification" erroneous. They claimed metaplasia of the stylohyoid fibrocartilagenous tissue results in osseous tissue and not calcified ligament. Steinmann's theory (Steinmann 1968) of reactive hyperplasia proposes ossification of the stylohyoid ligament when appropriately stimulated by pharyngeal trauma. His second theory of reactive metaplasia involves trauma resulting in metaplastic changes to the ligaments creating ossified elements within these ligaments.

An hypothesis put forward by the author is that the styloid process may grow in response to continual tensile forces applied to it by its attachments. This response may initially be a local inflammatory response that progresses into a fibrous repair which becomes calcified in time. The amount and direction of growth of the styloid process may be determined by the nature and direction of the musculotendinous forces acting upon it. If the styloid process grows in response to functional musculotendinous tension, any constant mandibular protrusion or clenching may facilitate elongation of the process. In support of a possible association between functional forces and alteration of length and/or form of the styloid process, forward head posture, in some patients, has been shown to result in an altered cervical spine length (Harma 1967; Zohar et al 1986). There have, however, not been any studies into the relationship between elongation of the styloid process and postural abnormalities.

In addition, bone spurs are commonly found at tendinous attachments throughout the skeleton as a response to a local inflammatory condition. Further, it is unknown if some people have a greater potential for ossification of the stylohyoid and stylomandibular ligaments. In any case, there is no reliable evidence that a gradual elongation of the styloid process leads to an increase in pain experience.

## 6. CONCEPTS OF PATHOGENESIS

### 6.1 Elongated Styloid Process

Symptomatic elongation of the styloid process and the concomitant mineralisation and calcification of the stylohyoid structures, have been regularly reported.

Demanchetis as far back as 1652, and subsequently, Lucke (1870), Weinlecher (1872), Sterling (1896), Dwight (1907), and Thigpen (1932) have all been associated with published data on elongated styloid processes.

Since then, Eagle (Eagle 1948, 1949, 1958) tried to differentiate various clinical entities of the elongated styloid process syndrome or as it has been popularly called, Eagle's syndrome.

1. The Classic syndrome that follows a tonsillectomy procedure is characterised by symptoms of dysphagia, pain referred to the ear, dysphonia and a sensation of a foreign body in the pharynx. It is assumed that the healing tonsillectomy scar tissue tightens the mucosa across the tip of the elongated styloid process. Upon normal function such as yawning, eating and swallowing, the movement of this mucosa across the styloid process is thought to lead to the symptoms cited above (Sivers and Johnson 1985; Eagle 1958).

2. The Carotid Artery syndrome results in chronic neck pain, pain upon turning the head and pain radiating to the eye when lateral development of the styloid process impinges upon the sympathetic nerve supply associated with the walls of the external carotid artery. If medial development of the styloid process impinges upon the sympathetic nerve supply associated with the internal carotid artery, pain referral from the occiput to the ophthalmic region results. The primary patient complaint would be headache. This so called Carotid Artery syndrome has also been termed Carotidynia.

The glossopharyngeal nerve, the mandibular division of the trigeminal nerve and the chorda tympani nerve could all be, hypothetically, traumatized by an elongated or malpositioned styloid process (Harma 1967). Fractures of the styloid process that are not allowed to heal due to the constant movement in the area may result in the formation of granulation tissue (Balasubramanian 1964) within an enclosed fascial space that could correspond to a degree of neurovascular compression relative to the size of the mass. Such neurovascular irritation from elongations or as a result of elongations of the styloid processes are examples of other authors hypotheses of a cause of styloid-process initiated pain.

The above-mentioned symptoms of lateral head pain, ear pain, pain when eating or pain around the eye can also be symptoms of patients presenting with a suspected temporomandibular joint dysfunction or myofascial pain complaint.

Elongation of the styloid process has been found in 4% (Eagle 1948, 1949, 1958) to 7.3% (Kaufman et al 1970) of the population. If mineralisation along the stylohyoid chain was also considered, prevalence was raised to 28% (Kaufman et al 1970) or 33% (O'Carroll 1984) depending on the study.

In a large study, Correll et al (1979) found the incidence of elongated styloid processes at only 18.2% of the patients examined. Of these, only 8 patients, out of a reported 1,771, outlined symptoms related to Eagle's syndrome. Eighty three percent of the patients exhibiting elongated styloid processes had bilateral elongations.

However the patients exhibiting symptoms had only unilateral symptoms. If impingement was the sole cause of symptoms, then bilateral symptoms could be expected to be more common. This may suggest another mechanism other than a simple impingement. In another study, Harma (1967) attributed elongation of the styloid process as the cause of symptoms in only 50% of his symptomatic group.

In summary, elongated styloid processes are regarded as a relatively unusual occurrence. The proportion of people with elongated styloid processes that suffer impingement symptoms are even lower.

## 6.2 Styloid Process of Normal Length

There have been reports in the literature of patients with symptoms apparently arising from styloid processes of normal length. These symptoms have arisen following stretch or whiplash trauma, where an acute force is applied that exceeds the physiological limit of the osseous attachment sites. Such cases have been reported by Steinman (1968, 1970) and Wong et al (1995) and indirectly by Ernest (1986), Shankland (1987) and Harma (1967). These types of injury are the result of hyper-extension and flexion of not only the craniocervical structures but also the mandible, and can occur in patients with styloid processes that are not elongated.

Krespi et al (1981), Steinman (1968,1970), Ernest (1986), Shankland (1987) and Wong et al (1995) have all reported cases of pain and dysfunction arising from the region of the styloid process where there is normal bony architecture. However, there is anecdotal evidence that these patients exhibited long lasting relief of symptoms following an injection of local anaesthetic into the region of the styloid process.

While these studies were anecdotal, that is, there was no objective quantification of pain reduction and the technique of injection was not shown to result in accurate localisation to the styloid process, the observations suggest a pathogenic mechanism other than a simple mechanical hard tissue impingement (because of a styloid process elongation) as a cause of the patient's pain complaint. Rather, the data suggest a soft tissue injury at the tendinous attachment sites of the muscles and ligaments to the styloid process.

While elongations of the styloid processes have been easily quantified using standard radiological techniques such as an orthopantomogram (OPG), the use of a standard radiological survey to diagnose soft tissue injury would prove fruitless. There is a reliance therefore on palpation and local anaesthetic injections in the diagnostic sequence of a soft tissue lesion. The investigation of soft tissue trigger points relies on a similar approach of palpation and local anaesthetic injection. Currently, these procedures are rarely undertaken in the area of the styloid process. The potential, therefore, for a soft tissue injury at the styloid process to remain undiagnosed is quite high unless it is included in the soft tissue examination procedure for patients complaining of head and neck pain..

Treatment directed at the styloid process and its associated structures has been reported anecdotally to result in an alleviation of symptoms (Steinman 1968,1970; Krespi et al 1981; Wong et al 1995). If these improvements can be substantiated in a more controlled study, then it would illustrate a need for closer assessment of the area when diagnosing an orofacial pain complaint. It may also reveal an alternate approach to treatment for certain kinds of head and neck pain

The common denominator in most diagnostic and treatment approaches so far attempted for complaints relating to soft tissue injury involving the styloid process, is the injection of local anesthetic and corticosteroid into the area of the styloid structures (Dolan et al 1984; Evans and Clairmont 1976; Krespi et al 1981; Shankland 1987; Steinman 1968). If this technique were to be used as a therapeutic treatment in addition to it being a diagnostic aid, the benefits of sonography/ultrasound (Brophy et al 1995; Ptasznik and Hennessey 1995), scintigraphy (Dasgupta and Bowles 1995) or MRI (Zannetti and Hodler 1995; Klug 1995; Trepman et al 1995) to direct the injection to the precise site of inflammation may also be of great use. Other techniques of treating suspected styloid-initiated pain include, surgical shortening of elongated styloid processes (although they have been known to regenerate beyond their previous length (Steinman, 1970)), surgically fracturing elongated processes, and radiofrequency thermoneurolysis (Ernest 1986; Sataloff and Price 1984) of the stylomandibular ligament insertion site.

## 7. AIMS and OBJECTIVES

There appears to have been no follow-up studies that have used standardised, conservative treatment procedures and evaluation for patients who have symptoms related to pain in the vicinity of the styloid process of the temporal bone.

The aims of this study were:

- a) to describe a technique for injection of local anaesthetic and corticosteroid into the region of the styloid process and to validate the injection technique by dissection studies,
- b) to determine whether a local anaesthetic and corticosteroid injection into the region of the styloid process has an effect on subjective reports of pain intensity and on mandibular range of movement,
- c) to document qualitatively whether there are changes in other accompanying symptoms (such as headache).

## 8. MATERIALS and METHODS

### 8.1 Dissection Study

Three fresh, half-head cadaver specimens were obtained for dissection. The purpose of the dissections was to determine the position and type of structures surrounding the styloid process and to determine the final needle position in relation to the styloid process and following the technique for injecting the area described in this study. The cadaver subjects were all male, aged in their sixties without elongated styloid processes. No history of trauma was available.

Using the described injection technique (see Medication Injection Protocol below), the specimens were turned over and a careful medial dissection was performed in the area of the tonsillar fossa to locate the final needle position. Once this position was determined, a lateral dissection was commenced to determine the anatomy surrounding the styloid process in each specimen. Most importantly, the anatomical structures superficial, anterior and posterior to the needle pathway were noted on the way to the styloid process.

## 8.2 Patient/Clinical Study

Ethics approval for the study was obtained from the Western Sydney Area Health Service Ethics Committee.

Due to the preliminary nature of this study, there was no provision for a control group.

Fourteen patients were selected from a group of patients referred to the orofacial pain clinic at the Westmead Hospital dental school and to Dr. Palesy's practice for assessment and treatment for orofacial pain and temporomandibular joint dysfunction. At first consultation, a comprehensive intra- and extra-oral examination was performed.

Criteria for inclusion in the study were:

- (i) patients exhibited symptoms similar to those described in the literature as linked with an elongated styloid processes. Patient's symptoms are listed in **Table 2**.
- (ii) Palpation\* of the region lateral to the styloid process, below the ear lobe and behind the angle of the mandible revealed a swollen, painful area which when palpated resulted in pain referral to the lateral head and neck areas.
- (iii) none of the patients had a palpable process in the tonsillar fossae and their OPG X-rays revealed that the length of their styloid processes were within the normal range (i.e. ~2.5 cm in length).
- (vi) previous conventional treatment for each patient had been unsuccessful with minimal or no effect on their symptoms.

\* palpation of this area is carried out as if it were a trigger point, being mindful of the amount of pain capable of being elicited from this delicate area if too much pressure is applied. In asymptomatic individuals, palpation of this area does not result in pain.

	MA	MD	SG	MBL	WM	MM	RS	AT	JB	MF	GW	HF	TH	JH
<b>PATIENT</b>														
<b>SEX</b>	F	F	F	F	F	F	M	F	F	F	F	F	F	F
<b>AGE</b>	18	50	14	37	61	25	34	16	38	60	31	24	25	34
<b>STYLOID TENDERNESS</b>	*	*	*	*	*	*	*	*	*	*	*	*	*	*
<b>TMJ PAIN</b>	*	*	*	*		*			*			*	*	*
<b>EAR PAIN</b>	*							*						*
<b>EYE PAIN</b>		*			*									
<b>TENSION-TYPE H/A</b>		*	*	*	*	*	*	*	*	*	*	*	*	*
<b>FACIAL PAIN</b>	*	*	*	*	*	*				*	*	*	*	*
<b>CERVICAL PAIN</b>		*		*	*			*		*	*	*	*	*
<b>MANDIBULAR MOTION RESTRICTION</b>			*			*	*		*	*			*	*

Table 2. This table summarises the presenting symptoms of the patients involved in this study.

All patients, except WM, JH, HF, had had previous splint therapy. These patients who did not have a previous splint were deemed to be “non-bruxing” patients. All patients had undergone physiotherapy and anti-inflammatory medication had been prescribed. Three patients (SG, MA, AT) were acute trauma patients who had suffered a sudden onset of symptoms. The remaining eleven patients had symptoms lasting from one year to ten years and a direct, acute trauma-induced onset of symptoms could not be established for these eleven patients, although four patients gave a history of trauma. Informed consent was obtained from all patients.

### 8.3 Pain Assessment and Mobility Measurements

Pain intensity was assessed by measuring each patient's pain recordings at consultation on a 10 cm visual analogue scale (VAS) (0 = no pain and 10 = the most severe pain imaginable). VAS assessments were made just prior to injection and at ten- and twenty-minute intervals post-injection, and at two-week and six-month follow-up appointments.

The patient's mandibular range of opening was measured for maximum opening just before, and at ten- and twenty-minute intervals after the injection. Patients were asked to open their jaw as wide as possible without assistance. A metal ruler was used to measure the vertical distance between the upper and lower left central incisal edges. The jaw opening path was monitored qualitatively to detect deviations of movement.

After at least six months, a questionnaire was sent to all patients. Included in this questionnaire were questions on the characteristics of their pain situation, any treatment received between the injection and the re-evaluation, a measurement of incisal distance on maximal opening and a current VAS score. The measurement of maximum opening was obtained using the same methods as in the initial and following evaluations. The patients were instructed to use the same methods as used in the operatory.

#### 8.4 Medication

Each patient was seated comfortably in a dental chair and a single injection was given on the symptomatic side. It was noted that this injection is as painful as a trigger point injection with the patient "wincing" on insertion of the needle tip. The injected solution consisted of 4% Prilocaine hydrochloride (Astra Pharmaceuticals, North Ryde, N.S.W. Australia) together with 2 mg of Celestone Chronodose (Schering-Plough Pharmaceuticals, Baulkham Hills, N.S.W. Australia). An injection of 0.5 ml was made in the area of the stylomandibular ligament insertion at the angle of the mandible and a further 1.5 ml of the solution was deposited in the area of the styloid process.

#### 8.5 Medication Injection Protocol

The injection technique used was based on a series of three cadaver dissection studies (Fig. 11) carried out to determine the safest pathway for injecting reliably in the area of the styloid process (Grunweg and Palesy, 1992, Dept.Oral Medicine, UMDNJ, unpublished). The injection protocol was as follows:

The injection point through the skin was below the ear lobe and 1-2 mm distal to the posterior border of the ascending ramus at a level corresponding to the postero-superior border of the masseter muscle insertion (palpable when the patient clenches their teeth). This area was disinfected using 10% Betadine swabs (The Perdue Frederick Comp., Norwalk USA) by rubbing, in a centrifugal motion. This was followed with 70% Isopropyl Alcohol swabs (Briemar Nominees P/L, Australia) used in a similar fashion. A cooling spray (VOCO, Cuxhaven Germany) was applied to the area until frosting appeared, providing transient surface analgesia. The patient was instructed to lightly close with the teeth in light contact. The needle (25 gauge diameter and 38 mm in length, Terumo, Japan) was directed posteriorly at an angle of approximately 10 degrees posterior to the perpendicular at the skin surface and parallel to the Frankfort horizontal plane. The bevel of the needle was positioned to face posteriorly so that the major vascular structures are positioned distal to the bevel of the needle. Once the needle tip had penetrated 12 mm, 0.5 ml of local anaesthetic (4% Prilocaine, Astra, North Ryde, Australia) and 2.5 mg Celestone Chronodose (Schering Plough, Baulkham Hills Australia) solution were deposited. Based on our dissection studies, the injection site was assumed to be in the area of the insertion of the stylomandibular ligament. The needle was then inserted a further 6 mm to a total of 18 mm and the body of the syringe angled anteriorly, parallel to the Frankfort horizontal, approximately 30 degrees from the previous position and inserted to a total depth of approximately 24 mm when a further 1.5 ml of the solution was deposited.

## 9. RESULTS

### 9.1 Dissection Study

In all three specimens, the needle tip fell within a fascial space containing all the myotendinous attachments from the styloid process. In one specimen the advancing needle struck the tip of the styloid process at approximately 22 mm deep to the skin surface. The other two needle tips ended deep to the styloid process by some 2 mm. The latter two needle positions were inferior to the styloid process by between 2 and 4 mm. Allowances were made for the loss of tissue tone and shape in the specimens before a measurement was deduced for the safe injection of patients. This allowance was estimated at 2 mm, this being a measure that would be added to the measurements taken from the cadaver specimens. This means the depth of injection distances observed in the dissection series were 2 mm less than the external measured depth when the needle tip was position correctly in the clinical study patients. An estimate of 2mm was reached after observing patients of average weight for their age and comparing them visually with the cadaver specimens. Two millimetres quantifies the amount of loss of tissue tone of the fresh cadavers used in the dissection series and that of patients in the study. This is an approximate measurement only, since there is enormous patient to patient variances that would have to be adjudged individually and a clinical adjustment made to the aforementioned guide only measurements offered here.

The injection entry site at the postero-superior border of the masseter muscle insertion coincided with the superior border of the anterior triangle of the neck. At the 12 mm depth, the needle was anterior to the sternocleidomastoid and digastric muscles together with the parotid gland and its associated facial nerve and external carotid artery. The maxillary artery branching from the external carotid artery was usually postero-superior to the injection site and the facial artery branch usually lay inferior to the injection site.

The internal carotid artery and the internal jugular vein together with the glossopharyngeal and vagus nerves were situated deeper to the final needle position. The internal and external laryngeal nerves lay deeper and more inferiorly to the needle end position.

The muscle and ligamentous attachments to the styloid process are situated within the fascial compartment containing the final needle position. Although there are a number of important neurovascular structures surrounding the styloid process, the final needle tip position lies within a space containing primarily only the musculoskeletal structures from the styloid process. This conclusion is the result of an anatomical literature review and three cadaver dissections. Individual variations must, therefore, be taken into account based on soft tissue body mass and after an analysis of the OPG X-ray for skeletal variances.



Fig. 11 Dissection of the area superficial to the styloid process.

Black pin - Styloid Process,

Purple pin - Stylomandibular ligament,

Red pin - Stylohyoid ligament,

Yellow pin - Internal maxillary artery as it branches from the external carotid artery,

Green pin - Temporal artery as it branches from the external carotid artery,

Orange pin - Glossopharyngeal nerve.

## 9.2 Clinical Study

### 9.2.1 Effect of injection on pain intensity

#### VAS PAIN SCORES

PATIENT	PRE-INJ	POST-INJ	POST-INJ	POST-INJ	POST-INJ
		10 MIN.	20 MIN.	2 WEEKS	6 MON.
M.A.	7	2	2	2	1
M.D.	9	1	1	2	N.A.
S.G.	7	1	1	1	2
M.B.L.	8	3	4	2	8
W.M.	6	8	6	3	6
M.M.	8	4	4	4	N.A.
R.S.	7	2	1	1	6
A.T.	8	4	4	1	0
J.B.	7	5	4	4	N.A.
M.F.	6	4	2	2	3
G.W.	5	5	5	5	6
H.F.	8	3	4	7	3
T.H.	8	6	6	6	4
J.H.	5	3	1	3	1

Table 3. VAS scores for all patients immediately prior to injection and at four post injection times: 10 min., 20 min., 2 weeks and at six months. N.A. indicates the patients could not be contacted for their final evaluation.

For the 14 patients, Table 3 lists VAS scores immediately before the injection (PRE-INJ), and at 10 min., 20 min., 2 weeks and at least 6 months after the injection. At PRE-INJ, all patients had high VAS scores with 10 patients having scores of seven or more. These high PRE-INJ VAS scores are consistent with the subjective reports made by these patients that they were suffering severe pain. All patients reported that their individual PRE-INJ scores were representative of their general level of pain.

In comparison with PRE-INJ scores the injection of local anaesthetic and corticosteroid resulted in a statistically significant reduction in VAS pain scores, at 20 minutes, 2 weeks, and at 6 months ( $P < 0.005$ ,  $n = 14$ , paired t-Tests). There was, however, no statistically significant difference between any of the post-injection VAS scores ( $P > 0.05$ ,  $n = 14$ , paired t-Tests). These data indicate that the injection of a local anaesthetic and corticosteroid into the region of the styloid process in this group of patients results in a promising reduction of pain intensity as scored by the VAS index over an extended time frame.

One patient (W.M.) experienced an increase in pain level after 10 min. followed by a reduction of pain after 20 min. and two weeks. It is unclear the reason for this early increase in pain intensity. However, it perhaps may be due to an anatomical variation, whereby the injected solution took longer to reach its target site. No other patient experienced any increase in pain intensity at any stage of the experiment.

While a statistical analysis of VAS scores for all patients as a group, revealed significant differences at 20 minutes and at 6 months, one patient (G.W.) did not show any change in VAS scores. Despite the failure to lower the pain score for this patient, an increase in mandibular opening was noted. Three patients (S.G., M.A., A.T.), whose symptoms related to an acute event, improved markedly at 20 minutes and remained at this level for six months. Two patients (M.D., S.G.), who presented with severe unilateral head pain radiating from the temple to the supraorbital area, found their pain had almost completely resolved five minutes after the injection. Further, at two weeks post-injection, both patients were almost free of pain symptoms. No data were available at the final evaluation for M.D., however for patient S.G., VAS pain level scores remained very low.

Two patients (M.B.L., R.S.) showed an improvement at two weeks but reverted to their initial pain level at six months. Final evaluation data at 6 months was not obtained for three patients, who had changed address and could not be contacted.

## 9.2.2 Effect of injection on range of mandibular opening

**RANGE OF MANDIBULAR OPENING**

<b>PATIENT</b>	<b>PRE-INJECTION (MM)</b>	<b>POST-INJECTION (MM)</b>	<b>POST-INJECTION 6 MONTHS (MM)</b>
M.A.	48	50	48
M.D.	40	45	N.A.
S.G.	32	45	45
M.B.L.	55	55	54
W.M.	42	42	45
M.M.	32	42	N.A.
R.S.	50	50	40
A.T.	25	40	*
J.B.	28	38	N.A.
M.F.	22	38	35
G.W.	25	28	33
H.F.	40	43	50
T.H.	14	21	25
J.H.	30	38	43

Table 4. Range of mandibular opening measurements in millimetres, immediately prior to injection, at a 20 min. interval post injection and at the final evaluation post injection. N.A. indicates the patients that could not be contacted for their six month follow up.

\*One patient was undergoing a course of orthodontic therapy that restricted the mandibular opening measurement.

Mandibular opening (Table 4) at 20 minutes ( $41\pm 9$  mm) and at the final evaluation post-injection ( $42\pm 9$  mm) were statistically significantly greater (paired t-Test,  $P<0.02$ ) than at pre-injection ( $35\pm 12$  mm). All except three patients showed an increase in the range of mandibular opening at 20 minutes post-injection. Of the 10 patients where data was available at 6 months, seven patients exhibited an increase, two patients a decrease and one no change in the range of opening. The reason was unclear for the reduction in the range of opening but only one patient showed a marked reduction and scored a similar VAS score at PRE-INJ and six months.

All patients who presented with TMJ pain reported a reduction of pain consistent with their lowered post-injection VAS score. Consistent with these quantitative and qualitative observations, most patients reported that there was a reduction in the feeling of "tightness" in the facial region or a "less restricted, freer jaw movement".

### 9.2.3 Other effects noted

All patients, except M.A., reported headache as one of their chief complaints and that their head pain returned to a level they remembered prior to the onset of their general head and neck pain. These headaches either began after a traumatic episode or, after trauma, an existing headache became more consistent or intense. At evaluation after two weeks, patients reported that their headaches were occasional and not of the chronic variety that had developed following their traumatic event. Although headaches were still a complaint at final evaluation, they were considered less of a problem than the presenting head pain.

Following the injection, four patients (W.M.,J.H.,M.B.L.,M.F.,R.S.) displayed autonomic responses. Three patients had acute clear nasal discharge following the injection (J.H.,M.B.L.,R.S.) and also reported a clearing of their symptoms of nasal stuffiness. They were able to breathe more freely within minutes of the injection. This effect was not long lasting however, as the nasal symptoms returned gradually during a two week period following the injection. Prior to the injection, two patients (W.M.,M.F.) had complained of blurred vision. Fifteen minutes after the injection they observed an improvement in their vision. These patients did not report a return of blurred vision symptoms.

One patient (M.F.) reported a reduction in lateral facial reddening and puffiness that was becoming more frequent in the years following a motor vehicle accident. There has been no reported return of these symptoms following the injection.

Three patients (M.A.,H.F.,G.W.) sustained ipsilateral lower lip paraesthesia following the injection that lasted approximately twenty minutes without any prolonged effects. One patient (M.B.L.) experienced unilateral paraesthesia of the pharynx for approximately fifteen minutes following injection. These effects were attributed directly to the anaesthetic.

## 10. DISCUSSION

This is the first follow-up study of a standardised clinical protocol directed at the styloid process in a group of patients with facial pain symptoms. An injection of local anaesthetic and corticosteroid resulted in a statistically significant reduction in VAS pain scores and an increase in mandibular opening immediately following the injection. Although pain levels increased again in some patients, six month pain scores were significantly lower than the pre-injection scores. The data suggest the existence of inflammation in the region of the styloid process which may be a source of orofacial pain.

Of the less responsive patients, there may have been an error in needle localisation in the patient who failed to respond to the injection. Some patients in this study reported initial success followed by a relapse. Other studies (e.g. Solveborn et al 1995) have reported similar findings for injections in different areas of the body. Longer lasting benefits have been reported to occur with a further injection. Other studies (Dasgupta and Bowles 1995; Brophy et al 1995) have used scintigraphic and ultrasound guidance to improve the accuracy of soft tissue injections with an improved long-term success rate when compared with "unguided" injections. This may be a more efficient means of treatment delivery than the one employed in the current study.

Much of the previous work on pain originating from the area of the styloid process has concentrated on elongated processes as the cause of the symptoms. Pathogenic mechanisms for pain arising from an elongated styloid process usually involve impingement of pharyngeal mucosa as it is drawn against an elongated process during normal function (Donohue 1959), or impingement of the carotid vessels and their associated sympathetic chain (Ernest 1986). Eagle argued that pain from an elongated styloid process was due to "constant mechanoreceptor discharge in the area of the fifth, seventh, ninth and tenth cranial nerve endings" (Eagle 1949), initiated by a mechanical irritation from the styloid process. However, many reports have indicated elongated styloid processes to be asymptomatic. Therefore the significance of an actual elongation of the styloid process in predisposing an individual to pain, is unclear. In the present study, radiographic and palpatory examination of the styloid process area ruled out any elongations of the styloid processes yet there were symptoms similar to those reported by patients who have elongated processes. The patients in this study were selected on the basis they exhibited symptoms reported in the literature as originating from an elongated styloid process, yet they possessed a styloid processes of normal length. It must be stressed, that the author regards the impingement theory as purely hypothetical and although not relevant to his own hypothetical study here, needs to be mentioned for completeness. Additionally, patients reported to have elongated styloid processes impinging on surrounding structures seem to have symptoms consistent with the symptoms the patients in this study presented with.

Every patient in the study presented with a palpably painful swollen area below the ear lobe on the symptomatic side. This could not be explained by a carotid artery anomaly since a characteristic pulsing effect was not detected. Enlarged palpable lymph nodes would not be evident at such a superior level in a healthy group of patients. Further, digastric myospasm would not be palpable in such a position, being superior to the normal position of the digastric muscle. It is hypothesised that a soft-tissue oedematous lesion is induced by trauma at the musculotendinous attachments to the styloid process within the lateral pharyngeal fascial compartment. This lesion could be initiated by acute or chronic trauma and may be perpetuated by function thereby creating a defined palpable and painful area.

## 10.1 Hypothesis for Pain Referred From The Region of the Styloid Process and Possible Theoretical Mechanisms that may have a Role within the Hypothesis Concept.

### 10.1.1 Trauma

Trauma to the head and neck can be broadly classified as acute or chronic trauma. Chronic trauma may be typified in the head and neck area as, poor head posture (Zohar et al 1986) and repetitive strain forms of work. Dental bruxing could be included here as a form of repetitive poor oral or mandibular posture. Acute trauma (such as whiplash injuries) may result in a sudden strain at the bony sites of attachment of myofascial structures. These examples of parafunctional forces may be the cause of an occult soft tissue injury at the musulo-tendinous attachments on the styloid process.

### 10.1.2 Predisposition to Injury

It is hypothesised that a styloid process of normal length may be predisposed to injury. The styloid process is a relatively fragile structure with a number of myofascial forces applied to it. Three muscles (stylopharyngeus, styloglossus and stylohyoid) as well as three ligaments (stylohyoid, stylomandibular and stylopharyngeus) originate from the styloid process, all of which apply different force vectors to this structure. All of the structures attached to the styloid process insert into structures that are mobile. These anchorage-type attachments are expected to stabilise and restrict the movement of their insertion structures. Trauma, such as acute or chronic mandibular displacement, involving the soft tissue attachments at the styloid process may result in tensile forces great enough and prolonged enough to induce a soft tissue injury at these seemingly vulnerable sites (Palesy 1997). In describing his insertion tendinosis theory, Steinman (1968) concluded, *large powerful muscles inserting into relatively (relative to their size) small areas of bone or periosteum created a site that would probably be susceptible to injury*. With six structures attaching via a number of narrow attachments to a slender styloid process, it is proposed there is a potential for an attachment disruption or injury. In other parts of the body this type of injury would be referred to as tendon strain, tendinitis, tendinosis (Steinman 1970) or enthesopathy. Steinman described insertion tendinosis as a *"degenerative and inflammatory condition in the tendon portion of the muscle insertions with a narrow base, directly at their anchorage to bone"* (Steinmann 1968).

Steinman also mentioned epicondylitis humeri (an inflammation of the hard tissue of the attachment site) as a classic example of insertion tendinosis. Steinman goes on to quote Belart (1957) as having reported *apophysitis* in the transverse process of the cervical and lumbar vertebrae. Fahlgren et al (1966) are credited with describing acute tendinitis of the long muscle of the neck at its atlas insertion.

Ernest (1986) published an extensive paper on stylomandibular ligament tendinosis which implicated the insertion of this ligament as the site of inflammation resulting in a cascade of symptoms. Harma (1967) used the term, "stylalgia" when describing an inflammation in the styloid process region.

### 10.1.3 Trauma Resulting in an Inflammatory Response

#### i) Nociceptor Sensitisation

The patients in this study presenting with an acute trauma injury seemed to be afforded longer lasting relief than did the patients suffering from chronic pain complaints. An inflammatory condition present at the injury site would be expected to respond well to peripherally applied anti-inflammatory medication (i.e. corticosteroid). This may reduce the amount of chemical induced C-fibre afferent discharge. If the injury site is treated early enough, the amount of nociceptor sensitisation and neuroplastic changes should be minimised.

If, however, the injury is undiagnosed and is allowed to remain as an occult inflammatory lesion, then a chronic pain situation may be established. An injury at the styloid process is rarely diagnosed since it is rarely palpated in any orofacial pain examination. Since the symptoms referred from such an injury site mimic those of several pain disorders, the styloid process is frequently overlooked.

The constant peripheral chemical barrage of C-fibre afferents from a chronically inflamed lesion at the styloid process may result in the "wind up" of dorsal horn activity mentioned by Mendell (1966). Also mentioned previously was the expansion of dorsal horn peripheral receptive fields as C-fibre inhibition in the substantia gelatinosa is diminished due to a chronic injury creating and maintaining a chemical stimulated firing of C-fibre afferents. This nociceptor sensitisation is reflected by increased spontaneous activity, reduced thresholds and an increased responsiveness to afferent inputs. The patients in this study that did not report an acute traumatic event resulting in their pain symptoms were complaining of reducing their daily activities for fear of aggravating or initiating their chronic pain symptoms. Most of the patients in the study had an occlusal splint made for them with equivocal success. Unless the underlying source of the complaint is treated, splint therapy may not be as effective as hoped.

Mense (1993) and Perl (1992) indicated how chronic pain can establish a hyperalgesic or allodynic state, the latter causes ordinary loads of daily life to become translated into painful stimuli. The majority of the patients in this study could not remember a single event that would "trigger" their pain. It would seem there does not have to be an acute triggering event if there is an underlying lesion working at lowering the nociceptive threshold thereby predisposing the patient to continued symptoms.

Reeh and Sauer (1997) deduced that most inflammatory states, including post-injury conditions, are associated with hyperalgesia. Certainly every patient in this study was extremely sensitive to external palpation of the styloid process area, indicating perhaps an elevation of a primary hyperalgesic state to an allodynic-type response.

Hoheisel et al (1993) reported that even relatively mild inflammatory lesions of the peripheral tissue (ie myositis) are likely to increase the responses of dorsal horn neurones to ensuing stimuli or may even lead to the appearance of new receptive fields. A chronic chemical exudate resulting in a barrage of nociceptive input results in enlargement of receptive fields and increased excitability in response to mechanical and thermal stimuli. Awakening of silent or "sleeping" (Schmidt et al 1994) nociceptors (including C and A-delta fibres) that are insensitive to mechanical stimuli in normal tissue has been mentioned as occurring during inflammation, thereby contributing to a hyperalgesic state.

This may be an explanation for inflammatory initiated oedema rapidly reducing mechanical thresholds of specific nociceptors (Cooper et al 1991, Cooper 1993) or inflammation initiated swelling of knee joints resulting in C-fibre nociceptor sensitisation to mechanical stimulation (Scaible and Schmidt 1988b). Sensitized nociceptors have lowered mechanical thresholds into the innocuous range (Mense 1993). An inflammatory byproduct, bradykinin, has been shown to reduce nociceptor heat thresholds as far as into the range of body temperatures (Koltzenburg et al 1992) thereby reducing the threshold of the nociceptors into the "everyday"- type range of stimuli. All the patients in this study had palpably raised areas over the affected styloid processes. The author hypothesises that oedema from the inflamed area is responsible. Any abnormal mandibular movement, for example, nocturnal bruxing, may activate sensitised mechanoreceptors sufficiently to be responsible for referred pain symptoms. This may provide a further explanation for the tension-type headaches, especially upon awakening, of the patients in this group, noting a reduction in the intensity and frequency of these headaches after the injection. Nocturnal bruxing may be considered a noxious stimuli by some, in which case the primary hyperalgesic state of the injury site would be stimulated. Any guarded chewing patterns exhibited by patients in this study may not necessarily be related to muscle tightness or myositis arising from a bruxing habit. It may be an avoidance pattern to reduce the chance of stimulating sensitised mechanoreceptors in the area. Bruxing itself may not be enough to result in pain symptoms. There may need to be an additional element of nociceptor sensitisation before a wide range of referred symptoms can result. A reduction of this abnormal peripheral nociceptor input and a return to a "neutral state" (Reynolds and Hutchings 1948) may create an environment where occasional parafunction remains below the pain threshold.

## ii) Reflex Myofascial Spasm

Nociceptor sensitization accompanies all types of tissue lesions and is thought to be the main peripheral mechanism responsible for clinical signs of muscle tenderness (Mense 1993). Simone et al (1991) found that repeated electrical stimuli of muscle nociceptor afferents resulted in cramp-like sensations. Ferrell et al (1988) reported inflammation of the knee joint elicited or facilitated the flexor reflex. A similar distant nociceptive reflex activation of masticatory muscles was reported by Broton and Sessle (1988) following injection of algescic chemicals into the TMJ capsule. Deep nociceptive inputs may activate neurons in subnucleus caudalis which, via their connections with the brainstem reflex centres can result in the co-contraction of agonist and antagonist muscles that can serve to limit movement in pathophysiological conditions affecting jaw musculature (Hu et al 1997). Mense (1993) described any increase in alpha- and gamma-motorneurone activity (resulting in a vicious cycle of pain-spasm-pain feedback i.e. Fig. 5) as more likely if the site of the lesion is outside the muscle. Ligamentous strain has been listed as inducing muscle spasm in associated muscles (Simons and Mense 1998). Such a strain can be inherently painful in the presence or absence of secondarily induced muscle spasm. Unfortunately, the timing and intensity of any EMG activity (if any) does not appear to correlate well with the pain experience (Simons and Mense 1998).

These studies suggest that a distant peripheral lesion may be an initiator and perpetuator of a series of reflex symptoms. Yet no one has indicated a particular site predisposed to such a lesion, if there is such a site? A soft-tissue attachment lesion certainly conforms with all the criteria to be such a site? The resultant reflex myofascial responses from a nociceptive site at the styloid process could result in cramp-like symptoms of the masticatory and associated muscles in an effort to stabilise the injuries at the styloid process. Although anecdotal, any form of protective muscle splinting may be sufficient to result in the feelings of tightness and functional restrictions felt by most of the patients in the study. Parafunction (clenching and bruxing) involving the inflammatory-initiated reflexly contracted muscle could create the ischaemic myofascial conditions needed to establish a hyperalgesic state thereby predisposing the patient to a number of pain symptoms.

The mechanism underlying the formation and maintenance of trigger points is largely unknown. If we are to believe, a persistent undiagnosed soft-tissue lesion constantly releasing sensitizing substances could result in the chronic sensitization of nociceptors and the initiation of local reflex contractions then this soft-tissue lesion-initiated feedback loop could contribute to the maintenance of trigger points and muscle tenderness in our patients.

### iii) "Compartment Syndrome" Influence

"Compartment syndrome" has been defined by Matsen "as a condition in which increased pressure within a limited space compromises the circulation and function of the tissues within that space". Most compartment syndromes are associated with fractures and related bleeding or muscle damage. However they can be seen in any condition that interferes with the blood flow or damages muscle enclosed within a fascial space, for example, vigorous exercise (Wilson and Walt 1996).

The styloid process lies within the lateral pharyngeal space also known as the pterygo-pharyngeal space or the parapharyngeal space or the lateral pterygoid space. This space is bordered medially by the lateral pharyngeal wall and laterally by the medial pterygoid muscle and the parotid gland. The posterior border is comprised of the carotid sheath while the superior border is the base of the skull. The contents of the space are, the carotid arteries, the jugular veins, the vagus, hypoglossal, glossopharyngeal and accessory nerves, the sympathetic chain and lymph nodes. The styloid process and its myotendinous attachments are also found here. This space communicates anteriorly with the submandibular and sublingual spaces, inferiorly with the thoracic inlet and paratracheal region and posteriorly with the carotid sheath, parotid gland and the paravertebral muscles.

Hypothetically, if a soft-tissue lesion at the styloid process results in an inflammatory response, any post-trauma inflammatory oedema that may result from such a chronic lesion could be contained in the lateral pharyngeal space. It may be possible this could create an elevation in intracompartmental pressure which may affect the neurovascular contents of this space.

The results of an inflammatory initiated oedema it may be hypothesised, could contribute to a secondary hyperalgesic state where normally "silent" nociceptors have become sensitised to mechanical stimulation. Any movement in an oedematous, confined tissue space, may be sufficient to stimulate a sensitised nociceptor with a lowered mechanical threshold thereby creating the possibility of pain in the area.

The effect of inflammatory oedema within a tissue space or fascial compartment surrounding the styloid process has yet to be investigated. However, given the number of inexplicable symptoms recorded by the patients in this small group, an hypothesis for the generation of such symptoms could be put forward for comment and discussion. An hypothetical area of chronic injury/inflammation constantly being stimulated by function and occasionally parafunction, may result in a potential fluid build up that may outpace the ability of the lymphatic drainage system to remove it. If contained within a tight fibrous sheath, an increase in intra-compartmental pressure could retard venous return and capillary blood flow causing an ischaemic situation to develop.

It has been reported that peripheral nerves are much more sensitive to ischaemic conditions than muscle tissue (Wilson and Walt 1996). It may be hypothesised that any increase in intra-compartmental pressure brought about by oedema following trauma, could affect the cervical sympathetic chain adjacent to the carotid artery complex and therefore be potentially capable of producing altered autonomic effects. Earlier reports of Carotidynia (Table 1) indicated a direct impingement of this chain by either a medially directed styloid process of normal length or an elongated styloid process thereby resulting in a series of autonomic symptoms. The symptoms are frequently reported yet impingements are rare. The "compartment syndrome" hypothesis as applied to the area of the styloid process may be put forward as a different, further explanation of the symptoms of "Carotidynia" that have been previously linked solely to a hypothetical and, perhaps unlikely, mechanical impingement.

#### 10.1.4 Management

Management, by a specific procedure at the styloid process attachment sites could have a profound effect on a number of symptoms, as was witnessed during this study. Two patients (MBL, MF) experienced neuralgic-like responses (i.e. paroxysmal pain) to light touch and temperature changes on the symptomatic side, akin to an allodynic response. These responses were subjectively reduced after the injection. Patient MBL had a return of these symptoms after some months. In this patient, the injection may have been coincidental with a remission period of an underlying neuralgic problem.

The patients in the study who were able to be treated within weeks of a traumatic episode (AT, SG, MA) had an immediate and long-lasting return to normal jaw function and a pain free state. One of these patients (MA) underwent a general anaesthetic procedure several days after the diagnostic procedure was performed. Soon after, there was a return of the original symptoms. These however were relieved following a further single injection as described. The implication here is that the general aesthetic procedure placed a greater than normal force on the attachments at the styloid process thereby damaging and possibly disrupting their attachment to the bone. This recreated the original injury. The diagnostic procedure directed at this acute form of injury provided a protracted effect for this patient.

As expected, prompt treatment of an acute injury should resolve the current pain state and allow a complete return to normal. It is imperative a rest period that allows only gentle passive motion of the injected areas be instituted post-injection.

Reeh and Sauer (1997) maintain that chronicity is a matter of permanent nociceptor sensitisation due to the permanent presence of synergistic inflammatory mediators.

Nociceptor sensitisation only lasts minutes once the mediators are removed (Koltzenburg et al 1992). This may be a reason the local anaesthetic and corticosteroid injection had such a profound initial effect on many of the study's patients who had their symptoms for many years. Koltzenburg goes on to say, NSAIDS have an appreciable side effect of reducing inflammatory-induced nociceptor discharge and pain, but high, local drug concentrations are needed suggesting a transcutaneous route as well as oral administration. The peripheral specific injection here of local anaesthetic and corticosteroid is supported on this basis.

Research data show that a mild peripheral lesion can lead to central neuroplastic changes that mark the beginning of chronic problems. Interrupting the neuroplastic-inducing input to the spinal cord is the best way to prevent chronic pain (Mense 1997).

Hypothetically, if the source of the pain and dysfunction can be found, precise treatment of such an injury should result in an immediate reduction in the inflammatory component and an initiation of the healing phase, the alleviation of any referred symptoms and a reduced need for a muscle splinting response.

Of the causes of pain in the craniocervical region, the styloid structures are infrequently implicated, but their role may well be more common than is generally realized.

## 11. CONCLUSIONS

Previously, the styloid process was only thought to be of significance if it was elongated. In this study, the statistically significant reduction in pain and dysfunction in patients with styloid processes of "normal" length, suggests that the styloid process area should be included in any examination of orofacial pain patients. It is hypothesised that, a trauma-induced occult inflammatory process releases inflammatory mediators thereby inducing a nociceptor sensitisation and hyperalgesic state. A soft tissue lesion may be considered the source of pain. If the source of the response is to be treated, the clinician must treat the peripheral inflammatory lesions, directly and as soon as possible after the initial trauma. An injection of local anaesthetic and corticosteroid into the area of the styloid process in fourteen patients resulted in a statistically significant reduction in pain symptoms (i.e. referred, TMJ pain, ear pain, eye pain, lateral face pain and cervical pain) and a minor change in maximum jaw opening. These changes suggest that the positive effects of diagnostic injections, as also outlined by Steinman (1968, 1970) and Wong et al (1995). These authors propose a similar theory on the pathogenesis of lateral head and neck pain arising from the styloid process. To include or rule out the styloid process in clinical diagnosis, an injection of local anaesthetic into the area could be undertaken following a careful anatomical review of the region. Adding an anti-inflammatory agent such as corticosteroid, provides both analgesia and a means to suppress possible inflammation to encourage healing of the injured site. If this injection is followed by a period of restricted jaw movement this may encourage longer term resolution.

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