(iii) Pain arising from injury to, and lesions of, the jaws:

It is not proposed to deal exhaustively with the symptomatology of lesions affecting the bones of the jaw. Neurologic symptoms commonly accompany traumatic, bacterial or chemical injuries to the jaws, and these will be examined in a general way.

Traumatic injury to the jaws, resulting in fracture, may damage afferent nerves causing neuralgia, paraesthesia or anaesthesia. In mandibular fractures numbness or paraesthesia of the lips and chin is common, depending on whether the mandibular nerve is divided or merely traumatised. In middle third fractures, beside motor nerve injuries, numbness may affect the nose, infraorbital region, upper lip and more rarely the palate (159). Even when extensive and severe fractures of the jaws have been sustained, accompanied by severe soft tissue injuries, patients seldom complain of much pain except upon movement. (58). Pain may have been experienced at the time of the accident but this usually subsides and only recurs during manipulation of the fragments. Swelling and muscle spasm effectively prevents motion, and hyperaesthesia rather than hyperalgesia occurs in the area. If, however, the fragments are inadequately immobilised, impingement on the nerve trunks may occur with severe pain. In cases of fracture at the mandibular angle exhibiting a vertically unfavourable fracture line, immobilisation must be sufficiently effective to prevent any lingual movement; pain on swallowing usually denotes such movement and nerve impingement.

Pain following the trauma of tooth extraction is commonly met with. This is of variable intensity and of variable duration, and is usually directly related to the degree of trauma incurred during the operation. Laceration of soft tissue, traumatization of the periosteum, and irritation by fractured bone results in after pain which may be severe.

Very occasionally, nerve injury during extraction leads to persistent severe pain in the area supplied by the nerve, usually the inferior dental. This pain may be of exceptional severity
and persistence and seems to be similar to the causalgia which develops after injuries to the major limb nerves (156).

A distressing complication following tooth extraction is the so called 'dry socket' or alveolar osteitis. The blood clot disintergrates, leaving bare bone which is extremely sensitive. Severe neuralgic pain may persist for days or weeks. This complication seems to occur in about 3% of cases (159), - many times in spite of a most exacting operative technique.

The exciting cause of this condition is breakdown, or non formation of the blood clot, with supervening infection of devitalised cortical bone. Certain causes are readily discernible e.g. retained root fragments, retained cyst communicating with the socket, or preexisting acute Vincent’s infection. In many cases no such pathology can be observed and alveolar osteitis probably develops as a result of several complementary factors. Dense bone, possibly sclerosed due to chronic infection, has little blood supply and subsequent haemorrhage will be slight. If this is traumatised and crushed during extraction, and if the blood clot is unable to form due to constant interference with the wound, then it is possible for organisms to gain access to the devitalised bone and cause a localised osteitis.

Fish (55) subscribes to this view. He suggests that during extraction, the vessels in the crushed bone are damaged and the blood in them coagulates. Organisms, displaced from the paradontal sulcus, become imprisoned in the damaged vessel and have an unhampred opportunity of multiplying in the clot before any leucocytes can reach them. The result is sequestration and disintergration of the wall of the socket.

While the above description is an instance of localised osteitis and sequestration, more extensive necrosis may occur if osteomyelitis supervenes. In such cases, infection spreads widely in the bone and severe symptomd develop. Without dwelling on the subject at any length it must be stressed that osteomyelitis is a very rare complication and only develops when certain predisposing factors exist. Such predisposing factors interfere with the nutrition of the bone i.e. the integrity of
the blood supply to the bone, and subsequently increase its susceptibility to infection. Bone necrosis caused by chemicals or irradiation, systemic factors such as diabetes, blood dyscrasias and general debility, combinations of excessive trauma and subsequent infection with virulent organisms, may be cited as a few of the predisposing causes of osteomyelitis.

Symptoms are usually severe and several teeth become tender to percussion. Extreme pain due to periostitis supervenes which may be accompanied by muscle trismus, collateral oedema, lymphadenitis and general complications. If effective drainage is provided the acute symptoms subside and the infection pursues a chronic course with sequestration of necrotic bone.

Bone pains resulting from pathology such as tumour growth or generalised dysplasias have few definitive characters which aid differential diagnosis.

As a rule benign tumours are not painful. The common exostoses such as the tori are never painful. Cysts of the jaw very rarely cause neuralgia or compressive nerve symptoms unless complicated with acute infection.

Malignant tumours, however, are usually painful and severe pain with minimum sensory loss is sometimes observed (151), such as with osteogenic sarcoma. Primary carcinoma, secondary metastases and myeloma may cause vague bone pain but very often the symptoms are those of hypaesthesia rather than neuralgia. Compression and encroachment on nerve trunks followed by their destruction usually occurs.
Figure 43. The points stimulated on the septum are shown by crosses and on the lateral wall of the maxillary sinus as cross hatched circles. The areas in which pain of 1 plus to 2 plus intensity was felt are indicated by crosses within an outline on the small head above. Note that widely separated stimuli cause pain to be felt in the same areas.

Fig 61.

Figure 44. The points stimulated on the turbinates are indicated by crosses, from which lines lead to the indicated areas in which pain of 4 plus to 6 plus intensity was felt.

Fig 62.
(iv). Pain from the Nasal and Paranasal structures:

Pain from the nose and the paranasal sinuses tends to be widely referred, and the diagnoses of the pain source is often difficult to ascertain. The work of McAuliffe, Goodell and Wolff (109) has aided the understanding of such pain states considerably, and their investigations will be considered in some detail. They showed that the linings of the sinuses are, in fact, relatively insensitive as compared with the extreme sensitivity of the ducts, ostia and nasal turbinates.

It is evident, therefore, that the pain and headache of sinus disease is due to the concomitant engorgement of the mucosal covering of the turbinates in the area of the ducts and ostia; that is, the pain source is derived secondarily from nose structures rather than from the paranasal structures as such.

McAuliffe, et al, tested various nasal and paranasal structures for sensitivity and for local and referred pain, using mechanical and faradic stimulation and adrenalin 1:1000 solution. The following paragraphs are a summary of their findings:

Nasal Septum: Pain was felt localised and referred; stimulation of the middle part of the septum caused pain to be felt along the zygoma and towards the ear. Stimulation of the ethmoid region resulted in pain in both the inner and outer canthus of the eye on the homolateral side (Fig. 61).

Turbinates: The lower, middle and superior turbinates were considerably more sensitive than the nasal septum. A sharp burning pain was felt at the site of stimulation and along the lateral wall on the inside of the nose. A duller aching pain was referred into the upper teeth when faradic current or pressure was applied to the anterior portion of the lower turbinate; stimulation of the posterior portions produced pain under the eye, along the zygoma and towards the ear.

Stimulation of the middle turbinate caused pain along the zygoma, extending back towards the ear and into the temple and occasionally deep in the ear.
Stimulation of the superior turbinate caused pain in the inner canthus of the eye, which spread to the forehead and along the lateral wall of the nose (Fig. 62).

These workers found that in subjects with engorged mucous membranes, experimentally produced pain was more intense, was referred to a larger area and was long lasting. Severe and widespread pain with accompanying autonomic effects were noted when adrenalin solution was applied.

**Ostium of the maxillary sinus:**

Touching the walls of the ostium produced intense, sharp pain at the site of stimulation accompanied by profuse lacrimation and injection of the eye. Subsequently, severe aching pain developed in the posterior naso phasynx, in the posterior maxillary teeth, along the zygoma and back into the temple on the side stimulated. The skin over the zygoma was flushed and hyperalgesic (Fig. 63) and this hyperalgesia and dental pain lasted approximately 24 hours.

**Nasofrontal duct:**

Again, severe pain was elicited following stimulation and this occurred at the inner canthus of the eye, under the eye, along the zygoma and into the temple. Pain was also felt at the angle of the jaw and in the last two or three upper teeth. Autonomic effects—lacrimation and conjunctival injection—were again noted (Fig. 64).

**Superior nasal cavity:**

Stimulation in the region of the anterior cells of the ethmoid sinus produced intense pain directly over the eye, and deep in the eye at its inner canthus. Pain was also felt in the upper jaw, just above the canine and premolar teeth. Similar pain, extending more up the side of the nose was experienced when the region of the superior nasal cavity over the posterior ethmoid cells was stimulated. (Fig. 65).
Figure 63. Large crosses indicate stimulation of the ostium of the maxillary sinus. Lines lead to the areas, indicated by small crosses, in which pain of 8 or 9 plus intensity was felt. A dotted circle over the zygoma indicates the area of erythema and hyperalgesia which long outlasted stimulation of the ostium.

Fig 63.

Figure 64. Lines lead from the points stimulated in the nasofrontal duct to the areas in which pain of 5 or 7 plus intensity was felt. On stimulation of the inner wall of the frontal sinus minimal pain of no more than 1 plus intensity was felt only in the area indicated directly over the sinus.

Fig 64.
The frontal sinus:

Stimulation of the walls and roof of the sinus produced only minimal discomfort over the site of stimulation. This is in marked contrast to the extreme sensitivity of the duct.

The maxillary sinus:

Stimulation of the upper wall of the sinus was felt up through the eye. Stimulation of the lower lateral wall was felt in the jaw and posterior teeth. When the mucous membrane close to the ostium was stimulated, however, definite pain was experienced in the upper teeth, in and over the eye and along the zygoma towards the left temple (Fig. 66).

It can be seen that although the term 'sinus pain' is commonly assigned to those headaches associated with sinus disease, the proof that the major portion of the pain has its source in the paranasal structures is lacking. The state of inflammation and engorgement of the mucosa of the turbinates, ostia and superior nasal structures is the basis of most of the pain attributed to the paranasal sinuses. This can be readily appreciated when we consider that sinus inflammation rarely occurs without concomitant inflammation of the nasal structures. The rare exception is sinus infection secondary to periapical abscess, but this type of sinus disease causes but minor discomfort until the nasal structures are directly or indirectly inflamed as well.

Hypersensitivity of the maxillary posterior teeth is also shown to be an indirect or referred phenomenon following stimulation of sensitive nasal structures. In such cases, several teeth may be hypersensitive to vitality tests but tenderness to percussion is absent. In some instances of sinus disease however, toothache is so severe as to simulate pulpitis and this is due to direct irritation of the dental nerves, some of which are in actual contact with the inflamed mucosa.

Clinically, acute maxillary sinusitis presents with pain over the malar region, the pain being of a deep, dull, aching, non pulsatile quality which is seldom, if ever, associated with
Figure 57. Crosses indicate the points of pressure against the walls of the superior nasal cavity in the region of the sphenoid and ethmoid sinuses, with the indicated areas in which pain of 5 plus to 6 plus intensity was felt.

Fig 65.

Figure 58a. The thin rubber balloon is shown in the maxillary sinus. The areas are indicated in which pain was felt when positive pressure was applied to the walls of the sinuses for prolonged periods.

Fig 66.
vomiting or nausea (81). Accompanying headache may be severe.
Pain and headache from nasal and paranasal disease varies in
intensity according to posture and movement. The intensity of
pain is increased by shaking the head or by the head down
position and by procedures which increase the venous pressure,
such as coughing or a tight collar. Also it is intensified by
states that increase the engorgement of the mucosa, such as
anxiety, menstruation, cold air, or the effects of alcohol (104).

Pain associated with maxillary sinus disease gradually
diminishes when the patient assumes a lying down position with
the diseased side uppermost, allowing drainage through the ostium.

Since the anterior ethmoidal sinuses, the frontal and
maxillary sinuses have a common place of drainage into the middle
meatus, pansinusitis is not an infrequent complication of
frontal sinusitis, and the pain may be widespread over the frontal
and maxillary regions of the face due to secondary contraction
of cervical and fifth muscles (180). Commonly, when of sufficient
duration, pain may be experienced in the back of the head, neck
and shoulders.

Pain is not such a marked feature in infection of the
sphenoidal and posterior ethmoidal sinuses, but face pain and
headache may be observed between and behind the eyes and over
the vertex of the skull (81, 151).

With regard to sinusitis caused by factors other than
dental disease, it must be remembered that it is a recurrent
condition and frequently manifests itself in conjunction with
upper respiratory tract infectious or activities producing intra
nasal changes such as swimming. Tension and vascular cephalalgias,
which are also recurrent, produce sinus like pains owing to the
manifestation of sympathetic symptoms including swelling of the
nasal mucosa (37).

History is of first importance, but diagnosis is aided by
occipito-mental radiography for sinus opacity; examination of the
turbinates for pus and engorgement; and naso-pharyngoscopy for
post nasal discharge.
(v) Pain arising from the temporo-mandibular joint:

Pain in and around the temporo-mandibular joint is of common occurrence, either due to joint pathology or as a result of pain referred from surrounding muscles. While the symptoms and signs of derangement due to infection or acute trauma are usually diagnostic, the same cannot be said about those derangements due to chronic degenerative processes viz. rheumatoid arthritis, osteoarthritis and other chronic arthroses.

Bellinger (13) has classified internal derangements of the joint into four categories: traumatic arthritis, infectious arthritis, rheumatoid arthritis and degenerative joint disease. In view of the fact that osteoarthritis is a clinical and pathological entity and osteoarthritic changes are rarely found as the underlying pathology in the arthroses associated with malocclusion, it is considered necessary to subdivide the latter group so as to distinguish between the two conditions.

Pain in traumatic arthritis:

Severe blows or extreme motion of the mandible may cause damage of varying degree to the joint e.g. tearing of the ligaments or the capsule, injury to the meniscus or fracture of the condylar head. The resultant inflammation causes pain, swelling and tenderness which may become exquisite with opening or on palpation. The diagnosis is usually obvious since the association of pain immediately after the traumatic episode is readily recalled (26, 157).

While pain is felt following fracture dislocation of a condyle, it is interesting to note that persistent chronic pain is a rare symptom even though treatment is conservative without surgical intervention (103). It would be expected that severe derangement of the joint would persist with predisposition to recurrent dislocation and chronic arthrosis.

Pain in infectious arthritis:

This is due to specific organisms which may involve the joint following direct wounds, surgical intervention, haematogenous
spread or by direct extension from the parotid gland, the ear, the mandible or surrounding tissues. Acute arthritis associated with acute febrile diseases such as scarlet fever, pneumonia and measles or due to the gonococeus are also known (157, 81). Symptomatically the local picture is one of pain, swelling and loss of motion, associated with temperature rise and leucocytosis, even an attempt at passive motion is extremely painful and there is no question in the mind of the patient as to the cause of the symptoms.

**Pain in Rheumatoid Arthritis:**

Involvement of the temporomandibular joint alone is rare, and usually there are associated flexion contractures of other joints.

The disease has a long history with an insidious onset and the first sign of joint involvement is pain and swelling in the region due to periarticular and intra-articular inflammation. Onset may be at any age but is more common between the ages of 20 and 45 in women, and there is a tendency for progression of the disease from peripheral joints towards the trunk.

The cause of the disease is obscure. The inflammatory process suggests an infectious etiology but no causative agents have been found and association with focal infection has not been proved.

Inflammation is not acute and the pain is described as a constant dull or aching type intensified by movement. Due to this pain on mastication and progressive deformity of the joint, the range of movement of the jaw may become permanently limited and malocclusion may be noticed. Stiffness due to fibrous adhesions is seen but bony ankylosis is rare.

**Pain in Osteoarthritis:**

Osteoarthritis is a chronic arthropathy characterised by degeneration and hypertrophy of the cartilage and bone, and clinically by pain. It is generally believed that this condition is related to the physiologic process of ageing aggravated by strain, trauma and dysfunction (26). Commonly found among males
in the older age group, we find eburnation of the joint surface together with proliferation at the osteocartilaginous junction and osteophytic outgrowths. The articular fossa becomes shallow due to erosion of the articular eminence, and gross deformity may result.

Clinically these arthritic changes make themselves apparent by the insidious onset of aches and pains, particularly on movement. A dull ache may persist during rest. Stiffness and limited mobility follow, but in some cases hypermobility due to looseness of the joint capsule may result. Along with this irregularity of condylar excursion, there may be cracking or grating sensations due to breakdown of the meniscus.

**Temporomandibular Arthrosis:**

In recent years there has been increasing stress on Costen's Syndrome in which temporomandibular joint pain arises without gross pathology therein. It is usually seen in edentulous or partially edentulous patients over the age of 40 in which the joint is overstrained due to malocclusion or loss or molar support with overclosure of the jaw (33). It is characterised by pain of moderate intensity in parietal, supraorbital and occipital regions, and about the ear and in the lower jaw, which is aggravated by prolonged mastication. In addition, some patients complain of burning of the throat, tongue and side of the nose. (111)

In most cases of temporomandibular arthrosis mutilated occlusions are seen - deep overbites, teeth in crossbite, centric prematurities and interferences and distortion of the occlusion due to failure to replace missing teeth.

Stones (151) describes how such abnormalities in the dental arch may result in changes in the joint. "After the extraction of teeth the correct vertical dimension of the jaws is not maintained when making dentures, so that there is overclosure. The position of the head of the mandible in the articular fossa is not centrally placed but encroaches upon the posterior joint space. This abnormality may lead to thinning
of the tympanic plate and may affect the interarticular disc which also may be thinned.

This disequilibrium may affect the cartilage layer of the subchondral bone, which leads to progressive alterations in the surface of the disc and ... the joint.

Thoma (157) elaborates on these internal derangements of the joints. Tearing and thickening of the disc, sometimes with anterior or posterior detachment may cause various clicking noises during condylar excursions. In many cases articular eminence may become flattened out and new bone is deposited in the fossa. The concave convex architecture tends to become smoothed out and a curious bending forward of the condylar neck occurs. Frank osteoarthritic changes are rare.

Brussell (25) examined joints in cadavers and while many showed considerable looseness of the articular cartilages and displacement of the disc, there were no obvious perforations and few erosions and irregularities of the joint surface were seen.

In 1934 Costen (40) proposed a syndrome of headache, facial pain and ear disturbances which were the result of these internal derangements in the temporo mandibular joint produced by overclosure. The symptoms as described by Costen include:

(a) Ear symptoms - tinnitus, impaired hearing, pain in and about the ears;
(b) Joint symptoms - tenderness on palpation, clicking of the joint on movement, subluxation or deviation, pain on movement and either hypermobility or limited mobility of the joint;
(c) Head symptoms - headache localised to the vertex, occiput or behind the ears;
(d) Nasopharyngeal symptoms - painful burning or pricking sensation in the tongue, throat and the side of the nose.

While Costen's syndrome is still basically accepted, there has been a great deal of controversy over certain of his findings and anatomical explanations of the neurological symptoms observed.
Wing (172), after surveying the literature on the subject, concludes that ear symptoms are present less frequently than was thought by Costen and considers that they should be excluded from the syndrome.

The basic assumption that the syndrome invariably develops as a result of overclosure of the vertical dimension or occlusal imbalance has been challenged by Campbell (27). "The occurrence of pain in endentulous persons, and in those who have recently suffered occlusal breakdown has thrown suspicion on mandibular overclosure as a causative factor, and, in fact, the pain will occasionally disappear when the occlusal level is raised. However, overclosure is by no means the sole cause; indeed the condition has been recognised in subjects with immaculate occlusions".

Campbell proceeds to quote examples of cases where pain was experienced on the side where the joint was seemingly healthy, while no discomfort was experienced in the joint which was plainly pathological.

This paradox of pain in the contra-lateral side is not uncommon and in itself throws doubt on Costen's explanation of the cause of pain.

In a large proportion of cases, however, the combination of clicking of the joint, pain either local or referred, and occasionally glosso-pharyngeal symptoms, is seen associated with occlusal disequilibrium.

Costen reasoned that pain symptoms were produced as follows:

(1) Vertex pain is caused by pressure on, and erosion of, the roof of the glenoid fossa, thus allowing direct stimulation of the pain endings in the dura.

Wing (172) queries whether this thinning of the tympanic plate does in fact occur. Apart from the cushioning effect of the meniscus and the occlusion of the teeth, it is feasible to assume that any pressure resorption of the roof of the glenoid fossa would be compensated for by the laying down of
new bone on the cerebral side of the roof. Histologic evidence points more to the remodelling of the condylar head and the fossa, rather than a tendency to perforation.

Again, MacRae (106) and Wolff (180) state that most of the dura is insensitive to pain, the pain receptors existing predominately in the walls of the dural and meningeal arteries.

Cohen (33) suggests that pain may be produced by distortion of the thin tympanic plate which ends in a free edge of the petrotympanic fissure. This may compress the 5th, 7th, 9th and 10th cranial nerves with pain of wide distribution including the tongue, side of the nose, local tenderness and ear symptoms.

As previously stated however, the degree of direct pressure delivered by the condyle on the tympanic plate is a matter of some speculation.

(ii) Pain in the temporal region, states Costen, is due to pressure on the auriculo-temporal nerve which passes in close relation to the medial side of the capsule and between the condyle and tympanic plate. Thoma (157), Harrigan (81) and Thonner (160) subscribe to this view.

Although Costen reaffirmed this conclusion in a succeeding paper (39) it is extremely doubtful, in view of anatomical relationships, whether such impingement could occur. Sicher (142) has shown that the auriculo-temporal nerve does not cross the condyle but crosses the mandibular neck below the insertion of the external pterygoid muscle, the insertion of the capsule and the level of the tympanic bone.

(iii) Glossodynia, states Costen is due to impingement of the chorda tympani nerve by the condyle as it is thrust upward, backward and medially through a perforated meniscus.

Sicher (142) (140) considers this erroneous for the chorda tympani nerve is safely tucked away in the depth of the petro-tympanic fissure to emerge from the protection of the bone only at the medial surface of the angular spine of the sphenoid. Only fracture of bones, he states and never displacement of the condyle, could endanger these nerves.
Dechaume et al (42) state that only a disturbance of the sympathetic nervous system can explain both the articular difficulties and the related sensory disturbances. Impressed by the rich periarterial innervation of the maxillary arteries and the connection of the temporomandibular articulation with these plexuses they stated that irritation originating in the articulation spreads to the sympathetic fibres around contiguous blood vessels, resulting in symptoms of the most 'varied types'.

The work of Sicher (142), (140), (141), Campbell (27) and Schwarz (131), (132) have done much to clarify the problem and it now appears that pain arises most often in the musculature.

Campbell carried out an extensive survey of temporomandibular arthralgia patients, and divided them into 20 groups depending on the distribution of facial pain. He found that pain distribution was remarkably symmetrical and the common sites of occurrence were (i) the temporomandibular joint (ii) the gonial angle (iii) the ear (iv) the zygomatic arch (v) the anterior part of the temple (vi) the submandibular space. In addition, there was a surprising amount of pain in the suboccipital region some of which was relieved coincidentally with the facial pain.

Such pain distributions defy rationalisation on a neurologic basis.

Why, for example, is pain restricted to the posterior end of the mandible behind the point of entry of the inferior dental nerve, rarely appearing anteriorly to the first molar?

If this truly represents neuralgia surely the pain must run to the middle line more often.

Campbell points out that the dense concentrations of pain occurrence are coincident with the origin and insertion of muscles. The pain in the anterior part of the temporalis could be attributed to a straining of appropriate fibres. These particular fibres would, in fact, be stressed when the condyle is forced behind its physiological position. Again, pain at the zygoma and at the gonial angle coincides with the attachments
of the masseter. Pain in the glands could emanate from the suprahypoid muscles.

As far as pain in the joint itself is concerned, it is only to be expected that pain would converge here, the meniscus becoming the focal point of inordinate muscle tension.

While direct pressure of the condyle on nocrve trunks is very improbable, the sharp stabbing pain commonly complained of in the region of the joint may well be due to pressure, direct or indirect on the numerous nerve endings present in the ligaments, blood vessels and in the fibrous zone posterior to the condyle.

Sicher (14) agrees with Campbell and states that local joint pain is caused by pressure of the condyle on the loose connective tissue behind the disc. Chasens (29) adds that oedema within the joint itself may be an indirect source of pressure thus producing symptoms of pain.

Certain facts tend to support the hypothesis that extraarticular pain is usually muscle pain

(i) The pain is usually described as dull, constant and aggravated by mandibular movements and this coincides with Lewis' (100) findings on muscle pain characteristics.

He found that pain induced in muscles by injecting hypotonic saline was indistinguishable from that provoked by the same muscles working under ischaemic conditions. The nature of the pain - disagreeable, rather diffuse, difficult to locate and continuous - suggested a similarity to that described in above patients.

(ii) Pain receptors are relatively scarce in the belly of a muscle, but are abundant at the tendinous ends of muscle (27). It is also known that tendon and fascial sheath gives a relatively localised pain while muscle gives diffuse pain (100).

These facts may explain the concentration of pain at the origins and insertions of muscle in the region of the temperomandibular joint.
(iii) For the muscles to become painful, prolonged tension is necessary; sustained tension without commensurate blood supply will starve the muscle of their metabolic requirements - nor will the muscle be flushed of the painful waste products of metabolism. After all, Campbell reasons, if leg muscles are subject to cramp, why not the masticatory muscles or even the muscles of facial expression?

It is well known that a painful tooth may compel a bite of accommodation, and if this abnormal action continues for any length of time, then the muscles might get cramp and become tense and painful. Similarly if the occlusion was such that it forced the mandible into an eccentric retruded position, then the muscles would inevitably take a lot of strain in an effort to protect the joint.

Sicher (142) asserts that "pain in the temporal region is pain in the spastic temporal muscle. The pain in the cheek and jaws is from pain in the masseter muscle; pain in the throat is from pain in the lateral and medial pterygoid muscles; pain in the tongue is from pain in the digastric and geniohyoid muscles. These muscles if accessible to examination, are found to be extremely sensitive to touch, especially in the morning after a night of spastic contractions".

Sicher (141) visualises a disturbance of the normal proprioceptive nerve endings in the muscle, articular capsule and the periodontal ligaments. In some cases of occlusal imbalance, disturbance of the proprioceptive signals may lead to over-excitation of the mandibular musculature and bruxism. "Such disturbances are self perpetuating and self aggravating".

Schwartz (131) instances the investigations of Travell et al (163) who described a group of disorders termed 'myalgesias' which are characterised by muscle spasm. In these conditions pain and limited motion are associated with trigger areas i.e. small areas of the myofascial structures hypersensitive to stretch and giving rise to muscle spasm with referred pain. Examination of above arthralgic cases discloses the presence of tender muscles and the effectiveness of local anaesthetics followed by
therapeutic exercises point to the fact that temporo mandibular joint pain may be due to a painful self perpetuating spasm of the masticatory muscles. This concept of inco-ordination of the muscles with the joint and dental arches, followed by painful spasm of the masticatory muscles is, however basically similar to Sicher's views.

In a later paper (132) Schwartz stresses that this painful limitation of mandibular movements due to muscle spasm is aggravated by excessive function and most cases examined seemed to be highly tensed individuals with noticeable oral habits.

Schwartz feels that while malocclusion is important, in that it can undoubtedly increase the amount of force exerted by the musculature and alter its direction, of far more significance is what the patient does with his occlusion when reacting to stress.

Changing an existing occlusion by grinding or restoration is frequently associated with the onset of the syndrome.

It seems that anxiety is a most frequent underlying cause contributing to the development of these pains dysfunction syndromes, and in many people, increased muscle tension and a tendency to muscle dysfunction is seen previous to local dental trauma, such as extractions, which may be cited as the cause.

Dental malocclusion or local trauma of a minor or major nature is not in itself liable to produce such a pain syndrome. Some predisposition, such as described, above, seems to antedate any such occurrence.

If this were not the case, it would be expected that severe derangements of the joint following fracture dislocations of the condyle treated without surgical intervention would be a frequent source of joint pain. Macgregor (103), however, from a perusal of many case reports states that chronic pain is very rare.
(vi) **Ear Involvement and facial pain:**

For its size, the ear has a larger nerve supply than any other organ in the body, receiving twigs from the 5th, 7th, 9th and 10th cranial nerves and C1 and C2. Details of this innervation are given in Chapter 4. In view of this, noxious stimuli from parts remote from the ear may cause pain to be apparent in the vicinity of the ear. For example, pain that occurs due to irritation of the third division of the 5th nerve from pulpitis may be referred to the ear (151). Again, pain associated with tonsillitis, with pharyngitis and with disorders of the tempero mandibular joint may be felt in the ear. Harrigan (81) states that aural pain may also be associated with disorders of the cervical spine, as the scalp and muscles in the immediate vicinity of the ear are supplied by branches of the upper cervical roots. Pain in the ear may become associated with typical and atypical facial neuralgias and follow virus infections such as herpes.

Conversely, noxious stimuli emanating from the ear may give rise to referred pain along the nerves of supply i.e. to the front of the head, the naso pharynx, larynx and back of the head and neck (151).

The commonest affections of the ear are otitis externa and otitis media. Pain from aural furunculosis is severe and radiates down the side of the neck. The ear is extremely painful to motion and there is associated rigidity of the masseter and temporal muscles.

Otitis media may be preceded by recurrent upper respiratory infection, and depending on its duration, the pain may be referred from the malar areas to the vertex and occiput of the skull. The pain is throbbing in character and the patient complains of difficulty in hearing associated with a heaviness and fullness in the ear. Tenderness of the sterno mastoid muscle and muscles of the neck may be present.
Fig. 39. Sensitivity of deep ocular structures and areas of pain reference

Fig 67.
(vii) Pain arising from the eye:

Apart from acute infections such as iritis and glaucoma, eye disorders are not a common cause of persistent facial pain (156). Elevation of intraocular pressure is a cause of severe pain and in some cases of glaucoma, pain is felt over the distribution of the opthalmic division of the fifth nerve (137), (180) and occasionally in the upper jaw (33). (Fig. 67).

The headache which is frequently experienced with visual disturbance, is not due to structures within the orbit, but arises from the increased contractile state of the muscle of the head in association with the tension accompanying visual difficulties.
Muscles of the head and neck as a source of pain:

Muscle pain may become apparent in various ways. Lewis (100) has remarked that muscles may be the source of pain and tenderness after steady voluntary contraction of two minutes duration, and tenderness of a muscle which has been continuously contracted for hours or days, may outlast the actual contraction as it does after unaccustomed exercise. It has been shown that long continued pain in the head or emotional tension is accompanied by muscle contraction in the scalp and neck, and that when this muscle contraction is sufficiently sustained, pain in the neck or dysaesthesia of the scalp develops. That this pain is due to muscle spasm is verified by Simons et al (143) who showed that procedures which increase the action potentials increased the head pain, and vice versa. Since action potentials are an indication of muscle contraction, the inference is justified that sustained contraction of the skeletal muscles of the scalp and neck is the source of pain. Furthermore, the character of the pain - a deep, steady ache rather than a throbbing ache or sharp pain indicates that the pain is not vascular or cutaneous in origin.

Travell's work (163) has been cited in relation to the possibility of muscle pain being the basis of temporo mandibular arthralgia (page 228). He described a group of disorders termed myalgias characterised by muscle spasm.

In these conditions there appear to be trigger areas in the muscle or connective tissue which are hypersensitive to stretch and which cause a syndrome of pain, muscle spasm and tenderness. Monica (111) claims that this 'myofascial pain syndrome' may affect such muscles as the sterno mastoid, temporals and masseter and give rise to obscure facial pain. He states that such trigger areas may develop as a result of acute myositis, nerve injuries or arthritis and that such abnormal foci can be activated by pressure, motion or by intense heat or cold, acting as a chronic focus of noxious impulses.

Attention has already been drawn to the pain following
on muscle injury or inflammation. The continuous aching pain resulting from local anaesthetic administration where the needle is not sharp, or the solution is cold or non isotonic, or where injection is carried out too rapidly, is a common clinical observation.
(ix) **Pain caused by elongated styloid processes:**

This uncommon pain syndrome may be caused by a grossly elongated styloid process, which may ossify to a length of up to 6 cms. Battensby (11) and Stones (151) state that symptoms may be due to compression of the 5th, 7th and 10th cranial nerves while Donohue (46) observes that the glossopharyngeal nerve is most frequently involved, the 7th and 10th nerves being rarely affected.

Eagle (49) believes that pressure on either the external or internal carotid arteries and the perivascular nerve plexuses, may in some cases cause further disturbances.

Elongated styloids do not invariably cause pain but the syndrome is most frequently found among males after the third decade (46). Symptoms of dull pain in the pharynx, dysphagia or the sensation of a foreign body in the pharynx may be present. In some cases, otalgia and generalised head pains may be observed. Donohue quotes a reported case where excruciating pain radiating along the 3rd division of the 5th nerve and accentuated by chewing was observed.

Diagnosis is aided by radiographs and digital examination of the tonsillar region on the involved side.
(x) **Facial Pain due to heart disease:**

Patients suffering from myocardial ischaemia due to coronary artery disease sometimes present with severe substernal pain which may radiate to the throat, jaws or arms (156).

Very occasionally, the substernal element of this pain is missing and the patient may complain only of pain in the jaw. This is usually felt in the lower jaw, often more marked on the left side, and occurs especially in the cold or during emotional stress (33). Stribling (153) reports a case where a throbbing and aching localised pain in the angle of the left mandible of a 43 year old man preceded more common symptoms of angina pectoris.

The extensive reference of cardiac pain to the jaw is due to the close association between the spinal nucleus of the 5th nerve and the top of the dorsal column of the grey matter of the spinal cord. Reference to the arms and neck occurs via the upper cervical nerves and the great auricular nerve (151).
Vascular Pain and Headache:

There is considerable evidence that deep pain can be produced by periodic vascular disturbances, particularly vasodilatation and by traction or displacement of blood vessels.

As pointed out in Chapter 3, blood vessels possess a sparse sensory supply as well as an autonomic innervation, the former fibres supplying both vessel walls and adjacent connective tissue. These sensory nerves reach the vessels either as direct branches of the spinal and cranial nerves or by passing through the sympathetic and parasympathetic nerves and plexuses where they mingle with the true autonomic motor fibres of these systems (50).

Pain from blood vessels is similar in character to deep pain from visceral structures—diffuse, difficult to localise, having superficial and deep reference and may be accompanied by feelings of illness.

The pain engendered by vascular diseases may be of a dual origin—both from the affected vessels and from the tissues whose circulation is altered.

That pain from blood vessels is in itself severe, is apparent when considering Berger's disease—the characteristic arteritis and phlebitis is typically extremely painful. The pain of vessel inflammation is also noted in temporal arteritis and polyarteritis nodosa.

Where vascular disturbance involves vascular obstruction, however, the resultant ischaemic changes in the tissues supplied by the involved vessel are more generally productive of pain (189). For example the syndrome of intermittent claudication seen after exercising an arterio-sclerotic limb is associated with intense cramp like pain; Raynaud's Disease produces a combination of numbness and pain.

Physical stimuli necessary to produce blood vessel pain cause deformation—puncture of or traction on a vessel is painful (174). Wolff and Hardy also demonstrated that pain is
produced by vessel dilatation but they doubt that vessel constriction is painful.

Vessel dilatation seems to be the basic cause of most headaches, and migrainous conditions (189). Behrman (12) states that dilatation leads to increased amplitude of pulsation of the affected arteries and factors which decrease the amplitude of pulsations also decrease the intensity of headaches.

The cause of dilatation of the cranial arteries is not clear. Some authors (37, 177) point out that tensions produce vasoconstriction, muscular contraction and ischaemia in the vessel wall. This ischaemia causes damage to the endothelium of the capillaries with subsequent dilatation and fluid leakage in the surrounding tissue spaces.

Goltz (189) considers that vasodilatation following vasoconstriction may be due to either vasospasm of a vessel's vasovasorum with subsequent oedema in the parent vessel wall, or it may be due to dilatation of arteries proximal to constricted terminal arterioles.

In either event, the periarterial sensory nerve plexus is stretched and pain occurs, which may be referred to some area of the head or face.

Migraine is explained in this fashion – the prodromal scotomata occurring in the vaso constriction and ischaemia stage, headache in the vasodilatation stage and residual low grade headache and nausea during the subsequent cerebral oedema. Interruption of the process during the prodromal phase with vaso pressor drugs such as ergotamine tartrate may thus abort an attack.

Other suggest that the eventual vasodilatation is the result of the formation of histamine as a degradation product of the amino acid histidine. Histamine is a powerful vasodilator and it has been demonstrated that administration of this substance to susceptible persons produces several temporal headache due to dilatation of the cerebral arteries and an increase in cerebro spinal fluid pressure. Wolff (180) states that headache following
injection of histamine seems to use the fifth nerve on each side as the principle afferent pathway for headache, resulting from dilatation of the supra tentorial cerebral arteries, and is felt in the fronto-tempero parietal region of the head.

While some people have been relieved of migrainous conditions by 'desensitisation' i.e. fractional injections of this drug, there is now considerable doubt that histamine is the aetiologic factor in any but rare cases. It has been the experience of recent investigators (130) that small doses of histamine fail to provoke a characteristic attack in the majority of patients while larger doses produce non specific histamine headaches in a fair number of susceptible persons.

Wolff (178) points out that headache may be induced with either increased or decreased blood volume, when there are no compensatory changes in the tone of the vessel wall and some traction occurs.

It must be remembered that any of the many vessels and branches of vessels in the head and neck may be involved in vascular pain and the clinical picture may be quite variable.

Vasodilatation pain arising in one or other of the branches of the external carotid may account for much of the pain in the face, head and neck for which no definite and obvious pathology can be found.

Before considering specific types of vascular pain, it is considered necessary to examine the sensitivity to pain of the cranial arteries and intracranial structures and relate the area of pain reference to the structures concerned.

Of the intracranial structures, the great venous sinuses and the venous tributaries from the surface of the brain, parts of the dura at the base, the dural arteries and the cerebral arteries at the base of the brain are pain sensitive (180, 106). The sensory fibres of the fifth nerve supply the superior surface of the tentorium cerebelli and all pain sensitive areas above it. The nerve fibres are closely related to the blood vessels and come from all three divisions of the trigeminal. Pain
resulting from stimulation of intracranial structures innervated by the fifth nerve is perceived in the topographic distribution of the fifth nerve.

The inferior surface of the tentorium and the pain sensitive structures in the posterior fossa are supplied by the 9th, 10th, 11th and 12th cranial nerves and the upper three cervical. Wolff states that the 9th and 10th cranial and upper cervical nerves are the most important afferent pathways for headaches resulting from dilatation of the arteries of the posterior fossa and are felt in the occipital region of the head.

The cranium itself, the diploic and emissary veins, the parenbhyema of the brain, most of the dura, most of the pia, arachnoid mater, the appendymal lining of the ventricles, and the choroid plexuses, were not sensitive to pain.

From the above data, six basic mechanisms of headache from intracranial sources may be formulated (180).

(i) Traction on the veins that pass to the venous sinuses from the surface of the brain and displacement of the great venous sinuses.

(ii) Traction on the middle meningeal arteries.

(iii) Traction on the large arteries at the base of the brain and their main branches.

(iv) Distention and dilatation of intracranial arteries.

(v) Inflammation in or about any of the pain sensitive structures of the head.

(vi) Direct pressure by tumours on the cranial and cervical nerves containing many afferent fibres from the head.

Extracranial arteries are in general very sensitive and as a source of pain, the cranial vascular structures far outway in number and distribution all others.

(1) **Headache:**

Headache is a common complaint and may be associated with disease in other parts of the body, being a symptom rather than
a local affliction.

They may accompany various somatic disorders such as gastritis, constipation, acute alcoholism or acute infections with fever. More local conditions which cause headache are sinusitis, especially frontal and ethmoidal, eye strain or diseases of the eye and various cerebral disturbances, such as concussion, meningitis, or cerebral tumours (157).

There is great variation in the location, depth, duration, frequency, intensity and associated signs and symptoms of headaches.

The headache may be fairly diagnostic in some cases however. For example in hypertension, lateral, occipital and frontal headaches occur regularly in the morning and soon wear off. With regard to hypertensive headaches, it has been found that the headache bears no direct relationship to the level of the blood pressure. For instance when ergotamine tartrate was used, the headache markedly decreased - the amplitude of pulsation of the cranial arteries being decreased. This was in spite of the fact that the ergotamine considerably increased the already elevated systolic and diastolic pressure. It seems that a degree of change in the contractile state of the arterial wall compatible with comfort when blood pressure is average, would be associated with pain when the blood pressure is elevated (180).

The role of muscle tension in producing pain and headache has already been referred to (page 233). Monica(110) comments on the recurrent headaches and pain in the area of the temporo mandibular joint often found in association with malocclusion and bruxism. Forceful and prolonged contractions of the temporal muscles may cause bilateral headaches in such cases.

(2) Migraine:

Migraine has been characterised as a familial, periodic sick headache with a vascular component (171). The headache is typically a one sided hemicrania but at times it is bilateral or shifts from one side to the other. They are frequently
associated with unpleasant affective states and in many cases with visual disturbances.

In such cases the pain is preceded by subjective phenomena such as scotomata or clusters of bright lights which gradually increase until there is almost total blindness. As the scotomata disappear the headache begins. Other prodromal phenomena are nausea and vomiting or giddiness and numbness of the face, neck and arms (157) (189).

Although most attacks of migraine are limited to the temporal, the frontal or the occipital region, some patients have pain elsewhere - in the face, below the eye and behind and below the zygoma.

Severe throbbing pain which seems to emanate from the back teeth of the upper jaw occasionally occurs. Another variant is facial pain which spreads behind the angle of the jaw down the neck and into the shoulder (180). Nausea and vomiting may occur at the height of the attack.

Evidence indicates that the headache of migraine is produced mainly by the dilatation, or changes in the amplitude of pulsations of chiefly the branches of the external carotid artery. The pains described probably result from distention of the extracranial portion of the middle meningeal artery between its origin and its point of entry into the skull, the internal maxillary artery, and the trunks of the external and common carotid artery. There is some evidence, according to Wolf, that the pial and cerebral arteries may contribute to the pain, since faradic stimulation of the proximal few centimetres of the anterior, posterior and middle cerebral arteries and the first few centimetres of the intracranial portion of the internal carotid artery causes pain, which occurs behind or over the homolateral eye.

Wolf (177) considers there is a stage of arterial constriction which is not painful followed by arterial dilatation, and anything which will constrict the painfully dilated arteries will relieve the pain. Herein lies the rationale for administer-
ing ergotamine tartrate to abort migraine attacks. It has been found that ergotamine tartrate reduces the amplitude of pulsation of these arteries by about 50%.

White and Sweet (171) mention that permanent relief may be obtained surgically by: (i) Prevention of arterial distention by proximal occlusion of arteries or by division of vasodilator nerves or (ii) the cutting of the afferent fibres transmitting pain impulses from the dilated vessels to the sensory areas of the brain.

(3) Paroxysmal Migrainous Neuralgia:

Migrainous neuralgia is the term now applied to a syndrome of vascular headache which in the past was classed among the rather uncertain group of atypical neuralgias. Although Horton (90) in 1939 is commonly cited as the person who primarily described this syndrome under the name "erythro-myalgia of the head" -, many earlier writers had observed similar examples of this syndrome. A number of Sliidier's patients with 'sphenopalatine neuralgia with sympathetic signs' (146) and Glaser's atypical neuralgias with associated 'sympathetic phenomena' (57) probably included many of this category. In 1925, Vallery-Radot and Blamantier (quoted by 171) had described a case of the disorder as the syndrome of 'hemicephalic vasodilatation'. Other names given to this syndrome include 'ciliary neuralgia and migrainous neuralgia' (Harris 1926 (84)); 'Vidian neuralgia' (Vail. 1932 (166)); 'Petrosal neuralgia' (Gardiner 1947 (64)); 'Autonomic facio-cephalalgia' (Brickner 1935 (23)); 'Limited migraine variant' (Schiller (130)); 'Cluster Headache' (Kunkle) and 'Atypical facial neuralgia' (Fay 1932 (52)).

In 1941 Horton renamed this syndrome 'Histaminic cephalgia' (91) for he found that the attacks of pain could be reproduced by subcutaneous injections of histamine and in some cases complete relief could be obtained for varying periods after desensitisation by progressively increased minute doses of histamine (see also page 238).

Hence the name - 'Horton's Histamine Headaches' under which this syndrome is still commonly known.
While the condition seems to be somewhere between migraine and trigeminal neuralgia, it has features which distinguish it from both.

Less frequent than migraine, it is probably more common than Tic Douloureux. It occurs in both sexes but favours the males as much as 8:1. The age incidence is highest in 3rd - 5th decade (130, 156). In contrast to the hereditary background of migraine, familial tendencies are very rare (171).

While attacks of migraine may last a whole day recurring perhaps every few weeks, and attacks of tic douloureux last a few seconds at intervals of minutes to hours, the headaches under consideration have a tendency to occur in cycles, bouts or clusters lasting for four to eight weeks. A single attack has a duration of one half to two hours, and in the course of a day during such a cycle the patient will experience one or two attacks. Characteristically the attack will wake the patient one or two hours after he has fallen asleep, continue for an hour or two and subside perhaps to wake him later for another attack (130, 171, 156, 104). During an attack, the reclining position is intolerable, perhaps because of additional engorgement of the vessels of the head. The attacks are rarely accompanied by nausea, vomiting or visual disturbances. Once the cycle is over, the patient may enjoy freedom from pain from a few months to many years.

The centre of the pain is usually one orbit, the side remaining constant in the cycle and rarely changing from this during the next bout. From behind the eye the pain spreads mostly laterally and backwards towards the temple, around and behind the ear and sometimes downward. It affects the side of the nose, the zygoma and the maxilla together with the upper teeth (130). As the headache matures, the upper molars may ache (37). Occasionally the pain may spread to the carotid triangle and very frequently extends beyond the confines of the trigeminal zone.

The attacks are associated with striking manifestations
of cranial autonomic activity on the side of the pain. There is conjunctival and often nasal congestion, and lacrimal and nasal discharge. A red, tearful eye and stuffy running nostril, is diagnostic of Horton's syndrome (156, 171). Ptosis and constriction of the pupil is also a common observation.

As far as possible precipitating factors are concerned little is actually known. The uncertain role of histamine has already been discussed (page 239) and attempts to link emotional instability with vasomotor instability is not convincing (130). Slüder stated that it was due to chronic inflammatory change of the sphenopalatine ganglion or its main afferent nerve, the vidian, but his views have not been substantiated.

Dott (48) considers the cause to be a periodic relaxation and dilatation of the larger branches of the external carotid artery while Harris considers this entity is due to vaso-motor disturbances of the blood vessels of the dura, especially the middle meningeal artery. Wolff (1948) considers that this pain is due to dilatation of the third portion of the maxillary artery that supplies the area round the ganglion.

Anatomically, it is apparent that there may be combined activity of efferent autonomic and afferent sensory nerves - the latter covering the cutaneous field of the trigeminal and the upper cervical segments. The greater superficial petrosal nerve, being the efferent pathway of vasodilator and secretomotor fibres to the lacrimal gland as well as for the mucosa of the nose and upper pharynx seems to be the pathway for parasympathetic overactivity. The greater superficial petrosal nerve also contains sensory afferent fibres from the carotid artery, including its facial and orbital branches.

It seems likely to Gardner (64) and others that periodic discharges of parasympathetic impulses over one greater superficial petrosal nerve might account for unilateral lacrimation, swelling and secretion of the nasal mucosa, and pain in the head (171).

The possibility of sympathetic paresis has been considered
by some, rather than parasympathetic overactivity. That this is a possibility may be seen from the case report by Dodd (44) who observed very similar symptoms to Horner's syndrome following an aberrant block injection in the region of the internal carotid artery and plexus.

From the preceding paragraphs, it seems doubtful that the operation of sympathectomy, as practiced by Dandy (171), could hope to be successful. Even the more logical procedure of dividing the greater superficial petrosal nerve, as practiced by Gardner, provided a failure of 25% with 25% cures and 50% fair results only.

In order to study the problem further, White and Sweet (171) stimulated the greater, lesser and external superficial petrosal nerve and the middle meningeal artery in the course of operations for trigeminal and atypical facial neuralgia. (The external superficial petrosal is an autonomic twig connecting the middle meningeal artery with the geniculate ganglion of the facial nerve).

Stimulation of the greater superficial petrosal nerve causes pain localised in the eye, ear or adjoining parts of the head and face in a certain number of patients with Horton's neuralgia. In others, stimulation of the middle meningeal artery also reproduces the pain complained of. The pain is apparently not caused by vasodilator fibres sending impulses to the periphery which then indirectly sets up pain, because after cutting the greater superficial petrosal nerve, pain is evoked only upon stimulation of the central end.

This suggests that good results are related to interruption of afferent pathways for pain rather than of efferent fibres mediating vasodilatation.

The problem is still open. Gardner in 1957 states, concerning the value of petrosal neurectomy, that he is less and less inclined to resort to resection of the greater superficial petrosal nerve in treatment of headaches.

"Probably the most successful are the typical Horner's
type. However, pretty close to half of them do not obtain enough relief to justify the procedure. In addition to the immediate failures, we find that it is seldom possible to prevent regeneration of the nerve. We have had just enough undoubted successes to make me hesitate to abandon the procedure completely; we have had patients not relieved of unilateral headache by ligation of the middle meningeal artery in whom the subsequent resection of the greater superficial petrosal nerve gave lasting benefit.

White and Sweet (171) state "In patients deriving clear cut relief from petrosal neurectomy, we propose, upon recurrence of severe pain, and resumption of secretion of tears to investigate the advisability of division of the nervus intermedius.

Since the tympanic plexus, of glosso-pharyngeal origin, as well as the nervus intermedius via the geniculate ganglion both send fibres to the greater superficial petrosal nerve, we propose to stimulate the rootlets of both nerves in the posterior cranial fossa. Those which appear to transmit clinically significant pain will be cut.

While surgical procedures as described above, desensitisation with histamine diphosphate, and alcohol injection of the gasserian ganglion have all failed to produce invariable relief, conservative treatment is now aimed at preventing the vasodilatation and hence reducing some of the painful features of the syndrome by the use of vaso constricting agents.

Ergotamine tartrate (0.5 mg.) is now injected subcutaneously three times daily for six days. On the seventh day treatment is stopped and if the pain recurs, injections are resumed for a further six days (156, 130, 151). This form of treatment is effective for a time, but sooner or later the attacks seem to escape control and the ergotamine becomes ineffective.

Two other measures are being used with some success.

Isoprenaline - hydrochloride tablets sublingually in 20 mg. doses are used. These cut the attacks short, but do not prevent their recurrence. Again, spraying the area with ethyl chloride
for 20 or 30 seconds and freezing the skin, quite frequently stops the pain immediately.

(4) **Superficial Temporal Arteritis:**

This is a generalised vascular disease of elderly people and was so called because the lesion was mostly easily observed in this artery. Cohen (33) terms this disease 'giant celled arteritis'. In this disease subacute inflammation of the media leads to thrombosis (156) with much perivascular inflammation. The superficial arteries are palpable and tender and cause persistent severe pain in the temple and forehead associated with loss of appetite and weight, diffuse pain, fever and exhaustion. Visual disturbances may occur. Cohen reports a case where the internal maxillary artery was affected at an early stage causing severe pain in the upper jaw.
(B) THE PRIMARY NEURALGIAS:

The cranial nerves in which primary neuralgias occur are the trigeminal, glossopharyngeal, facial and superior laryngeal nerves. These nerves possess ganglia in which are found the bipolar cells of the first sensory path, and the frequency of occurrence of primary neuralgia in these nerves is directly proportional to the size of their ganglia (33). Cohen states that in his series, for every case of geniculate and superior laryngeal neuralgia, there were six of glossopharyngeal neuralgia and 135 of trigeminal neuralgia.

Primary neuralgias have similar characteristics no matter which nerve is involved. There are no objective signs of nerve involvement and spread to other nerves is extremely rare. Diagnosis, then, depends on the history.

(i) Tic Douloureux or Primary Idiopathic Trigeminal Neuralgia:

Tic Douloureux is one of the most characteristic and one of the most severe pains known to man. The condition affects women twice as often as men and is commonest in middle age (156).

The condition may begin quite suddenly in an otherwise healthy person or it may be ushered in over a period of years with mild manifestations.

Identification of the syndrome depends upon certain features which are pathognomonic: – (171, 23, 156, 151, 106, 157, 111).

(1) The pain is paroxysmal, lasting only seconds to a few minutes and is usually of extreme intensity. It is described as shocklike, stabbing or lightning. These flashes of pain may follow each other very rapidly for minutes on end. In the intervals between these violent experiences, there is usually no pain or at the most mild dull ache.

(2) These spasms are provoked by obvious stimuli to the face. Any touch, a draught of air or movement of the face may suffice to evoke a lancinating attack. Commonly found are 'trigger
zones' i.e. areas of special sensitivity which, when superficially stimulated, provoke an attack.

Such trigger areas may be anywhere in the territory of the fifth nerve, but are commonest in the skin of the cheek and the mucous membranes of the gums and throat. There may be more than one trigger area in one patient. Section of the nerve supply to these areas alone will stop all pain both local and referred.

Often there is a transitory period of immunity after a brief period of torture, during which time the patient can rapidly gulp down a few bites of food before the response to casual stimuli returns.

In other cases, respite from the paroxysmal tendency is only momentary and nutrition suffers due to the patient's fear of triggering off a further attack by eating.

(3) The pain is confined to the trigeminal zone, the pain being felt in the territory of one or two divisions of the fifth nerve, sometimes in all three. The commonest site is the lower jaw and the least common is the opthalmic division. Monica (ill) considers that the most frequently affected branch is the onfra-orbital, however. It is extremely unusual for pain to spread from one side to another or to other nerves, although Cohen states it as a rare possibility. Any spread of the pain to the ear, neck, throat or posterior part of the scalp or to the other side, should lead one to suspect some other diagnosis.

White and Sweet state that the pain is referred to only one side of the midline even in those who develop a bilateral trigeminal neuralgia.

(4) There is no hypaesthesia or hypalgesia upon routine testing with cotton wool or pin prick. A zone of partial sensory loss may indicate a tumour.

Certain other non diagnostic clinical features may be noticed. The face is often distorted with anguish, the eyes fixed, speech arrested and breath held. The term 'tic' is derived from the involuntary jerking of the angle of the mouth
or the eyelids which may coincide with each flash of pain. Lacrimation during the attack and reddening of the face is often noticed, together with stimulation of nasal secretion and salivation (151).

A symptom which helps distinguish trigeminal neuralgia affecting the mandibular nerve from other pains in the lower jaw is that pain in the tongue almost invariably accompanies the former.

The attacks rarely occur at night, since the trigger zones are less likely to be stimulated during sleep.

Paroxysms come in cycles, each lasting for several weeks or months.

Remissions may last for months, weeks or years, but eventually the pain always recurs. The occurrence of such remissions following therapy has been responsible for many groundless claims of therapeutic efficiency for a host of drugs and other measures.

Successive cycles tend to be worse and to come more often, and spontaneous recovery is rare.

It will be seen certain chronic lesions of the nervous system if appropriately placed, are capable of producing pain indistinguishable from that met with in idiopathic trigeminal neuralgia (page 273, 274). The 'tic' like pain may occur in the early stages of a progressive lesion which involves, invades or compresses the ganglion e.g. tumours or aneurysm, but this stage is transient and is soon followed by destruction of the ganglion cells with ensuing motor and sensory loss. Slowly progressive lesions such as tabes dorsalis, cranial hyperostosis or disseminated sclerosis may also be responsible for pain resembling Tic Douloureux.

In such cases, additional objective signs are present and differential diagnosis from true primary trigeminal neuralgia should be made.

Secondary neuralgia is usually manifested by pain which
is less severe and of longer duration, and is also accompanied with sensory changes.

Aetiology:

The cause and mechanism of this syndrome remains obscure but many theories have been advanced.

Harris (93) considers that the pain must be of peripheral origin, for if it were of central origin, neither peripheral nerve block nor neurectomy would give relief.

Levy and Grant in 1938, on the other hand, placed the lesion more centrally and suggested that lesions in the thalamus or the thalamo cortical tract are responsible for the pain and that major trigeminal neuralgia is a special form of a thalamic syndrome.

Wolff (1948) produced evidence that a paroxysmal ischaemia of the sensory root, gasserian ganglion, or its peripheral division may cause tic douloureux. This suggestion that angio-spasm is the basic of the trouble has appeared many times in the literature; other vascular changes, such as arterio sclerosis have been held responsible. It is known that pain may result from pressure on, or ischaemia of, sensory nerves and their ganglia. It is also established that the blood supply of the gasserian ganglion is relatively poor (179).

Peabody (etal) (118) feel that there is a definite relation between tic douloureux and defects in cranial circulation and cite McKenchie (1943) who reported the success of two cases where surgical procedures induced cranial vasodilatation. They also state that administration of vasodilator agents such as B-methylcholine chloride, amyl nitrate or nicotinic acid has reduced both the intensity and frequency of attacks of Tic Douloureux. In a very limited series using histamine, amyl nitrite, carbon dioxide and nicotinic acid, the authors found spontaneous and experimentally induced attacks of pain were diminished in intensity and duration by at least one of the vasodilatory agents used. They assumed that tic douloureux is the result of a paroxysmal ischaemia of trigeminal structures, peripheral
in site.

It is, however, difficult to envisage such a circumscribed vasoconstriction which affects the function of only the gasserian ganglion, sensory root and nerve — indeed, often discrete enough to produce pain limited to one division of the fifth nerve only.

Peabody postulated that afferent stimuli such as touch, pressure or cold arising from the trigger area evoke reflex vasoconstriction either widespread or local but involving the trigeminal structures. Such reflex vasoconstriction alone, or specially when superimposed upon structurely narrow vessels, results in a sudden and critical increase in ischaemia and pain.

Harrigan (81) subscribes to this view but feels that an associated generalised arterio sclerosis is often a pre-disposing cause.

Frazier in 1918 stated that multiple sclerosis might be the causative factor. Rowbotham (127) however, examined lengths of the trigeminal nerve and the ganglion histologically and failed to find definite lesions in such cases.

Dandy in 1934 contended that the disorder must affect the sensory root since he found tumour, basilar aneurysm or cavernous angioma adjoining the root in 10% and some affection of the root by an artery in 31% of 215 cases of trigeminal neuralgia treated by his subcerebellar approach. Because he was unable to find any report of tumour of a peripheral branch accompanied by tic douloureux, be considered compression of the sensory root to be peculiarly likely to produce this syndrome.

This is not generally accepted, for typical trigeminal neuralgias due to external compression of peripheral trigeminal branches have been reported (171). One such patient had carcinoma of the maxillary antrum.

Dandy's contention that the disorder affects the sensory root does not appear to be invariably the cause. Taarnhøj (155) has obtained relief of pain without loss of sensation in many cases by the operation of decompression and freeing the sheath
of the nerve root of the fifth nerve and the ganglion. This suggests that the ganglion, and/or the neurones proximal to the ganglion, are the pathologic site (106).

Cohen (33) considers that the mechanism of primary neuralgia is similar to that met with in epilepsy. Epilepsy, like neuralgias, may be 'symptomatic' associated with gross lesions of the brain - and 'idiopathic' in which no gross lesion is found. Whichever the group, the basic mechanism of epilepsy, as shown by electro-encephalographic investigation, is the same, namely, a sudden explosive irregular discharge of energy from the nerve cell.

Cohen reasons that a similar mechanism may account for primary neuralgia, the discharge occurring from the sensory root ganglion cells. He lists the evidence on which he bases this theory as follows:

1) The nature of the pain - sudden paroxysms of 'bright' pain.

2) The presence of 'trigger zones' from which a sensory impulse causes the 'explosion' of the unstable ganglion cell.

3) The presence of a refractory period, as shown by the fact that if a paroxysm of pain is induced by stimulation of a 'trigger zone', an interval of 2-3 minutes must elapse before a further paroxysm can be induced by stimulation of the same area.

4) Even though temporary benefit may follow any form of interruption of the peripheral nerve before it reaches the ganglion e.g. by alcohol injection, avulsion, or section, ultimately the pain always recurs; temporary benefit results from removing the trigger zone and long periods of relief may follow injection of a local anaesthetic.

5) Preganglionic root section cures the vast majority of cases, though it may be that if it is too long delayed, higher stations will become unstable by repeated bombardment from the lower cell stations and preganglionic root section may be ineffective.
6) There is recent evidence that primary neuralgias are benefited by anti convulsant drugs, but it is too early to assess their value.

Dorsey (47) recently reported that 5 out of 12 cases were relieved by using diphenylhydantoin.

While this theory provides a rational explanation of the mechanism of Tic Douloureux, the underlying cause of such nerve cell 'instability' is unknown.

Surgical methods for the relief of Idiopathic Trigeminal Neuralgia:

While the problem of relieving the extreme pain of primary neuraglia seems simple viz. the sectioning of the sensory root behind the ganglion, Grant (76) points out that the price paid for the relief of pain is sometimes higher than either the surgeon or the patient reckons on. Not only must surgical hazards be considered e.g. the mortality rate and the possibility of postoperative scarring, but the operative procedure, while relieving the pain, must not produce other discomfits in the face of a permanent nature which may result in as much distress to the patient as his former intermittent paroxysmal pain.

Little need be said about such temporary procedures as inhalation of trichlorethylene. This vapour seems to have a specific action of the sensory nerve endings of the trigeminal nerve but differing individuals show variations in susceptibility (157).

Surgical intervention is usually necessary.

(i) Alcohol Block:

Temporary denervation of the appropriate trigeminal area can be accomplished for up to 18 months by injection of alcohol into the 2nd or 3rd divisions of the trigeminal via the foramen rotundum or at the foramen ovale respectively. If the first division is involved, it is recommended that a peripheral neurotomy of the ophthalmic branches be undertaken.

There are three very real advantages in administering a preliminary alcohol block.
(1) If a successful injection relieves the pain and results in anaesthesia, than a permanent denervation will afford the same relief (76, 171). If alcohol block is unsuccessful, true tic douloureux is not present and a more central attack on the fibres in the posterior root will also fail.

(2) Following retrogasserian neurectomy (posterior trigeminal rhizotomy) there is a variable degree of constant unpleasant sensation in the face. Anaesthesia is permanent and Grant states that 25% of patients will complain about the stiffness, swollen feeling and apparent lack of mobility in the affected side of the face.

Still more serious is the possibility of paraesthesias developing. In about 5% of cases a persistent hot, burning or itching sensation develops in the area and is such a continual annoyance that the patient is as much distressed as previously due to the intermittent neuralgia.

By preliminary alcohol block, the patient is introduced to the feeling of facial anaesthesia and during the months prior to nerve regeneration, is able to decide whether permanent denervation will be acceptable.

Unfortunately, it has been found that in some cases 'anaesthesia dolorosa' may develop subsequently to rhizotomy even though the alcohol injections elicited few complaints.

(3) In patients weakened by inability to eat, injection of the nerve will permit normal food intake and decrease the operative risk. In those of advanced years, alcohol block may allow completion of the life span without operation.

It is apparent, however, that alcohol injection is a painful procedure and it often must be repeated to obtain a successful result. Patient co-operation is therefore lacking in some cases.

(ii) Alcohol injection or electro coagulation of the gasserian ganglion:

While peripheral nerve block with alcohol has been previously mentioned, it has been reported by Harris in 1912 and
Fig. 68. Approach to the Gasserian ganglion between maxilla and mandibular ramus for alcohol injection.

Fig. 69. Dandy's diagram to illustrate the danger of the injection of alcohol into the Gasserian ganglion. The alcohol may be "injected into the cerebrospinal fluid surrounding the ganglion and immediately passes to the base of the brain causing a dreadful series of paralyses of all the nerves on the side of the injection".
and Hartel in 1914 that destruction of the gasserian ganglion with alcohol prevented recurrence of pain for 3 or more years. The approach used by Hartel is via the foramen ovale, inserting the needle between the maxilla and ramus of the mandible (Fig. 68).

This procedure can be hazardous, however, for spread of the alcohol into the cerebrospinal fluid may occur with consequent cranial palsies (Fig. 69). Occasionally the carotid artery or cavernous sinus may be punctured. There is also a peculiar tendency for the alcohol to produce anaesthesia in the 1st division before affecting the 2nd. Severe ocular complications may occur which can only be prevented by coincident cervical sympathectomy (156). The motor branches of the 5th nerve are also blocked causing unilateral paralysis of the masticatory muscles, but regeration usually occurs. Complications such as chemical meningitis, vertigo and nystagmus, weakness of the ocular muscles, facial palsy and facial haematoma may be noted.

Instead of injecting alcohol, Kirschner in 1931 proposed electro-coagulation of the ganglion by means of a needle insulated except at its tip. Recurrence rate seems to be high, probably because only the central area of the ganglion is necrosed.

It is probably best that most patients with trigeminal neuralgia should be treated by retrogasserian rhizotomy but in patients not likely to stand this operation, the Kirschner procedure should be considered especially in those cases affecting the 3rd division (171).

(iii) Peripheral nerve sections and intra oral avulsions of branches leading to the trigger zone have been tried in the past, but, due to early recurrence, surgical procedures have tended to attack more and more central parts of the 5th nerve pathways. Hartley's technique of intracranial section of the peripheral branches was followed by Krause's extirpation of the gasserian ganglion.

(iv) Retrogasserian Rhizotomy in the middle fossa:

Division of a part or all of the posterior root of the trigeminal nerve has come to be a standard operation for producing
permanent anaesthesia in the zone of the trigeminal nerve.

In 1901 Spiller and Frazier introduced the operation of retrogasserian root section by an extradural subtemporal approach and many refinements of the original technique have been introduced. The integrity of the motor root can be assured by differential section, and subtotal section of the sensory root enables conservation of the ophthalmic fibres, maintaining corneal sensation and reducing the risk of Keratitis (157, 145, 171). (Fig. 70 A. & B.) One of the greatest disadvantages of this operation is the fact that there is complete loss of sensation within the area of the face innervated by the divided fibres.

Frazier in 1925 reported that the area of numbness could be reduced and paroxysms prevented if the fibres supplying only the trigger zones are divided. The means on ensuring such a differential division seems, however, to be rather empiric.

Various complications may occur following this operation. Facial paralysis occurs in about 5% of cases and lasts for about 3-6 months. (76). This is due to traction of the greater superficial petrosal nerves or haemorrhage into its sheath with consequent trauma to the geniculate ganglion and depression of the main motor fibres of the facial nerve in the facial canal (171).

An extradural haematoma can be a most dangerous complication if a branch of the middle meningeal artery breaks loose. This is signalled by the development of Jacksonian seizures or of hemiparesis during recovery from anaesthesia.

The principle complication of trigeminal rhizotomy which has led to efforts to improve this operation is seen in the small group of patients whose continuous paraesthesias in the anaesthetic area may be intolerable. This complication is not relieved by completing the partial rhizotomy, by removal of the inferior cervical or upper two thoracic sympathetic ganglia, by stripping of the carotid artery or by bulbar tractotomy.
Fig. 70 A. & B. Retrogasserian neurectomy, subtemporal approach

(H) exposure of the ganglion and its posterior root.
(I) opening of root sheath exposing sensory rootlets.
(J) lower two thirds of sensory rootlets elevated on fine hook prior to sectioning.

Upper rootlets of opthalmic division and motor root are preserved.
The only treatment for anaesthesia dolorosa seems to be frontal lobotomy.

Death due to septic meningitis, massive haemorrhage from the middle meningeal artery, postoperative bleeding requiring secondary craniotomy and temporal lobe epilepsy have all been observed. This operation is therefore far from safe and satisfactory (156).

(v) Decompensation of the trigeminal branches, ganglion or root:

Taarnhoj (155) believes that slight compression of the posterior rootlets may occur as they pass from the posterior to the middle fossa over the petrous bone.

He opened the dural sheath of the root from the back of the gasserian ganglion posteriorly over the petrosal ridge and reported successful relief from pain in 10 patients up to 8 months afterwards.

Gardner and Miklos (65) reported on two series of 100 patients in whom decompression of the sensory root had been undertaken using either the extradural approach of Frazier or Taarnhoj's intradural approach. 62% of patients gained complete relief; 11.5% partial relief and 26.5% were failures. They feel that the cause of trigeminal neuralgia is localised in the posterior root of the 5th nerve and gentle manipulation of the sensory root where it crosses the apex of the petrous temporal bone, in some way corrects the abnormality.

(vi) Trigeminal Rhizotomy in the posterior fossa:

In 1925 Dandy published his method of sectioning the trigeminal root at the pons after exposing it by a subcerebellar approach. The posterior-inferior half of the root is divided with a blunt hood after elevating the cerebellum. This portion of the root consists of the superior part of the tract and is formed by maxillo-mandibular fibres. If the 1st division is involved the upper half of the cephalic portion of the root is divided. The main advantages states are that the motor root is never divided, keratitis is avoided and the loss of sensation is small and may be entirely absent (145).
Fig. 71 & 72. Position of descending trigeminal and spinothalamic tracts in the upper (71) and middle portion to the medulla.

White & Sweet (171) suggest incising to the depth of the heavy stippling denoting the trigeminal tract in Fig. 72 for the medial zone of the nucleus consists mainly of relatively fine myelinated fibres running longitudinally, which may well be important pathways for pain.
This approach, however, is difficult and the postoperative course protracted. Again, recurrence rate is high.

The approach is a valuable one when it is necessary to investigate the cerebello pontine angle for neoplasm, but routine used for tic douloureux surgery is now avoided (171).

(vii) **Bulbar Trigeminal Tractotomy:**

The descending or spinal tract of the trigeminal nerve extends from the point of entry of the trigeminal rootlets in the upper pons down the full length of the medulla oblongata into the upper cervical cord (page 126) (Figs. 71, 72).

Sjoquist (145) was the first to demonstrate the incisions into this tract in the upper bulb may produce loss of pain sensation in the trigeminal area. Although there is analgesia, complete anaesthesia is not seen for touch sensation is preserved. This disagreeable paraesthesiae occasionally seen after trigeminal rhizotomy do not occur.

Weinberg and Grant in 1941 modified the Sjoquist technique in such a way as to avoid injury to the restiform body and thus reduced postoperative ipsilateral ataxia and other neurologic sequelae. Their incision was made 5 mm below instead of 9 mm above the obex at which point the tract is nearly at the surface of the bulb and the restiform body is above it (171) (Figs. 73, 74).

While trigeminal tractotomy is in general remarkably successful, there are disadvantages. It is a major operation for any but the comparatively young and healthy. The landmarks for the incision are not the best. Also, the part of the descending root carrying fibres of the 3rd division, lies most centrally in the medulla and when the mandibular area is involved in trigeminal pain, the medulla must be incised deeply to section the necessary fibres (76).

(viii) **Frontal Leucotomy:**

A final approach to the problem of unbearable suffering in cephalic pain is the elimination of introspection and agitation
Fig. 73. Sjoquist's medially incision above the obex.

Fig. 74. Grant & Weinberger's incision of the medulla for section of the descending trigeminal tract. Inset shows their incision below the obex (oblique black lines) with the more extensive incision favoured by White & Sweet (white line). White & Sweet prefer to make the incision at the cross sectional level of the obex rather than below it.
in the sufferer by bilateral frontal leucotomy. As stated on page 140 this operation seems to suppress awareness of suffering, concern over incurable illness and the reaction expression of pain (171).

While this appears to be a relatively simple operation, it is a very radical procedure. If incision is too posteriorly placed, the damage is appalling; if sufficient frontal cortex is not disconnected from the thalamus, relief from pain is uncertain. A major problem is the psychological deterioration which follows leucotomy.

Certain qualities of personality are modified and many patients subsequently become a burden to their families.

Only when section of afferent pain pathways has proved ineffectual and pain has become a morbid preoccupation causing total invalidism, is leucotomy justified.
Glossopharyngeal Neuralgia (37, 111, 171, 81, 33, 101)

This form of neuralgia is relatively rare having only 1/70th the frequency of major trigeminal neuralgia. It exhibits pain similar to that of trigeminal neuralgia i.e. paroxysmal, unilateral excruciating pain of short duration.

The attacks are often set off by eating, for there is in many cases a sharply defined trigger zone at the base of the tongue. In other cases, the trigger zone may line in the pharynx, tonsil or ear and paroxysms are initiated by swallowing, talking, sneezing, coughing, laughing, yawning or shouting. Many patients are therefore dehydrated, undernourished and cachectic.

Pain of glossopharyngeal neuralgia may be felt in any area supplied by the 9th nerve:— the pharynx, palatine tonsil, posterior 1/3rd of the tongue, posterior part of the tympanum, posterior wall of the auditory canal, part of the concha or on part of the postero medial surface of the auricle.

As in trigeminal neuralgia, a severe attack may confer a brief period of immunity lasting a few seconds or more. The duration of each burst of pain is usually less than one minute. Total remissions lasting months or years are common. Most patients are over 50 years of age.

In differential diagnosis, elimination of organic causes such as tumours, either intra or extracranial is important. Pain of identical type has been reported with acoustic neuroma of the posterior fossa, malignancy of the tonsillar area and neoplasm of the larynx.

In some cases the pain may be limited to the angle of the mandible and be confused with mandibular neuralgia or other painful disorders.

Glossopharyngeal neuralgia is said to be accompanied by pronounced increase of secretion from the parotid gland on the affected side both between and during attacks.

Cocainisation of the trigger point aids diagnosis.

In treating this condition, extracranial alcohol injection
Fig. 75. Sensory distribution of the glossopharyngeal nerve

Fig. 76. Glossopharyngeal neurectomy in the posterior fossa.
is impractical because of the close proximity to other vital structures of the 9th nerve at the base of the skull.

Section of the 9th nerve intracranially leads to little disability either motor or sensory. There may be some slight lowering of the palatal arch in view of the loss of function of the stylo pharyngeus, but this is not noticeable.

This operation of glossopharyngeal rhizotomy relieves the pain completely and there has been no case reported in which the anesthesia has been accompanied by unpleasant paraesthesias.

Reduction in secretion from the salivary glands seems to be small due to the integrity of the chorda tympania nerve, and loss of taste on the ipsilateral side is not the subject of complaint.

In some cases of glossopharyngeal neuralgia, cardiac arrest with syncope is noticed. It seems that excessive discharge over the sinus nerve - the afferent pathways of the carotid sinus reflex - as well as over the pain path may occur simultaneously.

The reflex affects vary from slowing of the pulse during short attacks of pain to hypotension and even asystole in longer attacks. In such cases, where carotid sinus epilepsy accompanies glossopharyngeal neuralgia, denervation of the carotid sinus may be necessary.

In some atypical cases, the vagus nerve may also be involved and pain is not fully relieved until the rostral rootlets of the vagus, in addition to the glossopharyngeal nerve are divided. It is known that cephalic pain fibres may travel centrally via numerous vagal rootlets.
(iii) **Geniculate Neuralgia** (31, 171, 33).

It has been shown that the facial nerve, while predominately motor, subserves taste via the chorda tympani nerve and also possesses afferent sensory fibres which travel via the geniculate ganglion.

Such pain fibres from the geniculate ganglion supply a portion of the ear and possibly deep structures of the face and the facial musculature.

Following on Ramsay Hunt's observations on geniculate herpes, Brodal et al (24) have verified that the cutaneous distribution of the nervus intermedius covers part of the external auditory canal and tympanum and part of the lateral and medial surface of the auricle.

The extent of the geniculate innervation is highly variable, however, in line with its vestigial character as a sensory remnant, on which the trigeminal and cervical cutaneous zones have encroached more and more (171). Further evidence that the nervus intermedius has sensory components as described above, lies in the hyperaesthesias which may appear immediately preceding facial palsy.

Atypical primary neuralgia of the nervus intermedius may occur as painful spasms deep in the ear. There is never any pain in the throat and neck nor is the principal pain in the ear ever produced by swallowing, talking or eating.

There is great difficulty however in distinguishing between involvement of the nervus intermedius, glossopharyngeal or vagus nerves.

Mackenzie in 1938 described a case where all three nerves were implicated in severe neuralgia.

White and Sweet state that unless pain deep in the ear can be shown by stimulation to be transmitted by the nervus intermedius, then additional section of the glossopharyngeal and upper vagal rootlets should be resorted to. (Fig. 77).
Fig. 65. Exposure of nervus intermedius.
(iv) **Superior Laryngeal Neuralgia** (171)

There are several instances in which neuralgic pains involve vagal as well as glossopharyngeal rootlets as proved by recurrence of pain after section of the 9th nerve and subsequent relief upon dividing the upper rootlets of the 10th nerve. There are some clear cut cases which show that the superior laryngeal nerve itself may be primarily involved in neuralgia.

Neuralgic pains in such cases involve the sensory fibres distributed to the epiglottis, base of the tongue, and superior portion of the larynx via the internal branch. The pain starts in the throat in the general region of the hyoid bone and upper part of the thyroid cartilage and may extend widely even to the zygoma, thorax or gums and ear. Swallowing, yawning or coughing may evocate the pain.

It is interesting to note that the superior laryngeal branch of the vagus is the nerve primarily concerned with pain in tuberculosis or carcinoma of the larynx and vocal cords. Alleviation of pain in these conditions has been found possible by injection of alcohol into the nerve where it pierces the hyo-throid membrane.
(v) Post Herpetic Trigeminal Neuralgia:

While Herpes is a self limiting disease, in some instances, particularly in patients over 50 years of age, the condition persists and becomes an intractable post herpetic neuralgia, which may last for months or indefinitely. In contrast to the acute disease the pain of post herpetic neuralgia is lancinating and brief, similar to tic douloureux, and elicited by sensory stimulation. There are, however, many diagnostic points. Between attacks of paroxysmal pain there is a constant, sustained burning pain and hyperaesthesia (111); it is quite often involves the ophthalmic division (171); there are often areas of hyperaesthesia and even anaesthesia of the skin and whitish scars on the skin may be present.

The mechanism of the pain is not clear, and is often incurable.

Head and Campbell in 1900 described the posterior root ganglia of the cranial and spinal nerves as the principle sites of damage in herpes zoster, but section of the posterior roots yielded little success. It seems that inflammatory changes must occur in the grey matter of the spinal cord or brain but the precise location between posterior root ganglion and thalamus where this occurs is not known.

Attempts at trigeminal tractotomy have been unsuccessful. In some, the paroxysmal pains were relieved but left the severe burning pain; in others the paroxysms were as severe and frequent as ever.

Cervico thoracic sympathectomy has also yielded poor results.

Total excision of the painful skin has been tried with few positive successes. It is unfortunate that in such patients, even a bilateral frontal leucotomy fails to give useful relief of pain.
(C) **THE SECONDARY NEURALGIAS:**

(i) **Intracranial causes of oral and facial pain:**

Pain may be experienced either from involvement of the free nerve endings in relation to the dura and intracranial vascular structures, or from involvement of the afferent nerves and their cerebral connections.

The trigeminal nerve is vulnerable to various conditions which affect it between the foramina in the base of the skull and the brain stem. All are rare but they may cause severe and mystifying pain (156).

Neuralgia in the distribution of one or more branches of the trigeminal nerve may be caused by inflammation, traction, pressure or displacement (106). Cerebello-pontine tumours, neoplasms of the cranial fossa, aneurysm of the internal carotid and basilar arteries, cavernous angioma or any other vascular anomalies which may create irritation of the sensory root, gasserian ganglion or the proximal portion of its main divisions, may produce pain in the face. The pain produced by these lesions may resemble Tic Douloureux, but usually it has a slow onset, is of longer duration, and between paroxysms, there is a sustained steady ache. Moreover it is often accompanied by partial or complete loss of sensory and/or motor function in the trigeminal territory (111).

Pain arising from intracerebral pathways is perceived in the territory of the peripheral distribution of the neurones involved, not at the side of the lesion. Destructive lesions rarely affect a single division of the trigeminal only and there is practically always evidence of involvement of adjacent structures.

Interruption of trigeminal afferent pathways may be recognised not only by sensory loss but also by absence of certain reflexes and these will be considered further (1). While involvement of individual divisions is described the picture is usually a composite one.
Involvement of the **ophthalmic** division causes sensory loss in the nerve distribution and absence of the corneal, conjunctival and sneeze reflexes (on stimulation of the upper nasal mucosa). A lower central lesion may involve the ophthalmic division alone and result in analgesia of the forehead and loss of the corneal reflex only.

Involvement of the **maxillary** division, besides causing sensory loss in the skin and mucous membranes also results in loss of the sneeze and palatal reflexes.

Involvement of the **mandibular** division usually produced sensory and motor disturbances. Isolated fifth nerve palsy is very uncommon. If the lingual nerve is involved, loss of taste on the homolateral side of the anterior 2/3rds of the tongue may be added to sensory loss of the skin and mucous membrane in the region of the nerve distribution.

Motor disturbances differ according to the site of the lesion. Unilateral paralysis may occur in lesions affecting the lower motor neurones. Mastication is little affected, chewing being done on the normal side but there is deviation of the jaw towards the paralysed side on depression and there may be atrophy of the muscles on the affected side.

If paralysis of the lower motor neurone type occurs bilaterally the lower jaw cannot be raised and atrophy of all the muscles of mastication may occur.

With supranuclear lesions, the motor root escapes, if the lesion is unilateral, for there is a bilateral innervation. In bilateral supranuclear lesions however, paralysis of the upper motor neurone type occurs i.e. muscle spasticity, increased reflex response but no atrophy.

Supranuclear lesions (disseminated sclerosis, syringobulbia) cause sensory changes of the face and limb on the opposite side. In nuclear lesions hemianaesthesia occurs on the same side (1).

(1)(a) **Intracranial Tumours:**

Headache is a common symptom in intracranial tumours. It may be generalised, frontal or occipital, ipsi- or contra lateral.
The localising value of a headache is limited but its value as an altering system is great (106). Other signs, as described above, should be looked for.

Thus, while pain perceived in the orbital or maxillary area may be locally or distantly caused, an absent corneal reflex and depressed sensation over the area would indicate a lesion of the fifth nerve and suggest an intracranial cause, for instance a tumour. Signs due to local spread of a tumour will depend upon the anatomic situation.

Expanding tumours in the cerebello-pontine angle may produce symptoms of trigeminal neuralgia alone or in conjunction with auditory, vestibular or cerebellar signs. An acoustic neuroma most commonly presents with impaired hearing and tinnitus. The deafness may be overlooked or ignored until other neighbouring cranial nerves are involved e.g. 5th, 6th, and 7th. A diminished corneal reflex may be the only sign of involvement of the trigeminal nerve and is a frequent sign. Pain is less frequent but does occur, usually in the distribution of the 1st and 2nd division. (106) The pain may be severe and of long duration; it is not abrupt in character, its onset and termination being gradual. There is early sensory loss of the skin and other signs of intracranial tumour may be present - headache, vomiting, nausea, and visual disturbances (1). If raised intracranial pressure occurs, brain stem compression may be obvious with weakness of the opposite limbs. (156).

A meningioma arising in the region of the trigeminal nerve in the posterior fossa could produce a presentation closely similar to acoustic neuroma, but meningiomas situated in the middle fossa more frequently result in trigeminal pain. Occasionally they arise in close relation to the gasserian ganglion and directly involve the ganglion with pain and sensory loss in part or whole of the distribution of the ipsilateral trigeminal nerve.

The pain is generally steady and severe but rarely may be paroxysmal, simulating Tic Douloureux, though the presence of objective sensory impairment excludes Tic.
Extension of the tumour beyond the petrous ridge may result in oculomotor and later facial palsy with possible involvement of the 8th nerve and compression of the brain stem.

When meningiomas arise in other situations e.g. the sphenoid ridge and middle fossa, trigeminal involvement is generally preceded by signs and symptoms attributable to involvement of other structures. Meningiomas arising from the clinoid portion results in unilateral visual loss from pressure on the optic nerve which, with further expansion may give rise to the Foster Kennedy syndrome with papilloedema on the contra lateral eye due to raised intracranial pressure. The oculo-motor nerves are compressed with resulting progressive diplopia and ipsilateral ophthalmoplegia. Pain and hypaesthesia later may occur in the distribution of the trigeminal nerve. A unilateral proptosis may appear as the first sign due to interference with orbital drainage as well as to hyperostosis of the sphenoidal ridge from tumour invasion (106).

Other tumours arising elsewhere, for instance from the naso pharynx, may spread via the foramina or by bone erosion.

Recent knowledge indicates that many tumours of the trigeminal ganglion described in the literature and originally thought to be primary are due to malignant nasopharyngeal tumours (151).

These may infiltrate and involve the ganglion or metastasise into the base of the skull and adjacent parts. Godtfredson (73) states that ophthalo-neurological symptoms occur in 34.8% of cases 4-5 months before diagnosis has been made. In many cases rhinological, otological or lympatic involvement is present as well. In 70% of these cases trigeminal neuralgia is the first symptom in the maxillary area. The clinical features of trigeminal involvement by these tumours have been described by Trotter (164) (see page 284). The pain is intense and unremitting, as distinct from the quick paroxysms of trigeminal tic and there is usually unilateral paraesthesia of the cutaneous area sometimes with paralysis of the homolateral muscles of mastication. Occasionally oculomotor palsies are seen.
(1)(b) **Intracranial Aneurysms:**

Vascular dilatation may cause compression symptoms if occurring adjacent to the trigeminal nerve or its branches.

Aneurysms arise predominantly from the arteries forming the Circle of Willis. Symptomatology is diverse, the pain resulting essentially from either rupture and haemorrhage into the subarachnoid space or from dilatation and compression of adjacent nerve fibres or nerve trunks.

Aneurysms arising from the internal carotid are occasionally the cause of facial pain. Internal carotid aneurysms, if of slow development, exert compressive effects on the first division of the fifth nerve or possibly the first and second divisions. The pain is very severe and continuous and numbness may supervene. Diplopia and ophalmoplegia may follow from compression of the oculomotor and other motor nerves of the eye (151, 106).

(i) (c) **Intracranial Thrombosis:**

Thrombosis of the internal carotid does not usually result in pain or headache, but occasionally the onset is with severe pain in the face, forehead and temple. Neurologic function in the territory of the major branches of the carotid may be impaired and allow diagnosis. Carotid occlusion may, however, be silent as it is in the great majority of surgical ligations (106).

Harris (83) reports that thrombosis of the posterior, inferior cerebellar artery is a rare cause of intractable unilateral facial neuralgia. Sclerosis occurs in the medulla involving the descending spinal root of the fifth nerve and the ascending (crossed) spino-thalamic tract and results in hypaesthesia of the face on the side of the lesion and on the opposite side of the body below the face.

(i) (d) **Neuralgia secondary to infection:**

Inflammatory processes of the soft tissues or bones surrounding one or more branches of the trigeminal nerve, the gasserian ganglion or a sensory root may cause a neuritis by
extension of inflammation to the nerve. The consequent neuropathy is usually accompanied by constant pain with paroxysmal - like lancinating pain at irregular intervals (Ill).

In the middle ear or mastoid infection the inflammatory process may spread to involve the petrous temporal bone with erosion, so producing Gradenigo's Syndrome (106, 156). This becomes apparent as trigeminal pain with sensory loss and there is paralysis of the external rectus muscle (abducent palsy).

When an intracranial inflammatory lesion is localised as in the above case, then the pain and accompanying symptoms indicates such a local lesion. Pain from meningeal inflammation is more generalised and is of little localising value.

(i) (e) **Herpetic Neuralgia:** (104, 94, 11)

Herpes Facialis is due to a virus, Herpes Zoster, which may infect any sensory ganglia, the commonest involved being the gasserian. Pain is almost invariably present preceding or during the herpetic eruption.

It most often attacks the ophthalmic nerve and involvement of other divisions is rare. Cases affecting the mental nerve have been reported, however.

The pain of herpes may be severe and is described as burning, aching, continuous and non throbbing, often associated with cutaneous hyperalgesia dna paraesthesia. Conjunctival congestion on the affected side is one of the earliest and most constant signs.

Pain usually occurs before any other manifestation of the disease but vesicular eruption in the distribution of the involved nerve appears subsequently and makes diagnosis certain. The cornea may also be involved by vesicular eruption and keratitis and iridocyclitis may complicate. The cutaneous manifestations of this malady may be due to primary disturbances in the ganglion with secondary reddening, wealing and blistering of the skin due to centrifugal impulses releasing substances and setting up an erythralgic state in the skin (100). Some believe that the virus spreads along the nerve channels and directly provokes an
inflammatory reaction in the skin. Lewis points out that identical skin lesions sometimes follow injury to a posterior root ganglion from other sources and believes that the theory of antidromic centrifugal impulses is the correct one.

The Ramsay Hunt Syndrome is an uncommon form of Geniculate Zoster in which a vesicular eruption appears on the fauces, uvula and tongue. Lingual, facial and auditory paralysis may occur, with pain in the ear and deep structures of the face.

Rarely pain may persist for months or years, giving a rise to the syndrome of post herpetic neuralgia.
(ii) **Intracerebral causes of facial pain:**

Face pain occurs much less frequently from pathologic lesions within the brain substance, but some entities do cause painful symptoms. It will be remembered that pain and temperature fibres only are present in the spinal root of the trigeminal nerve, and brain stem lesions may produce dissociated phenomena such as loss of pin prick and temperature sensation, with preservation of light touch in the face (156).

Thalamic lesions, appropriately placed injury in the cord, bulb, pons, mid brain or cerebral hemisphere may give rise to pain presenting the following characteristics (171, 1).

1) Spontaneous constant character which may have an aching, gnawing, burning, boring, icy, or extremely unpleasant quality.

2) Superimposed there may be spontaneous paroxysms or these may be evoked by external stimuli.

3) Pain is often poorly localised and the reference of pain is usually over a wide area which may or may not include the spot stimulated.

4) Touch, heat, cold, pinprick may all evoke the same diffuse peculiarly disagreeable sensation which is more unpleasant than pain evoked from the normal side.

5) There may be exaggerated responses to mild stimuli; in other cases there may be an abnormally long period between the onset of effective stimulus and the sensation which may then long outlast the stimulus.

(ii)(a) **Multiple Sclerosis:**

Not infrequently the presenting symptoms of disseminated sclerosis is facial pain which has a close similarity to Tic Douloureux. Clinically there is little difference between this demyelinating disorder and Tic except that pain and paraesthesia may exist between attacks of flashing pain. The pain may also extend outside the trigeminal area on occasions. When a person under the age of 45 complains of flashing pain in the face with intercurrent burning sensation in the area, the diagnosis of
disseminated sclerosis must be considered (69).

As Multiple sclerosis occurs only within the white matter of the brain and spinal cord, the causative lesion is most probably in the region of the intrapontine fibres of the nucleus of the fifth nerve (106).

Harris (83) states that 4% of his cases of trigeminal neuralgia have the complication of disseminated sclerosis. Usually spinal symptoms occur before facial neuralgia but occasionally neuralgic tic may precede the symptoms of the disease by many years. Bilateral trigeminal tic is rare, but it occasionally occurs in disseminated sclerosis. Harris suggests that the scattered patches of sclerosis are apt to irritate the spinal trigeminal roots on both sides.

(ii)(b) **Brain Stem Tumours:**

As tumours of the brain stem spread, they cause defects of function in the cranial nerve nuclei and the long tracts. The fifth nerve nuceill may be involved with paraesthesias and diminished facial sensation, but pain in the trigeminus is rare.

Cerebellar tumours cause headaches frequently, the headache being located over the eye and forehead or in the occipital or suboccipital area. These headaches result from raised intracranial pressure as these tumours obstruct the fourth ventricle early.

(ii)(c) **Syringobulbia:**

Syringobulbia is a very chronic and slowly progressive cyst formation in the brain, - gliosis and cavitation occurring in the brain stem and often spreading down to the cervical spine. Because the descending root of the trigeminal nerve is invariably involved, loss of pain and temperature sensibility and absence of the corneal reflex occurs. Severe pain in the distribution of the trigeminus may appear early or at any time in the course of the disease (106). It may cause unvarying, dull, boring facial pain lasting for years (156).

Paralysis of the 9th, 10th and 11th and 12th nerves is common.
White and Sweet (171) instance a patient reported by Foix in 1922. The patient had continuous burning pain at both sides of the face, worse on the right. Paroxysms of more severe pain supervened whenever a current of air or cold water touched the face. In this case there was no objective sensory loss. At postmortem, two small cavities occupied nearly symmetrical areas within the nuclei of the descending trigeminal tract in the medulla. The much larger one was on the right side.

The diagnosis of such lesions constitute a real problem.

(ii)(d) **Thrombosis and Brainstem vascular lesions:**

Congenital vascular malformation in the brain stem are infrequent, but Macrae (106) instances an angioma which resulted in bilateral facial paraesthesia followed by bilateral face pain, headache and vertigo.

Defective blood supply to the brain stem from disease of the basilar or posterior inferior cerebellar arteries may produce symptoms from insufficiency (156). The descending root of the trigeminal nerve is supplied by the posterior inferior cerebellar artery whereas the basilar artery supplies the other nuclei and the spinothalamic and quintothalamic fibre tracts. Thrombosis of the former artery presents with vertigo, analgesia and thermalgiesia over the same side of the face and the opposite side of the body. Homolateral Horner's syndrome may be seen and paralysis of the 9th and 10th cranial nerves may occur.

(ii)(e) **Thalamic Pain:**

With a partial lesion of the thalamus or its afferent pain carrying fibres, (usually a vascular basis) spontaneous pain and painful dysesthesia may appear on the opposite side. This pain may be intractable and intolerable necessitating prefrontal lobotomy.

(ii)(f) **Dural Pain:**

Displacement of the dura by tumour growth is capable of causing pain and headache. Thus a malignant glioma or metastatic carcinoma in one hemisphere may, apart from headache from raised
pressure, result in local face and head pain caused by traction of pain sensitive dura (106).

The treatment of central pain is still in an evolutionary stage. Surgical treatment aimed at the primary afferent neurone or post central cerebral cortex, the mesencephalon, the thalamus or the frontal lobes have all been attempted with varying degrees of success. There is a problem in all such cases that surgical lesions may create additional similar symptoms and several such complications have been recorded following bulbo trigeminal tractotomy and upper cervical antro lateral cordotomy (171).
(C) (iii) **Extracranial Causes of Oro-facial pain:**

Extracranial causes of pain along the distribution of the trigeminal nerve are very numerous, for the neuropathy may be caused by inflammation, tumour, vascular lesions or skeletal trauma.

These secondary types of neuralgias are characterised by constant aching or burning pain which may be accompanied by secondary sensory disturbances in the form of hypaesthesia, hyperalgnesia or diminution of pain sensation. Some of the more important entities will now be discussed.

1) **Trotter's Syndrome** (33, 157)

Trotter in 1911 (164) described the clinical picture produced by lymphoepithelioma of the nasopharynx. This retropharyngeal tumour starts high in the fossa of Rosenmüller and grows beneath the mucosa extending along the base of the skull. The maxillary branch of the fifth nerve is involved at an early stage and eventually all the divisions may become effected, as well as the 9th and 10th cranial nerves and upper cervical roots.

Neuralgic pain may be felt in the lower jaw, the side of the head, tongue and ear with defective mobility of the soft palate on the same side. Due to involvement of the eustachian tube, deafness may eventuate.

Tumour growth is insidious and errors of diagnosis commonly prevent early location.

2) **Peripheral neuralgias following trauma and amputation:**

The common injuries which lead to persistent pain are amputations and partial or complete wounds of the peripheral nerve trunks. Faulty regeneration of the injured nerves, sepsis and resultant formation of scar tissue, appear to constitute the underlying factors which give rise to painful stimuli (171).

As previously discussed (page 158) it is noticed that in regenerating or amputated nerves, the threshold for burning pain is depressed, so that ordinary innocuous stimuli are perceived as painful, whereas the threshold for pricking pain is
elevated. In such cases, the response, when it comes, is of increased intensity. It is unusually unpleasant, diffuse and tends to be referred to remote parts (100).

The symptoms vary in intensity - at times being only a source of mild annoyance when skin is touched, at other times a cause of total incapacity (171).

Head in 1905 originally believed that the recovering area was one displaying only 'protopathic' sensation (page 144). He believed that the eventual return of the 'epicritic' sensation modifies 'protopathic' responses by a process of central inhibition and each response becomes localised.

Trotter and Davies found this explanation was unacceptable and suggested that pain in partial regeneration is due to a lack of insulating myelin. They considered that intensification of sensation in recovering areas is due to the regenerating fibres being subject to chronic irritation consequent upon contact of the new nervous tissue with surrounding non nervous tissues. This would imply that these fibres spontaneously discharge impulses while regenerating.

A theory perhaps more tenable than the above was proposed by Weddell and Sinclair on the basis of incomplete regeneration of the cutaneous sensory network for pain alone. The terminal network of sensory fibres in normal skin overlap in a most complex manner (page 146) so that ordinarily a painful stimulus affects a number of adjacent terminals simultaneously. If a single pain fibre is stimulated, however, it gives rise to a painful sensation of a particularly disagreeable and intolerable character.

While the exact physiological explanation of this dysaesthesia remains uncertain, it is probably related to the particular pattern of excitation aroused in the central nervous system by the arrival of a single instead of multiple sensory impulses from the sale sensory spot.

There may be a temporal factor involved, for it is well known that if impulses from the same sensory spot arrive at the
central nervous system at different times, the activity induced by the first impulse modifies and may itself be modified by the effects of oncoming volleys.

A likely contributory factor to the production of pain is anoxia either from local scarring or from widespread vasoconstriction. It has been shown by Lorente de Nó that a nerve rendered anoxic fires off repetitive stimuli, which suggests that impaired circulation may be a fundamental source of painful stimulation.

Lewis postulated that there is a centrifugal discharge from the point of nerve injury which liberates hypothetical metabolic substances in the tissues giving rise to pain as well as to cutaneous changes (page 152).

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Nerve amputation may result in the formation of neuromata which are generally painless. Occasionally, however, they are the causes of ill defined disagreeable sensation over wide areas and cause exquisite local tenderness (171). Although the local tenderness may be relieved by resection of the neuroma, various forms of pain may persist - burning dysesthesia, deep aching sensations with central radiation and at times lancinating pain.

Marsland and Fox (107) investigated the possibility that painful amputation neuromata might be the cause of sensitivity in edentulous areas following extraction.

Bradlaw in 1936 found evidence of regeneration of severed apical nerves after extraction and stated that it is known that a response to a stimuli is given by regenerated nerve fibrils with abnormal ease. The sensation thus elicited is unusual in character and spreads widely and he suggested that this might explain neuralgia following multiple extractions.

There are many cases of patients who are unable to tolerate dentures because of constant pain and discomfort along the alveolar ridge, and in such cases the pain may be of a severe and persistent neuralgic type.

Fox and Marsland examined several such patients and were
able to locate trigger spots, the mere touching of which produced quite severe paroxysms of pain.

The hypersensitive spots commonly measured about 3 mm in diameter.

Examinations of sections passing through such trigger spots revealed that a greater amount of nerve tissue was present than in normal mucosa.

In the connective tissue below the papillae localised groups of irregularly arranged nerve bundles were present in a complex, intertwining arrangement and large irregular nerve bundles were seen in close proximity to the rough bone.

Within the papillae of the connective tissue many nerve endings were seen situated much closer to the surface of the epithelium. Again, in such trigger spots, there was a remarkable increase in the number of ultraterminal fibrile in the epithelium.

Such aberrant nerve endings, situated as they are so close to the surface are extremely susceptible to injury and it does seem that in some instances, nerve fibres severed during extraction, can form painful amputation neuromata.

3) **Causalgia**: (151, 100, 171, 157).

Causalgic syndromes are most often seen after partial injuries of the limbs where the nerve is bruised or partly cut through. Although it is relatively rare for causalgia to occur following dental extractions, cases have been reported. It may occur occasionally following operations such as mandibular osteotomy or facio maxillary injuries.

While first discussed by Paget in 1864, Mitchell in 1872 introduced the term to describe the characteristic burning pain which may develop in the territory of the nerve some weeks after the original injury and after apparently normal healing. The term is derived from the greek 'Kausos' (burning) and 'Algos' (pain).

The peculiar burning pain is accompanied by cutaneous hyperalgesia of the corresponding territory and extreme tenderness
and pain is elicited by the slightest contact as well as by application of heat or cold.

The pain and dysaesthesia are frequently not restricted to the area of sensory defect.

It has been noticed that the skin of the tender parts may after a time assume a deeper blood colouration, and the skin becomes smooth and glossy, devoid of wrinkles and wet with sweat.

Lewis states that the pain and tenderness may be regarded as arising directly out of changes taking place at the site of injury. Stimuli which normally awake no pain may, on reaching the damaged area summate with impulses discharging spontaneously from the region of the injury.

The vasodilatation is usually ascribed to the antidromic vasodilator action of the posterior root fibres, this vasodilatation being accompanied by itching and also by burning pain. Some similarity to the erythralgic state (page 157) is noticed here.

Lewis feels that there is much evidence to support the idea that all symptoms of causalgia arise at the periphery in response to changes caused there by centrifugal impulses. For instance pain is always burning, in character with its origin from the skin itself. If the pain arose directly from irritation of sensory fibres in the main nerve trunk, it might be anticipated that pain of the deep variety would also be felt from time to time. The pain impulses seemingly ascend adjoining pain fibres, the territories of which overlay that of the affected nerve.

Elfenbaum (51) and Wright (182) consider that the causalgic state may be due to a continuing state of excitation within the neuraxis. This theory postulates that reverberatory circuits reexcite intracranial internuncial neurones, forgetting to discontinue their stimulation until a whirlpool is created. It is certainly true that blocking the peripheral nerves or removing the causalgic trigger point does not alleviate the symptoms in many cases.

A further theory states that the pain is due to the
'squeezing' and pinching of a nerve caught in the cicatrix, a possibility which correlates with the observation that pain is not evidenced immediately following the traumatisation. Healing of the tissue and pressure on the injured nerves is said to excite the continuous afferent stimulation that begins the creation of the vicious cycle.

Pain in this condition is closely associated with emotional stress as well as being elicited by thermal and tactile stimuli and events which excite an emotional reaction often produce paroxysmal exacerbations (151, 35).

The possibility that there may be some local abnormality of the sympathetic system is feasible when we consider the relief gained by sympathectomy.

Leriche reported that the pain and tenderness of causalgia may be abolished by sympathectomy and Bingham (16) in 1947 described two cases of causalgia of the face successfully treated in this way.

Stones (151) however; assesses that excitation of the lower half of the superior cervical ganglion is successful in only 40% of cases. A procaine block of the stellate ganglion should be initially tried.

The reason for the relief gained by sympathectomy is not clear. Stones considers that such an operation interrupts sensory afferents travelling by this route. Lewis (100) on the other hand, found that sensation in the area remains normal and considers that relief must be brought about by the persistently increased blood flow through the cutaneous vessels consequent upon the loss of vasomotor tone.

White and Sweet (171) quote Daupe, Callam and Chance (1944) who ascribe the peculiar qualities of causalgic pain to direct cross stimulation of sensory fibres by afferent sympathetic impulses at the point where the nerve trunk is injured. This theory deserves serious consideration as in certain stages there may be actual vasodilatation. It furnishes an explanation of many things, e.g. the increase of pain which so characteristically
takes place in a very hot or cold environment; the peculiar exacerbations during any form of emotional excitement and in extreme cases during every day visual stimuli; the relief gained by sympathectomy.

In corroboration of this theory of sympathetic activation of sensory fibres, it has been shown that in certain circumstances, efferent nerve impulses can alter the excitability of adjacent sensory axons. Recordings have been gained with a cathode ray oscilloscope of an afferent discharge from a sensory root when the motor roots are stimulated. In the absence of an injury to the peripheral nerve, no such returning discharge is observed. Katz and Schmitt, who undertook these investigations concluded that the small poorly myelinated pain axons of the 'c' group were especially susceptible to fibre interaction, and point out that this is a simple explanation for some of the symptoms of causalgia.

It is also a fact that causalgia is reduced in a quiet stable environment and during sleep when hypothalamic discharge is greatly diminished.

It has often been said that the condition is most likely to result after nerve wounds in which there is associated sepsis and arterial injury. Indeed, most of the rare cases of facial causalgia appear to be associated with injury to the inferior dental artery as well as the nerve.

Behrman (12) encountered twelve cases of dental causalgia following extraction of teeth. It was usually a difficult extraction of a multirooted tooth which was followed by severe pain.

Elfenbaum (51) reports on 30 cases of persistent post-extraction pain. This pain is not of a burning character as is seen in true causalgia of the skin, but resembles pulpitis and is of an aching quality. Symonds (154) reasons that this is to be expected, for deep somatic nerves in these cases are injured, not cutaneous, and the quality must be different.

Symonds and Elfenbaum state that dental 'causalgia'
exhibits certain characteristics which identify the state with 
true causalgia, viz. onset after 2-3 weeks, susceptibility to 
emotional fluctuation and failure of surgical procedures to 
alleviate the pain.

4) **Neuralgia secondary to trauma:**

Mechanical trauma resulting from fractures, lacerations 
or callus formations may cause sufficient pressure in one of the 
branches of the trigeminal nerve to produce neuralgia in its 
distribution (111).

An uncommon form of cephalic neuralgia may be secondary 
to injury of the upper cervical nerves. White and Sweet (171) 
state that the second cervical nerve is peculiarly subject to 
injury if the head is abruptly rotated. Such patients tend to 
have more or less constant discomfort in the suboccipital region 
on the side involved. Superimposed on this are attacks of pain 
which begin in this region and radiate to the vertex, temple 
or orbit, less often to the ear or entire head. Examination 
reveals tenderness over the point of emergency of the C2 root 
and the course of the greater occipital nerve. Interruption of 
the nerve is usually necessary for relief.

(5) **Frey's Auriculo Temporal Syndrome:**

This syndrome most frequently arises after infection of the 
parotid gland, although Thoma (157) reports a case which developed 
following a preauricular operation on the temporo mandibular 
joint.

The characteristics of this syndrome include paroxysms 
of burning pain in the ipsilateral temple associated with 
flushings and sweating when the patient eats (1, 33). In some 
cases there is a persistent hyperaesthesia in the area.

While section of the sympathetic pathways may cure the 
hyperhydrosis, avulsion of the auriculo temporal nerve is usually 
necessary when there are sensory disturbances.
(D) PSYCHOGENIC PAIN:

Many neurologists, in their experience with psycho-neurotic patients, are inclined to think that it is rarely true for a patient to imagine his pain. Some pain, however slight, is almost always at the basis of the reaction (171). An extremely nervous patient causes pain to become so dominant a factor in his own mind and actions, that a grave mental upset may be the end result (81). It may be neurotically exploited by exaggeration and introspection, so that the incapacity it causes is out of all proportion to the original pain.

White and Sweet state that "most normal persons experience in the course of a day various aches and pains in muscles, joints, viscera, head etc., but these are disregarded because they are leading active and satisfying lives; but let any person find himself in a psychological quandary with anxiety, doubts and depression, then the pain may develop larger and larger and occupy more attention and energy".

The term 'atypical facial neuralgia' has been often used to describe pain which does not follow the anatomical distribution of the trigeminal nerve, and does not have the paroxysmal characteristics of tic douloureux.

Many investigations have been carried out in such patients to eliminate a possible organic basis. Rushton et al (128) recently completed such a survey and found that a large proportion of so called atypical neuralgias actually had an organic basis viz. vasodilatation, dental disease, neuritis, neoplasms or causalgia following extraction.

There were a group of patients, however, whose complaints were definitely psychogenic. There were often associated neuroses such as depressive reactions, hysteria or schizophrenia.

The victims of this type of pain are usually women over the age of 40 and are commonly menopausal (33). The pain, which they describe in picturesque and extravagant terms, is never of the 'tic' type, but is more constant, dull and boring, perhaps lasting for hours and varying in intensity. The position of
the pain shifts from time to time and does not conform to the
distribution of a particular nerve (151). It may spread not
only to the other side of the face but to the neck and mastoid
region and even to other areas of the body and 'trigger zones'
which are tender to palpation, are innumerable. The patient
often states he is totally incapacitated and on further question-
ing often reveals a long history of professional visitations to
doctors and dentists.

Engle (1951) believes that the pain is a hysterical con-
version symptom. In such cases, on careful investigation, there
are usually elicited a host of neurotic reactions and symptoms
woven into a complex and disturbed social history.

Cohen (33) states that diagnosis of such cases, in which
pain has no organic basis or where there is gross over reaction
to very minor pathology, is most important, for surgical
procedures, in such cases not only fail to relieve the pain,
but invariably, aggravate it. He further states: "Sometimes
there are severe emotional problems which antedate the onset
of some minor dental operation, which is the firing mechanism
for a morbid obsessive fixation...."

"The (Psychogenic) label must not be the last refuge of
the diagnostically destitute. Every effort must be made to
exclude an organic basis and the diagnosis of psychogenic
'atypical' neuralgia accepted only if there are positive signs
of mental instability."

Simpson Hall(144) divides psychogenic neuralgias and
headaches into two groups. To one group belong the pure psycho-
paths in whom no organic disease of any kind can be found, nor
any derangement of physiology. The mental background of these
patients is not normal and psychiatric treatment is the only
means which will be of value.

The other group, he terms psycho-somatic. These people
have perfectly normal mental backgrounds, but such conditions
as frustrations and conflicts may cause an imbalance of the
autonomic nervous system to such an extent that: a definite
physiological derangement is produced, becoming clinically apparent as pain.

Many such patients are lonely and psychologically impoverished people who crave interest and attention or who have developed anxiety neuroses e.g. cancerophobia. With care and understanding - and the sacrifice of a little time at regular intervals - such people may be helped enormously merely by reassurance and sympathetic reception.


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