CHAPTER 5

THE CENTRAL ANATOMY OF FACIAL PAIN

It has been shown that peripheral pain receptor mechanisms are composed of free nerve endings and that noxious impulses are conveyed centrally in afferent fibres which are either unmyelinated or finely myelinated. The fibres conducting painful impulses have been grouped according to the speed of impulse conduction - small and larger calibre fibres conducting slow and fast pain respectively. (see Chapter 2).

Most pain sensory fibres are small and myelinated with diameters mainly from about 2∠5 u conducting impulses at velocities up to 40 metres/sec. (delta A group). A smaller proportion consists of unmyelinated fibres less than 2 u in diameter. In the mandibular nerve these comprise approximately 5% - 7% (184).

The main pathway for these afferent fibres from the face is via divisions of the trigeminal nerve, but some pain fibres run in company with visceral efferent fibres for varying distances on their way to the central nervous system. It has also been noted that some pain fibres travel centrally in cranial nerves other than the fifth. For instance, fibres derived from perivascular plexuses in the face travel as small myelinated fibres in the facial nerve and other pain afferents travel via the 9th, and 10th cranial and 2nd and 3rd cervical (184, 145, 106). It will be noted that the pain fibres, from these differing sources, terminate in close proximity to the main spinal nucleus of the 5th nerve and make connections with it.

From the time that a stimulus initiates impulses to the time that a painful sensation reaches consciousness, at least three neurones are necessary (81):-

1) The receptor neurone, which is the primary neurone

2) The connector neurone which is the neurone of the 2nd order, through which the impulse is conducted by way of special tracts within the spinal cord and brain.

3) The central neurone which is present in the receiving stations where perception or reality is registered. Pain afferents terminate in the higher nerve centres or cortex (35).

This tends to oversimplify the problem for many intervening neurones may be present along the central pathway.

The sensory fibres of the trigeminal nerve arise from the bipolar neurones of the gasserian ganglion, (except those conveying propriception and deep pressure which travel directly to their cells of origin in the mesencephalic nucleus of the 5th nerve (page 130)). Neurone I for the pain fibres in the trigeminal nerve is thus contained in this ganglion which is outside the central nervous system and corresponds, or is homologous to, the dorsal root ganglia on the spinal nerves (81).

Sjoquist (145) applied the techniques of Haggquist in carrying out a fibre analysis of the trigeminal root. By this technique of differential nerve staining (Alzheimer Mann) a systematic measurement of nerve fibre diameters is possible from photographed sections of the central nervous system or peripheral nerves. He calculated that the average number of sensory root fibres totalled 140,000. The fibres less than 4 u to which the group of fibres conducting pain and temperature belong, he found to be more numerous in the upper portion of the root. This portion to some degree holds its individuality and position throughout the entire length of the root. There are, however, a considerable number of pain afferents randomly scattered amongst larger diameter fibres. Wyke (184) assesses the number of pain afferents in the sensory root of the 5th nerve as one-third of the total.

From the cell body in the trigeminal ganglion by way of its axon, the impulse is transmitted to the nucleus of the spinal tract of the 5th nerve in the central nervous system. The sensory root is seen passing over the apex of the petrous temporal bone from the middle cranial fossa to the pons. The 5th nerve is the only cranial nerve entering the pons and is attached to the superolateral aspect of its ventral surface (95).

The pathway taken by sensory afferents after entering the pons varies. The differing modalities of touch, pressure, pain and temperature, while grouped together in the peripheral divisions of the trigeminal, become physiologically separated into specific tracts when they reach the central nervous system (147).

After entering the pons, the fibres of the sensory root of the 5th nerve pass to one or other of the following situations:-

(i) The spinal tract and nucleus of the 5th nerve.

(ii) The principal sensory (pontine) nucleus of the 5th nerve.

(iii) The mesencephalic nucleus of the 5th nerve which is in close connection with the 5th nerve motor nucleus.

The main bulk of fibres divide into ascending and descending divisions. The ascending fibres are short and almost immediately enter the main sensory nucleus and end within it. The descending fibres turn shaprly downwards and form the spinal tract of the 5th nerve.

The main sensory nucleus and the spinal tract resemble a tadpole shaped structure, the swollen body of which is situated in the pons (i.e. the main sensory nucleus) and the tail forming a long column of grey matter which is directly continuous with the substantia gelatinosa of the spinal cord.

More specifically the spinal tract of the 5th nerve is situated near the postero lateral surface of the medulla (95). Its upper end is situated not far from the upper and lateral edge of the 4th ventricle at the level of the entrance of the sensory 5th nerve root, and it descends as far down as the 3rd cervical segment. As it passes downwards, it is covered by the inferior cerebellar peduncle (the restiform body) and then, at a level corresponding to the inferior third of the inferior olive, it lies close to the lateral surface of the medulla. (This is of importance when considering the operation of trigeminal tractotomy).

During its descent in the pons, the vestibular nerve is

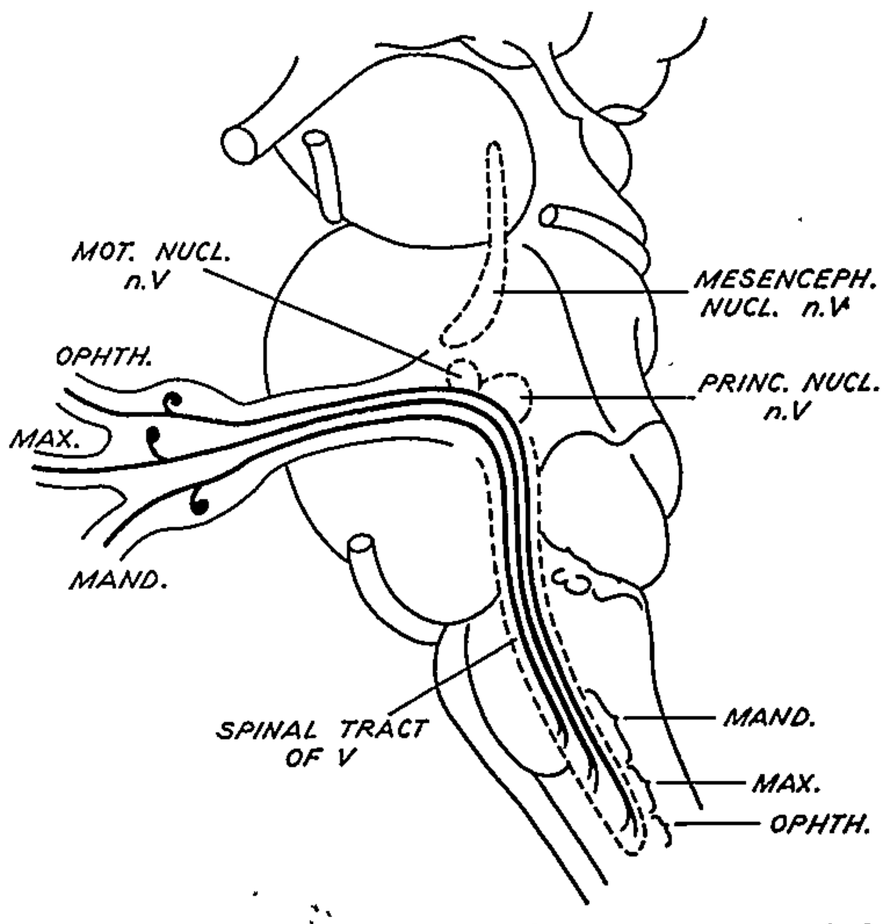

Fig. 10. Diagrammatic representation of the arrangements of the pain fibers of the fifth cranial nerve according to anatomic, physiologic, and clinical data. (Modified from Mettler, F. A.: Neuroanatomy. St. Louis, C. V. Mosby Co., 1942.)

Fig. 36.

on its lateral side and the facial nucleus on its medial side; having entered the medulla it is traversed by the roots of the 9th and 10th nerves and is crossed by the posterior spino cerebellar tract (93).

The tract has a length of 60-70 mm of which the upper 40 mm belong to the medulla oblongata (145). Its square gradually decreases during its passage downwards and during its descent, fibres leave it to synapse with the cells of the spinal nucleus of the 5th nerve that lie on its medial aspect (184). The 'tail' of the spinal tract is continuous with the tip of the dorsal column of the grey matter in the spinal cord.

The spinal tract of the 5th nerve is the site where fibres subserving pain and temperature synapse (Fig. 36). This nucleus subserves all oro-facial pain whether mediated by the 5th, 7th, 9th or 10th cranial nerves (184, 106).

Brodal (24) investigated this problem as to whether pain impulses from the oro-facial region conveyed in nerves other than the trigeminal do in fact join with those of the trigeminal field in their central course. According to the doctrine of nerve components, fibres conveying the same type of impulses and from adjacent areas have a tendency to pass to the same nucleus. In fact, fibres entering the 7th, 9th and 10th cranial nerves and joining the bulbosphinal tract of the trigeminal have been described in several vertebrates, such as cyclostomes, ganoids and Teleosts. By analysis of results following tractotomy and cranial nerve section, Brodal confirmed the fact that pain afferents from the face in these other cranial nerves, travel to the spinal tract of the 5th nerve.

The small myelinated and unmyelinated fibres of the 5th nerve which constitute the descending tract exhibit a ventro dorsal lamination according to the peripheral divisions they represent (184, 106, 145, 147). Consequently the fibres of the mandibular nerve are most dorsally situated with the maxillary fibres ventral to them. Ventral to these again are the opthalmic fibres which also descend farthest into the cervical cord to C4 where they synapse. The maxillary division

fibres synapse in the middle part of the tract and superior part of the nucleus receives the mandibular fibres. In other words, the face is represented upside down in the nucleus.

This anatomic and segmental arrangement of fibres in the spinal tract accounts for the fact that lesions arising intracranically may irritate certain segments of the nucleus and give rise to pain in the face and head within the trigeminal distribution. Again, this anatomic arrangement permits the neurosurgeon to produce relief of pain in the face by differential section of the appropriate parts of the spinal tract.

The main sensory nucleus of the 5th nerve is an oval mass of grey matter placed half way up the pons in the lateral part of its dorsal portion and is immediately superior and apparently directly continuous with the spinal tract of the 5th nerve. It lies within the pons beneath the superior cerebellar peduncle with the spinal and medial lemniscus medial to it.

(Note. 1. The spinal lemniscus is formed from the anterior and lateral spinothalamic tracts which convey temperature and pain sensation in the spinal cord from peripheral segments on the contralateral side.

2. The medial lemniscus takes origin from the gracile and cuneate nuclei in the lower part of the medulla on the opposite side. The posterior part of the white column of the spinal cord constitutes the fasciculus gracilis and cuneatus which end in these nuclei. These fasciculi mediate sensations from joints, tendons and muscles and finer sensations of touch and pressure (p.891 C).

The spinal, medial and trigeminal lemnisci pass to the thalamus).

It seems that the fibres subserving mainly touch and possibly pressure are relayed in the main sensory nucleus of the 5th nerve (147, 1).

Dorsal to the main sensory nucleus near the floor of the 4th ventricle is the motor nucleus of the 5th nerve at which level the spinal tract of the trigeminal nerve begins by the

bending downwards of the fibres of the sensory portion. The motor nucleus of the trigeminal is serially homologous with the motor nuclei of the branchial group, namely, the facial nucleus and the nucleus ambigiuus (i.e. the nucleus of the cranial accessory, and of the motor fibres of the 9th and 10th nerves) (41). Fibres of the motor nucleus are distributed chiefly to the muscles of mastication.

As the sensory root enters the pons a comparatively small number of large non bifurcating fibres ascend to the mesencehpalic nucleus of the trigeminus (145).

This nucleus, located in the midbrain is the proprioceptive nucleus for the co-ordination and control of the muscles of mastication (147). It consists of a column of cells in the grey matter on the side of the aqueduct of the midbrain. The mesencephalic root descends along the medial side of the superior cerebellar peduncle into the pons and the site of entry of the sensory root (93). Because of the close similarity of the large cells of mesencephalic nucleus with those of the spinal ganglia which were demonstrated to be connected with muscle spindles and because of the peripheral distribution of the majority of the mesencephalic root fibres to the masticatory branches of the 5th nerve, the concensus of opinion has been that the mesencephalic root represented primary sensory fibres mediating muscle sensibility from the muscles of mastication. The reason why the afferent function of this tract was not suspected until recent times is due to the fact that its fibres arise not in some ganglion outside the central nervous system, like other afferent nerves, but from cells inside the midbrain (41). (The fibres passing to the mesencephalic nucleus of the 5th nerve do not have cell stations in the trigeminal ganglion). Cunningham considers that the neural crest in the mesencephalic region must presumably have been drawn into the neural tube during development and given rise to this sensory nucleus of origin within the central nervous system.

Corbin and Harrison (38) conducted experiments on the function of the mesencephalic root of the trigeminus. They

confirmed that the fibres of the root mediate proprioceptive sensibility from the masticatory muscles. Upon stretching the muscles of mastication, action potentials were elicited from the entire extent of the trigeminal mesencephalic root. These responses were identical with the proprioceptive impulses from peripheral nerves on stretching striated muscle (i.e. slow to adapt, elicitable by pressure over the muscles concerned and immediately abolished by section of the masticatory nerves. The size of the potentials indicated fairly large fibres.

They also demonstrated by degeneration experiments that fibres of this root are found in purely sensory branches such as the superior alveolar, inferior alveolar and palatine nerves. Moreover, responses were evoked in the root from pressure stimulation of the upper teeth and hard palate.

IT IS THUS EVIDENT THAT DEEP PRESSURE IMPULSES FROM THE PERIODONTAL MEMBRANE AND RELATED STRUCTURES ARE RELAYED TO THE MESENCEPHALIC ROOT OF THE TRIGEMINUS.

Responses in the root have been from homolateral teeth, palate and masticator muscles, never from contralateral structures. This in agreement with anatomical studies which show that a small portion only of the mesencephalic root fibres cross to the opposite side.

Medullated fibres arising from the large cells of the mesencephalic nucleus are considered as forming the afferent limbs of communicator reflex arcs, some passing directly to the motor nucleus of the 5th nerve via the medial longitudinal fasciculus for short reflexes.

It seems that the proprioceptive impulses from the masticatory muscles combine with the "pressure sense" of the dental soft tissues and periodontal ligaments serving to co-ordinate mandibular movements and control the forces of mastication (168).

Summary of Neurone I connections of the trigeminal nerve

Main sensory nucleus of the 5th nerve - common sensation e.g. touch

Spinal Tract and nucleus of 5th nerve - pain and temperature fibres

Mesencephalic tract of 5th nerve - proprioception and pressure sense from muscles of mastication and periodontal tissues motor nucleus of 5th nerve higher centres

Neurone II connections

Fibres of the second order arise from the cells located in the main sensory nucleus and the spinal nucleus of the 5th nerve. Many of them cross the lower brain stem to the opposite side to ascend in the bulbo thalamic tract. However, a considerable number of the fibres ascend in the ipsilateral bulbo thalamic tract or trigeminal lemniscus (95) (81) (184). There is thus a bilateral projection of facial pain and the resultant homolateral and contralateral cortical representation results in incomplete analgesia after unilateral hemispherectomy.

Ascending from the pons, the fibres of the bulbo thalamic tract spread out in the midbrain until they end in the thalamus.

Appreciation or consciousness of pain arises from structures above the level of the pons and lesions of the upper pons will permanently abolish the appreciation of pain from peripheral stimulation (77).

The thalamus, from which arise the third order neurone fibres, is described as the great sensory relay station in whcih all ascending sensory impulses are transmitted to the sensory area of the cortex. The thalamus and hypothalamus seem to function as a complex relay centre for the expression of emotion and autonomic adjustment (168).

The thalamus presumably synthesises all the impulses coming in from the peripheral branches of the trigeminal nerve and adds a synergic affective tone, not only for the pain impulses, but also for the impulses of touch and pressure (147).

The posterior part of the ventral nucleus of the thalamus

is the terminal station of ascending fibres which reach it via the lemniscus system both medial, spinal and and trigeminal, and is therefore a terminal centre for proprioceptive and exteroceptive sensory impulses derived from all parts of the body. The different regions of the body acquire quite a definite topographical representation within the 'lemniscus nucleus' of the thalamus. Sensory impulses from the lower and upper limb terminate in its most lateral and intermediate part respectively, while those from the head and face lie in the most medial portion of the posterior ventral nucleus. Consequently it is possible for a small localised lesion in the thalamus to give rise to sensory disturbances in one part of the body only (41).

Neurone III Connections

The neuronal organisation beyond the thalamus is less well known (106), but it appears that stimuli pass predominately in thalamo-cortical fibres to excite cortical neurones in the inferior parietal region, including the lower part of the post central gyrus (184).

There is again a definite topographical organisation in the post central convolution, so that those fibres from the medial portion of the ventral nucleus of the thalamus (cephalad parts) end in the lower part of the gyrus, those from the lateral portion in the paracentral region and those from the middle in the intermediate region.

The post central projection system is involved in PERCEPTION of the pain experience and is mostly discriminative. It makes possible the anatomic localisation of the source of pain and identification of the physical nature of the stimulus e.g. pinprick, blow, burning pain etc. (106). Awareness of the nature of the stimulus is due to the stimulation of other receptors besides pain and subsequent analysis and intergration of this accompanying information at a cortical level.

It will be remembered that the wide intercommunications of nerve nets in the skin subserving pain results in the feeding

of stimuli into multiple afferent channels and this enables accurate spot localisation. Again, it has been suggested that many specialised receptors may have an associated accessory pain fibres. Thus vigorous scratching would stimulate first touch and light pressure receptors and then the associated pain receptors. The stimulus to touch and light pressure is accurately localised and this localisation ia then appended to the associated pain sensation (183).

At the highest cortical level, pain sensation is highly localised and coloured by associated tactile and pressure sensation (77).

The fact that pulpal pain and deep visceral pain is poorly localised and identified is probably a reflection of the fact that these structures exhibit a paucity of sensory receptors other than for pain sense.----

Apart from the thalamo cortical projection, a second relay arises in the thalamus which passes to the medial nucleus of that structure. Projections from this nucleus pass mainly to the cortex of the <u>frontal lobe</u>. This projection is important, for it subserves the function of EMOTIONAL RESPONSE to painful stimulation. Pain is thus interpreted as unpleasant, agonising or intolerable by the sufferer, and as will be described, the operation of orbito-frontial leucotomy is sometimes employed to relieve suffers from the intolerable emotional affect of pain.

Connections also exist between the medial nucleus of the thalamus and the <u>hypothalamus</u> which are responsible for the familiar visceral and hormonal changes which are the concomitants of pain. The connections of the thalamus with the frontal lobes and with the hypothalamus and further links between the frontal areas and the hypothalamus are responsible for not only the emotional component but also the attitudinal reactions of both pain and fear including autonomic reactions such as sweating, tachycardia and lacrimation (147), i.e. EMOTIONAL 'EXTERIORISATION'

The spinothalamic and bulbo thalamic tracts also send collaterals to the brain stem <u>reticular system</u>, which projects

to all areas of the cerebral cortex for which it acts as an alerting mechanism (106). The awareness of an individual to his environment and the INTENSITY OF SENSORY EXPERIENCES are determined by the level of this reticular activation. The experimental evidence is that of all the afferent modalities, pain is the most potent in affecting the activity of the reticular formation and pain from the facial area results in maximal 'drive' to the cortical cells (184).

The activity of the reticulo cortical system therefore determines the intensity of the patient's whole experience of pain. It is by their action on this system that 'tranquilizers' potentiate the action of analgesics. They help to diminish the individual's contact with his environment and his awareness of painful stimuli.

This explains why there is such wide differences between patients in terms of their clinical tolerance to pain, even though thresholds for perception vary but little (see page 151). In the tense anxious subject, there is a high level of reticular discharge, and this reticular hyperexcitability sets his pain tolerance at a lower plane than exists in a more relaxed, phlegmatic person.

Concerning the Emotional Affect of Pain

Though pain is felt in a particular region, the patient's description is greatly influenced by the affective response. The emotional affect of pain is really of primary importance for it can dominate the sufferer's behavioural patterns to such an extent that normal existence is impossible. After prefrontal lobotomy (page 140) has been performed on a patient suffering from intractable pain, a complete release from emotional affect is seen. He admits to pain, and describes it as identical with the past pain i.e. there is no change in sensory perception, but he readily changes to another topic of conversation, for the dominating and destructive response to pain no longer exists.

It is interesting to consider the emotional affect of pain, as distinct from sensory perception, in more detail.

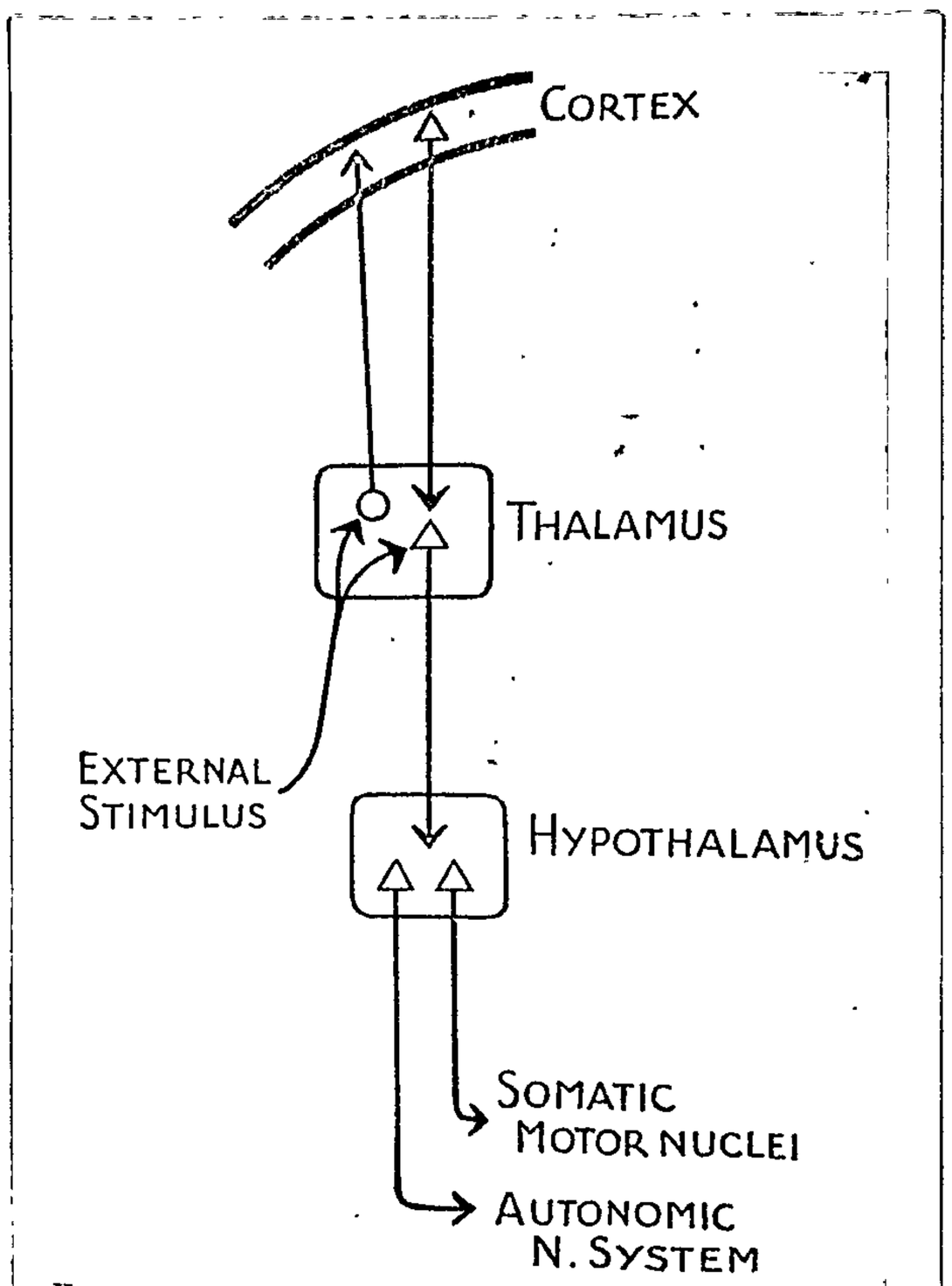

Fig. 37 Diagram to illustrate reflex arcs employed in exteriorisation of emotion.

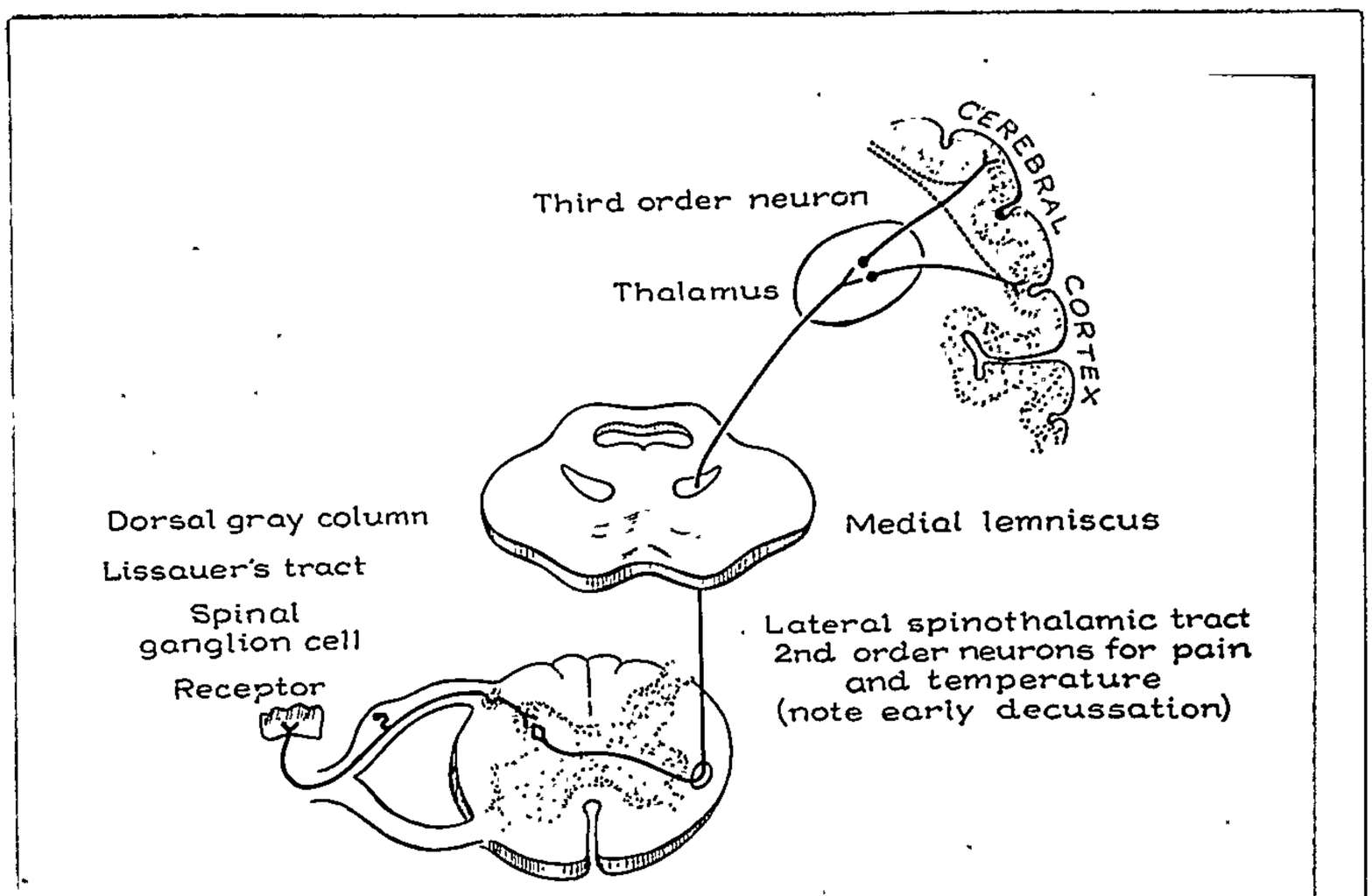

Fig. 6. Pathway of pain and temperature perception.

Fig. 38.

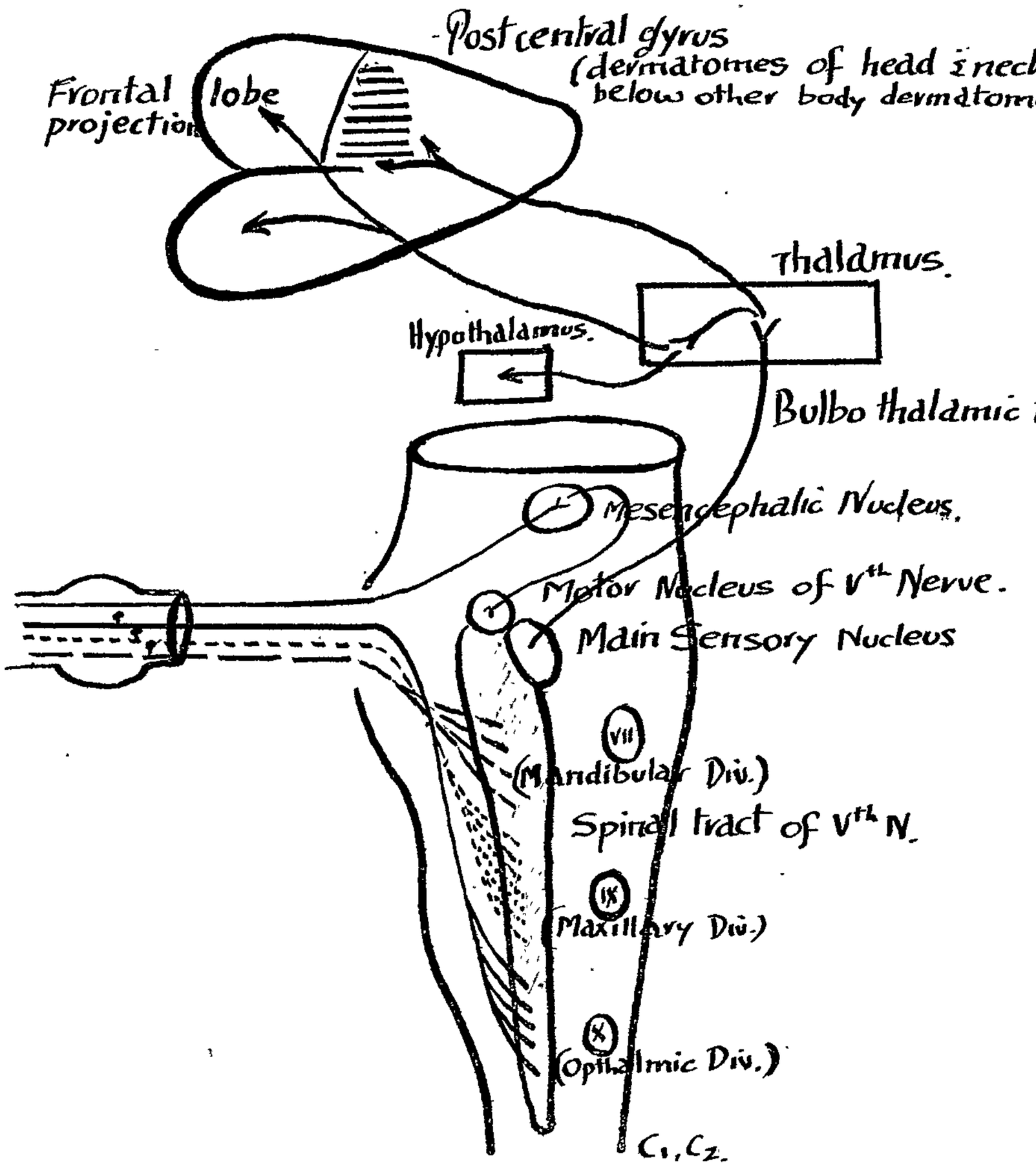

Fig. 39. Central connections of the 5th. cranial nerve.

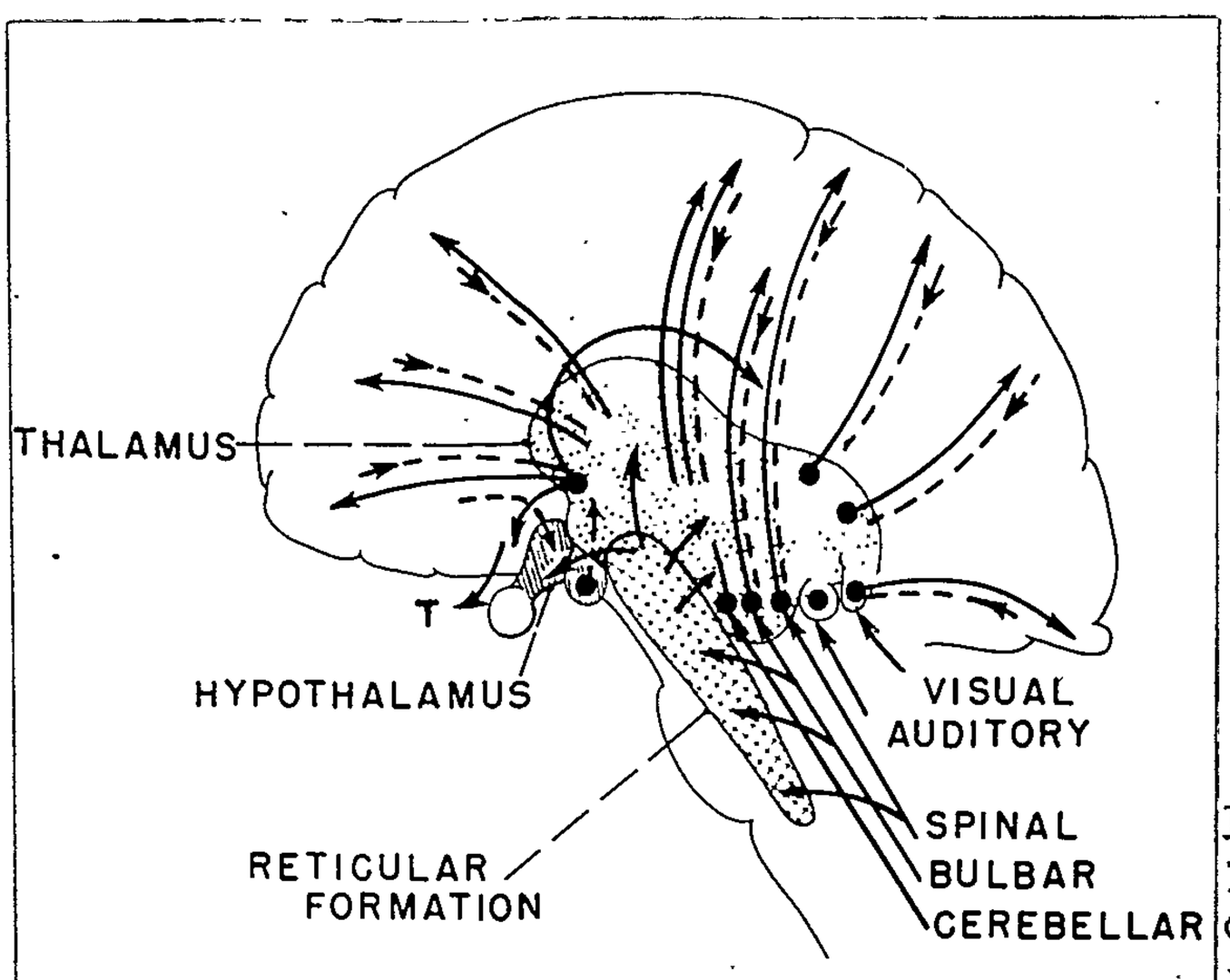

Fig.40. Afferent impulses from various sesory end-organs reach the thalamus and are relayed to the cerebral cortex, where returning neurones form thalamocortical circuits. Before reaching the thalamus, spinal, bulbar, and cerebellar tracts give off collateral branches to the reticular formation of the brain stem, which then acts as an activating mechanism for both thalamus and hypothalamus.

Leucotomy may interrupt the frontal thalamocortical circuits and the paths and the paths from the frontal areas to the temporal cortex (T) and the hypothalamus.

As an example one may examine an individual's reactions when he is hit in the face:-

a) Pain is felt (perception of pain as a sensation)

b) It is frightening (emotional affect)

c) It makes the sufferer want to run away (emotional reaction)

During emotional stimulation activity develops in large areas of the brain. These set up a discharge to viscera and muscles (emotional reaction or exteriorisation) and at the same time, in an unknown manner, they give rise to a feeling or mental state (emotional affect). There is a great variation, however, between emotional affect and emotion exteriorised i.e. between the mental and physiological changes in differing individuals.

For instance, in the above example, the immediate emotional reaction to the painful blow prompts flight, but the actual physical reaction may be modified and inhibited by cortical influences.

The emotional affect, however, may arouse widespread autonomic affects depending on the intensity of the feeling state.

The sympathetic nervous system may dominate the response - increased heart rate due to increased adrenalin secretion, vasoconstriction and 'cold sweat', irregular salivation etc.

In some cases parasympathetic overactivity predominates e.g. the vaso vagal syndrome in which there is depression of the vaso motor centre, lowering of the peripheral vascular tonus, slowing of the heart due to vagal inhibition and a fall in blood pressure.

Emotional states also influence the secretion of hormones e.g. adrenalin, the antidiuretic hormone and the discharge of anterior pituitary hormones.

Pain, therefore, depending upon the intensity of the stimulus and the emotional reaction of the individual, may result

in complex autonomic and somatic activity and hormonal effects.

The connections of pain pathways with the thalamus, hypothalamus, cerebral cortex and prefrontal areas have been examined and their respective functions in the regulation and mediation of these complex patterns of bodily activity will now be considered further.

Role of the Subcortex (183)

It has been shown that the thalamus intergrates and relays sensory stimuli to the higher cortical areas. Fibres also pass between the thalamic nuclei and the hypothalamus in both directions. Two way connections are present between the prefrontal cortex and the hypothalamus and from the hypothalamus, new relays pass firstly to the appropriate supero spinal autonomic centres e.g. the cardio-vaso motor and secondly to nucleii in the midbrain and the reticular formations of the pons and the medulla. From these latter cells, relays pass to the appropriate cranial and spinal lower motor neurones.

These connections of the hypothalamus are very important for it seems that the hypothalamus not only controls both divisions of the autonomic nervous system but also influences the activity of the lower motor neurones which supply skeletal muscles. It is thus an important centre in the mechanism controlling emotional exteriorisation.

The function of the thalamus - hypothalamic complex is observed after transection of the brain in cats, just anterior to the thalamus. The resultant 'thalamic animal' exhibits a remarkable 'sham rage' to trivial emotional stimuli. Widespread sympathetic activity occurs associated with erection of the hairs, rapid heart rate, hyperglycaemia etc. and is associated with bodily movements such as kicking, running and biting. It seems that the thalamic preparation responds to noxious stimuli by signs of intense fury - in other words emotional exteriorisation is completely uncontrolled.

The role of the thalamus - hypothalamus is thus seen in the mediation of coarse emotional reactions.

Role of the Cortex

Fibres from the precentral and prefrontal areas of the cortex reach the thalamic region and seem to modify its inherent reflex responses. The integrity of these cortical connections introduces the element of refinement, nicety of adjustment and greater variation of patterns in emotional exteriorisation, and the resultant reaction is better adapted to deal with the casual condition.

Evidence shows that stimulation or excision of parts of the prefrontal lobes may enhance or depress the outward manifestations of emotions, alter patterns of behaviour and produce changes in the personality.

The aim of the operation of prefrontal lobotomy is to sever connections with the thalamus. This is most effective in individuals suffering from intractable neuralgias and mental tension, for it releases the patient of the persistent emotional overlay.

It seems that emotional exteriorisation i.e. the reaction to a feeling state, is essentially an involuntary reaction, which can be mediated by subcortical levels. It is however modified and to a considerable extent inhibited by cortical influences. The extent of such cortical control varies in different individuals.

As the prefrontal lobes are linked up with the thalamus and hypothalamus by so many fibres passing in both directions, the whole system may be considered as functioning as an integrated whole. The activities of this nervous complex are correlated with some of the higher intellectual activities, the personality and the forms of behaviour of social significence.

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CHAPTER 6

CONCERNING THE NATURE OF PAIN

Certain aspects of the pain experience require further consideration and analysis. For instance, the intensity of pain and the sufferer's reaction to it varies greatly. This individual response is not directly proportional to the severity of the stimulus for it may be greatly exaggerated by the individual's subjective reaction.

Again, the region from which pain arises may be located in certain instances with great precision, while in other circumstances localisation is poor. Added to this variable reaction to pain and the difficulty encountered in localisation in some instances, is the difficulty experienced in describing the pain's quality or tone.

This chapter will therefore consider the essential differences between cutaneous and deep somatic pain as regards quality, localisation and associated phenomena.

Cutaneous or Superficial Pain:

Pain from the skin and mucous membrane is localised very accurately. It varies in intensity and in duration and may change from moment to moment. Pain may be provoked by injuring the skin in a larger number of different ways, as by pricking with a needle, by pulling of hairs, by burning or by the passage of an electric current.

Lewis draws attention to the fact that the quality of pain evoked in these several ways is unvarying and the subject is usually unable to differentiate between the stimuli applied (100). Lewis observed that a brief stimuli to the skin is usually described as pricking whereas a prolonged stimuli gives rise to a pain described as burning whether the pain arises from heat or not. Collins (34) states that tissues which give rise to pricking and burning pain such as skin and mucous membranes are derived from the ectoderm, as distinct from the aching pain elicited from mesodermal and visceral structures.

Pain which continues for a period after injury to the skin

e.g. after removal of a hot object, after abrasion, or after application of irritant substances, is often described as 'smarting' or 'stinging' as well as 'burning'. Lewis maintains that pain answering to these three descriptions cannot really be differentiated in respect to quality.

Lewis considers that pricking and burning pain are of the same quality i.e. brief noxious stimulation of the skin produces pricking pain while prolonged stimulation by the same agent causes burning pain.

Other investigators doubt that pricking and burning pain are variations of the one quality and maintain that they are two separate qualities mediated by nerve fibres of differing diameter ('78, 174, 180, 35, 15). Hardy and Goodell ('78) state - "in our opinion, these two qualities of pain are --------- distinguishable and quite independant of duration of the stimulus".

Some writers consider that there is a correlation between pricking and burning pain and the 'fast' and 'slow' pain conducting fibres in the skin.

This double nature of cutaneous pain as originally postulated by Kosenbach in 1884 and Goldscheider in 1892, has been clearly demonstrated by Gasser. When the finger near the nail bed is touched against a hot electric light bulb ..... "there are felt two distinct flashes of pain, one coming almost at once, the other after a discernable delay. The second may be more intense and prolonged". A similar double flash can be felt after a needle prick.

Lewis and Pochin (98) verified this double response from a single stimulus and noted that the threshold for the second response is markedly lower than for the primary response, and a light stimulus will give only the second pain response. This has been confirmed by other workers (174) who have shown, when using the technique of thermal irradiation of the forehead, that first pain threshold is appreciable higher than that for second pain.

Thulenberg (quoted by 100) supposed that this first

response was due to stimulation of nerve fibres and the second stimulation of nerve endings. This explanation also appealed to Von Frey.

Gasser and Erhlanger (68) clarified this problem by showing that fibres of peripheral nerve trunks conduct impulses at different rates according to their size and that pain from cutaneous areas may be carried by fibres of small and relatively large diameter. Medullated delta fibres conducting at a rate of 10 - 99 metres/sec. convey fast pain or first pain whereas slow or second pain is carried by small "c" fibres at a rate between 0.6 and 2 metres/sec. (page 38).

The opinion that "pricking" and "burning" pain are separate qualities and are conducted by "fast" and "slow fibres respectively is now generally accepted.

Bigelow (15) states that one type of pain is abrupt in onset and has a pricking quality which quickly terminates. The other is slow in onset and reaches a climax, receding slowly. The latter pain, they say, has a burning quality, whether initiated by pin prick or heat.

Wolf and Woolf consider that "bright pricking pain is carried in the thicker, rapidly conducting myelinated fibres. Burning pain felt in the skin is transported by fibres of small size which conduct more slowly".

Some workers further speculate that first pain is excited in the epidermis and second pain subepidermally i.e. epidermis and dermis mediate pain of different quality. It is also thought by some that the burning quality of superficial pain is intermediate between pricking pain and the aching quality of deep pain. It does seem that the fast conducting fibres have more superficial and widely distributed terminals than the slow fibres, but whether the first and second pain mechanisms are arranged in such a precise 'layer' fashion remains to be demonstrated more conclusively.

It is very probable however that the skin is endowed with two types of pain transporting fibres conveying two different

qualities of sensation.

Theories of Pain Localisation in Superficial Tissues:

The extreme accuracy with which cutaneous pain is localised is well known and several theories have been proposed to explain this.

Head in 1905 believed that cutaneous sensibilities were of two kinds, namely 'protopathic' sensibility capable of responding to painful cutaneous stimuli and to the extremes of heat and cold and 'epicritic' sensibility by which finer impressions allow the power of localisation, of two point discrimination and of the finer grades of temperature called cool or warm.

Such conclusions were based on observations made on the recovery of sensation after division and suture of the superficial radial nerve in Head's forearm. He found that 'protopathic' sensibility was first noticed in the area supplied by regenerating fibres. He assumed that this type of crude sensation is the normal condition in lower animals. The return of 'epicritic' sensibility gradually followed and it was postulated that this is a higher evolutionary form of sensation which inhibits the more primitive form.

In normal skin, the two systems were supposed to supplement each other with the epicritic suppressing the disagreeable intensity of the protopathic as well as adding to it finer modalities of sensation. The protopathic system, according to Head is the more primitive and pain from deep lying structures is solely of this type (dull, aching and poorly localised).

These observations were purely functional and no direct attempt was made to ascertain the actual existence of two distinct systems of receptive end organs and conducting axons in the nervous plexuses of the human skin.

Head and others, besides postulating this dual peripheral mechanisms also supposed that there were two separate central systems of distinct phylogenetic development viz. the thalamus and the cerebral cortex. This epicritic cortical control of the thalamus is, however, unsupportable, for protophatic sensibility

does not develop after hemispherectomy or removal of the post central cortex.

Trotter and Davies (1926) in similar experiments found no evidence of sensory return in two distinct stages. All modalities tend to reappear together and the idea that there were two separate fibre systems for crude and delicate sensibility was not borne out by their observations. Trotter ascribed the disagreeable quantity of pain experienced in a hypersensitive area of the regenerating nerve to the loss of normal insulation of the myelin sheath. This lack, he said, makes all regenerating sensory axones resemble pain fibres so that the result is over reaction to any stimulus in an exaggerated explosive way. After further regeneration, however, and the redeposition of myelin this disagreeable quality in sensation disappears.

This theory tends to ignore the differential central connections of sensory fibres. Also there is no experimental evidence that it is the fibres that are eventually destined to have more myelin whose impulses are actually responsible for the abnormal sensations.

Advances in our knowledge of nerve histology and physiology have rendered the epicritic - protophatic theory untenable. The views of Head and Trotter concerning the abnormal hyperaesthesia present in the skin supplied by a regenerating nerve are unsupported by research.

Localisation of somatic pain has also been ascribed to the associated stimulation of the tactile sense Lewis (10Q) by carefully conducted experiments, eliminated tactile sensibility from an area and found that localisation when stimulating the pain sense with a needle has the same order of accuracy. Although the necessity for associated tactile sensation for localisation was thus refuted, Lewis did find that deeper tissue which did not possess this sensibility, such as subcutaneous tissues, periosteum, fascia muscles, etc., exhibited progressively less ability for lcalising pain.

"Evidence shows that the depth at which tissues lie is

more influential in determining the accuracy with which pain is localised than is the nature of the tissue stimulated. Thus beneath the skin there is a second sensitive layer from which pain may be localised with fair accuracy, consisting of deep fascia encasing limbs, periosteum and ligaments. On the other hand, all structures deep to this layer give rise to diffuse pain which is of more or less segmental distribution".

Anatomical evidence presented by workers such as Widdell and Powers ascribes the normal quality of cutaneous sensation and spatial perception to a complex network of overlapping nerve fibres and multiple sensory terminals. This is to say that the accuracy of localisation of pain from a tissue seems in direct proportions to the multiplicity of pain fibres ennervating the tissue (Chapter 3).

The paucity of overlapping nervous networks in deeper tissues results in poor localisation.

The reason why localisation is poor and sensation abnormal from an area supplied by regenerating nerves is therefore clear. White and Sweet (3) state that ..... "in the phase of regeneration in which this multiple innervation is incomplete, there will be no anatomical basis for spatial perception. Therefore, reaction to stimuli will tend to conform to an all-or-nothing law, leading to the characteristic explosive type of sensation and there can be no recognition of graduation and no possibility of accurate localisation".

Pain Threshold:

A fundamental distinction must be made between the perception of pain and its associated reactions. Pain threshold may be defined as the lowest perceptible intensity of pain. The pain threshold stimulus is the amount of emergy required to induce threshold pain (180). Pain threshold is considered to be raised when more stimulus is required to induce threshold pain; conversely when less stimulus energy is required, the pain threshold is said to be lowered.

For pain threshold measurements, it is necessary to choose

a stimulus, the strength of which can be controlled and measured and which will allow a clearly defined end point of perception.

Many methods have been tried for obtaining pain threshold values, but most have been unsatisfactory because of:-

a) the failure to establish a firm relationship between the quantity measured as the stimulus and the amount of pain producing disturbance in the environment of the pain endings,

and

b) the fact that reaction to a noxious stimulus is often confused with perception of threshold pain (174).

In electrical stimulation of the teeth, for example, the amperage, voltage, frequency and resistance in the circuit are all of importance in inducing pain. Measurement of one of these quantities as the pain threshold might be expected to lead to considerable variability (126).

Mechanical methods of eliciting pain such as measuring pressure required to evoke pain in the skin or oesophagus or on the thyroid process are also unsuitable. The fact that relatively stoical people have a high threshold of reaction makes the measurement of the threshold for perception very difficult using these methods.

Quantitative measurements of pain threshold therefore requires an instrument which can induce pain without direct tissue damage by a stimulus whose intensity can be accurately measured in physical units (180). Pain threshold is established verbally from the subject.

The most satisfactory method of measuring threshold for cutaneous pain involves the exposure of an area of skin to radiant heat (180, 174). Sensory impulses arising at the end organs in the skin have been shown to be proportional to the thermal gradient and therefore the intensity of radiation is proportional to the amount of pain producing disturbance at the end organ.

By this method both pricking and burning pain threshold

can be ascertained. The stimulus is expressed in mgm. cals/ sec/sq.cms. The pricking pain end point seems to be the most convenient to measure.

It has been observed that the pricking pain threshold following thermal irradiation of the forehead of a single individual, could be reproduced with ±3% in succeeding experiments. The pain threshold on a group of 200 individuals was thus measured and was found to require the same intensity of stimulus to evoke pain, with a standard deviation of ±5% (134).

Such a result was gained testing similar individuals successively while under varying emotional states, and the pain threshold measurements did not vary beyond normal limits. Also, throughout a 24 hour period of forced wakefulness, the pain thresholds were uniform and normal.

It is therefore shown that the threshold for pain perception in man is relatively stable and uniform and is independant of age, sex, various emotional states and fatigue (35, 174, 80, 180, 134). There is little variation from day to day and person to person with normal people and normal tissue.

Wolff's work on pain threshold has not gone uncriticised. Schamp (129) failed to confirm the uniformity of pain threshold and found large variations. This is an instance of the difficulty found in separating threshold for pain perception from that of pain reaction.

Chapman et at (28) found in a study of 50 psychoneurotics patients and 56 normal control subjects, that both types of individual required essentially the same amount of stimulus to perceive cutaneous pain. The psychoneurotics however, required a much smaller stimulus to cause a motor withdrawal reaction.

Various investigators have shown that the pain threshold may be elevated under certain conditions. Distraction and suggestion has been found to raise the pain threshold (80, 176).

Distraction due to reading an adventurous story aloud raised the threshold 15%; playing a loud bell immediately behind the subject's head or the intense concentration of the subject on

such a task as repeating 5 - 9 digits forward and backwards resulted in a rise of 45%.

Autosuggestion, that is having the subject try and convince himself that he could not feel pain had the effect of raising threshold 20%. This elevation of the pain threshold during states of suggestion, distraction and excitement explains why injuries incurred during athletic exertions or extremes of emotion such as in sexual excitement cause so little pain even though the point of tissue damage has been reached (171, 81).

Hynosis also increases pain tolerance, much depending on the success in achieving an adequate depth of trance. While 15 - 20% of subjects readily pass into a deep trance where almost any operative procedure without chemical anaesthesia may be undertaken, many lie in an intermediate grouping varying from light to medium. In some cases anaesthetic reinforcement is necessary, the light trance serving to reassure anxieties and allay apprehension. Shallow hypnosis may itself raise the threshold for pain perception above 40%.

Analgesics act in most cases by raising the threshold. Indeed, morphine, codeine and alcohol do not affect the threshold of sensory experience other than pain (Figs. 41 & 42) (180). 0.3 gms of acetyl salicylic acid raises the threshold 30% in 1½ hours.

Wolff and Wolf state that with most analgesies, an optimal dosage was found which afforded close to maximum pain relieving action. Increasing the amount beyond this point did not raise the pain threshold significiantly, prolonged to effect only slightly, and accentuated the undesirable side effects. They also found that the combination of two analgesic agents did not produce an additive effect, but merely the effect of the stronger agent.

The importance of suggestibility was demonstrated by Wolff et al (176) who administered 0.9 gms of acetyl salicylic acid to such suggestible women. Threshold when tested was raised 31%. A lactose capsule was then administered under the name of

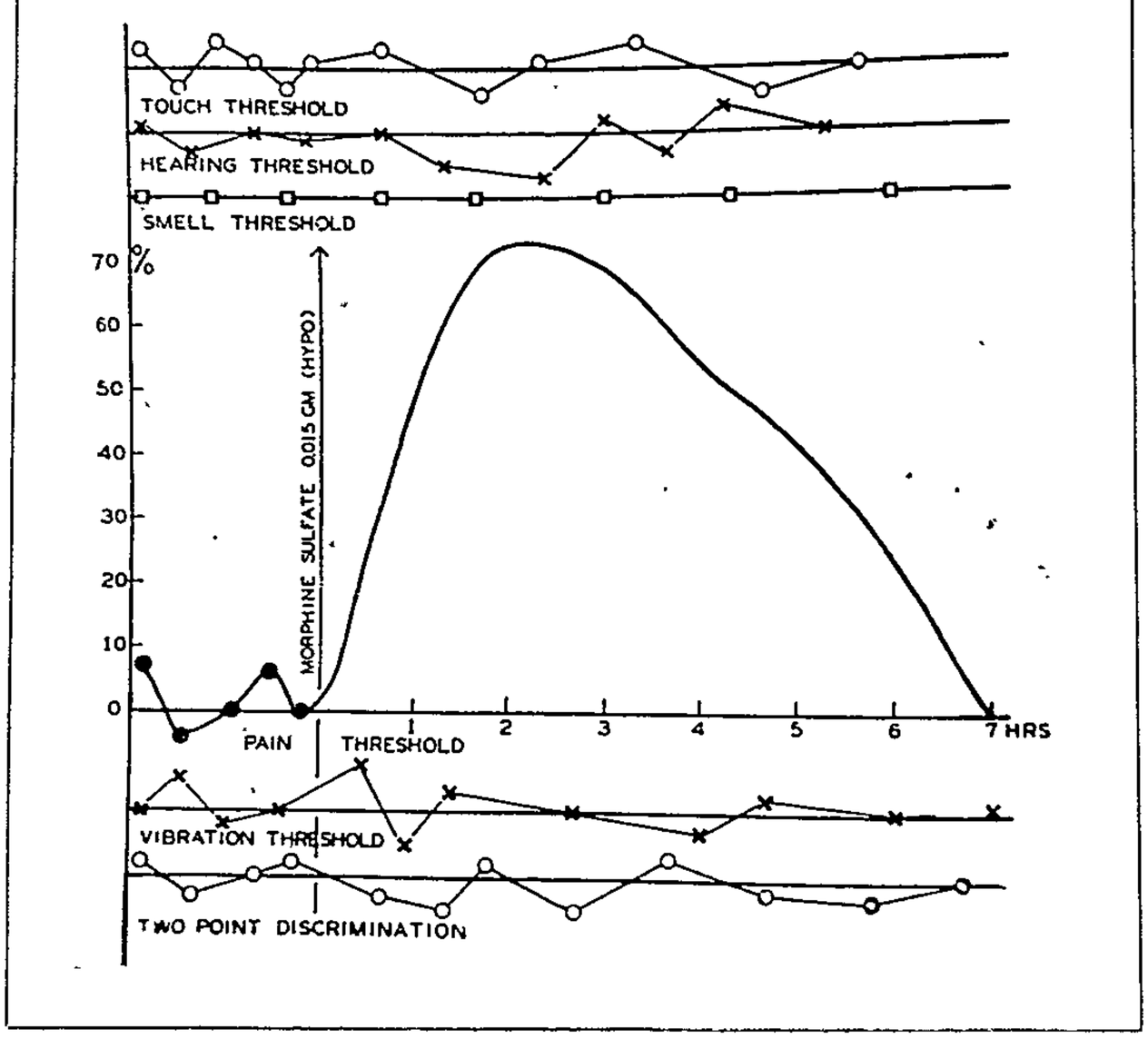


Fig. 41. The effect of morphine sulphate on the thresholds for touch (Von Frey hairs); hearing (audiometer); smell (Elsberg apparatus); vibration (Roth neurometer) and on two point discrimination - compared with its effect on the pain threshold.

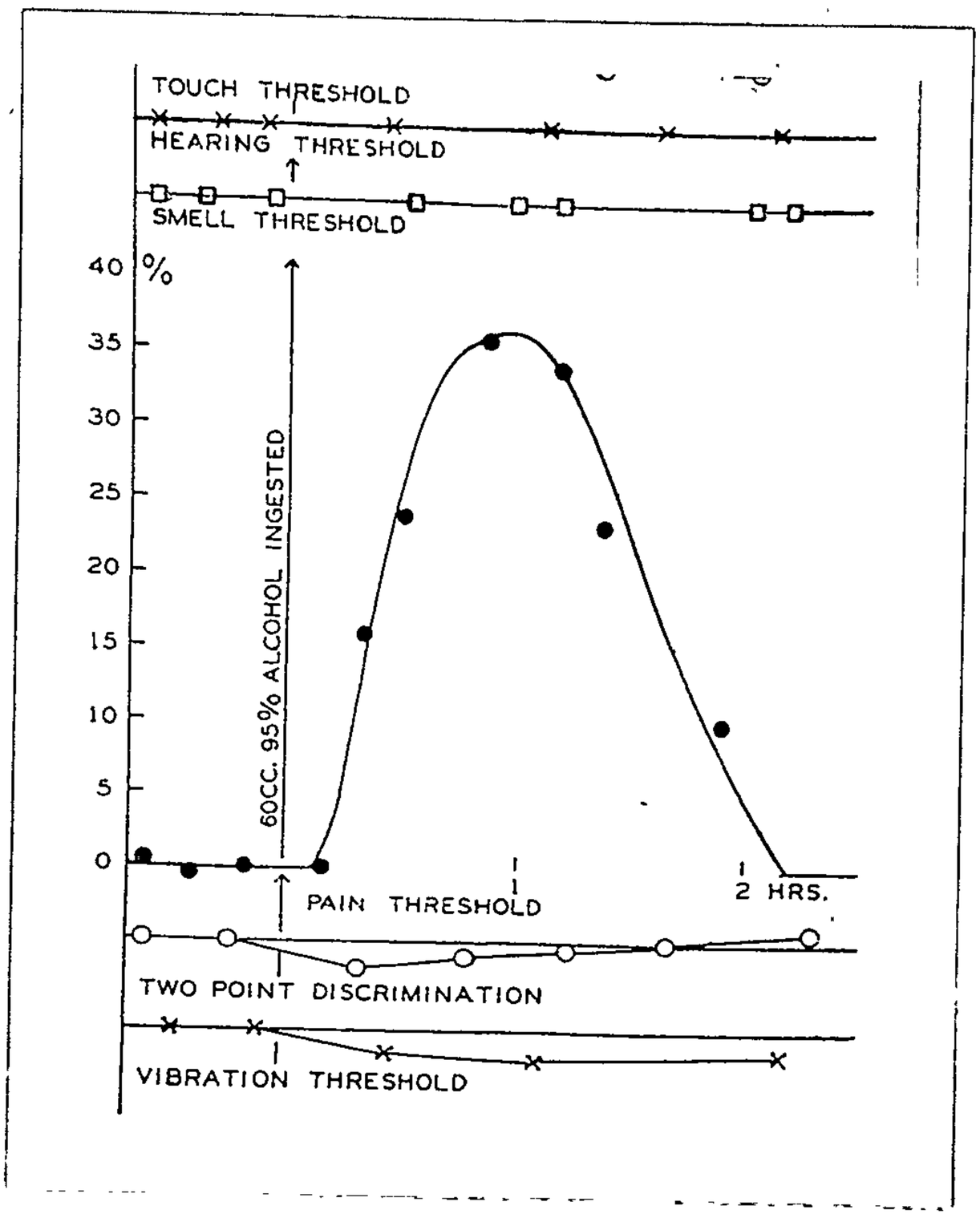


Fig. 42. Effect of ethyl alcohol (60 c.c., 95%) on pain threshold (as above)

an analgesic and threshold was found to be raised 20%. After the subject was informed of this experiment, more accurate results were obtained when given placebo or analgesic.

The existance of one pain has been found to raise the threshold for perception of another. This effect is subconsciously made use of when, persons in pain bite their lips or drive their fingernails into their palms. Gammon and Starr (63) have shown that the intensity of deep pain may be reduced by non-noxious tactile, pressure and thermal stimuli applied to the skin. The intensity of the counter stimulus however, must be just below that intensity which produced manifest discomfort when applied to normal skin. This fact incidentally proves that pain does not exhibit the phenomenon of spacial summation (Chapter 2).

Elevation of the pain threshold is also found in certain diseases of the nervous system e.g. syringomyelia (176).

Lowering of the pain threshold may occur in certain physical states such as in hyperalgesia of inflammation (see page 157). All else being equal however, lowered pain threshold has been found associated only with hysterical conditions and malingering. It is never due to structural disorder of the nervous system - the latter, if it causes any damage to pain threshold, always raises it (80).

Reaction to Pain:

Distinction has been made between pain perception and pain reaction (Chapter 5), and it is important to recognise this twofold nature of the pain experience (142). Pain perception has a physiologic basis whereas pain evaluation or reaction has a psychologic basis and the latter aspect of the pain process takes into consideration the patient's emotional as well as his physical status (109).

Most pains are interpreted as unpleasant and consequently give rise to some reaction on the part of the affected subject. What he feels, thinks or does about it constitutes an individual's reaction to pain (180). The pain reaction is really compounded

of an emotional effect (Chapter 5), as well as the autonomic and somatic effects including smooth, and skeletal muscle and glandular activity (176).

Although the threshold for perception is of the same order of uniformity as is the pulse rate or the number of white blood corpuscles, the reactions to pain vary greatly among individuals (80), and in the same individual under differing circumstances. Various electrical and mechanical devices have been used for measuring the threshold for pain reaction.

Chapman et al (28) used radiant heat as a painful stimulus, considering a wincing characterised by narrowing of the eyelids as evidence of pain reaction. Great variability was noticed, especially among patients with neurotic tendencies.

The dental surgeon, more than any other has the opportunity of noting the variability of patient reaction to pain. Intense pain may be borne stoically by the most diminutive patients, while in other more athletic individuals threshold pain stimulation results in severe reaction patterns - tachycardia, sweating, snycope together with a positive tendency to flight or fight.

The ability to perceive pain depends on the intactness of relatively simple and primitive nerve connections whereas reaction to pain is modified by the highest cognitive functions (142) involving the thalamic, hypothalamic central nervous system.

The patient's attitude to pain and the degree to which it dominates his outlook is often linked with certain tensions, apprehensions and fears. A mild degree of pain may become a major problem in a person if it is associated, for instance, with the fear of cancer. In all cases, the depressive state allows pain of low intensity to cause a disproportionate incapacity (157).

The value of distraction in modifying reaction patterns is well known e.g. the indifference to injury sustained during combat, religious practices or athletic games.

Such, however, is far from the case when a patient is dying of cancer or an individual is suffering from toothache in the small hours of the night. There is no solace from distraction and the pain is increased by introspection.

Drugs such as alcohol and morphine which induce in the subject a feeling of euphoria and freedom from anxiety may raise enormously the threshold for reaction to pain, thus exerting an effect several times greater than, and in addition to, their influence on pain perception (81, 180). These drugs modify the reaction pattern of pain characterised by anxiety and fear (80).

Excitance of Pain Nerves:

Pain is unique in that many methods of stimulation may evoke it (183) 202). It is a warning of impending or actual tissue damage, and therefore painful stimuli are in general strong enough to be injurious (77, 100). While it can be stated in general that where there is pain there is injury, it cannot be stated as a corollary that where there is injury there is always pain, e.g. skin may be damaged painlessly in extreme cases of urticaria factita (100).

The receptors which deal with pain are much more numerous in the skin than in the viscera of the body, and while skin receptors respond to a wider variation of stimuli whether mechanical, chemical, thermal or galvanic, pain receptors in the viscera seem to be activated primarily by tension stimuli.

Intense painful contraction of the muscular wall of a hollow viscus or alteration in the state of nutrition of a part where blood supply is inadequate, are other stimuli for visceral pain.

The amount by which a pressure must exceed that which is felt as deep pressure before it elicits a painful sensation, varies from one part of the body to another. On the arm, an increase by three times is adequate whereas on the fingertip, an increase of 100 times is necessary (202).

It might be argued that in the evolutionary scale, the

units subserving pain, and their susceptibility to stimulation, are distributed throughout the body in proportion to the functional requirements of the area.

Whatever the injurious stimulus applied to the skin, there is a close correlation between pain and damage to the cells of the skin, rather than damage to the pain endings.

If considerable heat is transiently applied to any point on the skin, a burning pain is felt momentarily during the application, and this quickly subsides. This pain is due to direct physical excitation of the pain nerve endings by the heat. However, we notice a similar burning returning after an interval of several seconds following the removal of the source of stimulation, and this is long maintained. This recurrent pain is not due to the direct effect of heat but must be due to chemico- or physico-chemical changes occurring in the skin during this interval which become adequate in providing a stimulus (100).

Lewis reasons that the pain-producing agents are probably products of cell damage. Since injury produces the triple responce and this is due to the release of histamine-like substances from the cells, he introduced histamine experimentally, into the skin to observe whether pain was produced. He found that histamine, pricked into the skin produced itching but it did not produce pain.

Itching seems to be an initial response to a slight stimulus which, when increased in strength, produces pain. Whether itch or pain is produced may depend on the grade of tissue damage and thus may be a question of how freely cell contents are liberated.

Lewis is of the opinion that, though similarly produced, itch and pain are separate phenomena and both, though due to release of tissue substances, are not necessarily due to the same substances. He further considers that the pain response may be mixed with itching but may be severe enough to disguise the underlying itch.

Other chemical substances besides histamine-like agents are known to cause pain, e.g. acetycholine applied to blister areas of the skin. Again the stimulus for muscle pain may arise out of muscle activity and the accumulation of metabolites during contraction, e.g. lactic acid due to local anoxia and acidosis (184).

Wyke states that as far as muco-cutaneous pain is concerned "mechanical deformation or the action of chemical agents such as histamine and 5-hydroxy tryptamine released from the damaged cells are the only stimuli operating".

Thus excessive heat or cold, stretch or tension, electric currents, scratching, cutting or chemicals and hypertonic solutions cause pain because they result in cell damage and the release of irritant substances.

One aspect which requires consideration is the pain elicited by local cooling. Wolff et al (175) found that the intensity of pain depended upon the degree of cooling and considered whether this is due to degrees of painful vasospasm as distinct from cell damage. They found that as the thermal gradient is decreased in the tissue, pain decreased or 'adapted'. This may well be due to local vasospasm. Again, it was noticed that intense pain was produced by placing an epinephrine pack in the nose and that this pain was accentuated by breathing cold air. Relief was gained by breathing warm air. It does seem therefore that pain due to local cooling is initiated by vaso-constriction causing either deformation of the perivascular pain plexuses or injury due to anoxia in the tissues of supply.

It is important to realise that the source of a noxious stimulis plays no part in altering the quality of the pain pattern except that different degrees of tissue damage may follow the initial injury. For instance, prolonged prick from a needle will produce a burning pain which will cease when the needle is removed whereas burning pain resulting from prolonged contact with a hot instrument may continue after the removal of the hot instrument because of extensive tissue damage (34).

Wyke refers to the wide variety of chemico-physical changes e.g. osmosis which may evoke dental pain while having little action on the muco-cutaneous pain network (184).

This may be simply a physical phenomenon - the rapid withdrawal or attempted transudation of fluid from the pulp via the tubules causing mechanical stimulation of the peri-odontoblastic plexuses.

Other forms of stimulation can only be explained on the assumption that the odontoblastic process is a specialised receptor organ which responds to a complex variety of stimuli and which transmmts non-specific impulses to the pain fibres of the pulp, e.g. the light stroking of an area of denuded cervical dentine.

Although a wide range of stimuli applied to dentine causes pain this does not necessarily imply that the pain fibres of the pulp will respond to direct stimuli other than mechanical deformation or chemical agents as described above.

Cutaneous Hyperalgesia:

Of all the cutaneous sensibilities pain is recognised to be the sensibility most easily disturbed, especially in the direction of increase (100). This heightened perception of pain is termed "HYPERALGESIA" and may be defined as a state in which ordinarily non-noxious stimuli become capable of inducing pain or in which pain of greater intensity than normal is induced by noxious stimuli (180).

In this section, hyperalgesia due to a lowering of the pain threshold in cutaneous areas only will be discussed.

Several examples of locally produced cutaneous hyperalgesia may be quoted. The possible mechanism which produce this height-ened sensibility (and often spontaneous pain) will then be considered:

(i) Irritation results in pronounced lowering of the sensory threshold (171). A strong ..... current of an intensity to produce almost unbearable pain is applied to the skin for

5 mins. The surrounding skin subsequently becomes sore and tender. In the affected area the sense of touch is unaffected or diminished but a needle prick produces an intense diffuse, long lasting pain. The skin is excessively sensitive to slight friction and certain degrees of spontaneous burning or smarting may be present. This cutaneous hyperalgesia increases in intensity, reaching its maximum in about 20 - 30 mins. It also spreads progressively up and down the skin and may ultimately involve an area of some 20 sq. ins. The tenderness persists for hours or longer (183).

(ii) Other local tissue changes such as sustained hyperaemia and inflammation lower the effective strength of stimulus required to induce pain (180, 100, 35, 77). Following the exposure of skin to sunlight redness and tenderness develops. Friction of clothing against the skin is painful and a sense of burning may be apparent. Lewis terms this cutaneous tenderness, the 'erythralgic state' and states that it may be excited by scratching, burning with molten wax, freezing, and ultra violet light. "Skin so affected responds to very light stimuli, the response being of unusual intensity, and it possesses a certain diffuseness though it is never referred to a remote region". A frictional movement elicits unpleasant burning during the act, it subsides almost at once but it is followed by recurrent pain after a clear interval of 10 - 15 secs, lasting 1 - 3 mins. (100). A lowering of the pain threshold as much as 50% may be found in erythematous skin.

(iii) The hyperalgesia of peripheral neuropathy is quoted by Wolff (180). Here hyperalgesia seems to result from a differential effect upon the thresholds of the two types of cutaneous pain. The threshold for the slowly conducted burning pain may be greatly lowered, when that for the more rapidly conducted pricking pain is elevated so that the latter sensation is partially or completely abolished. In Fig. 43 is contrasted the normal relationship of the thresholds for pricking and burning pain under normal conditions with the thresholds during hyperalgesia after injury to nerves.

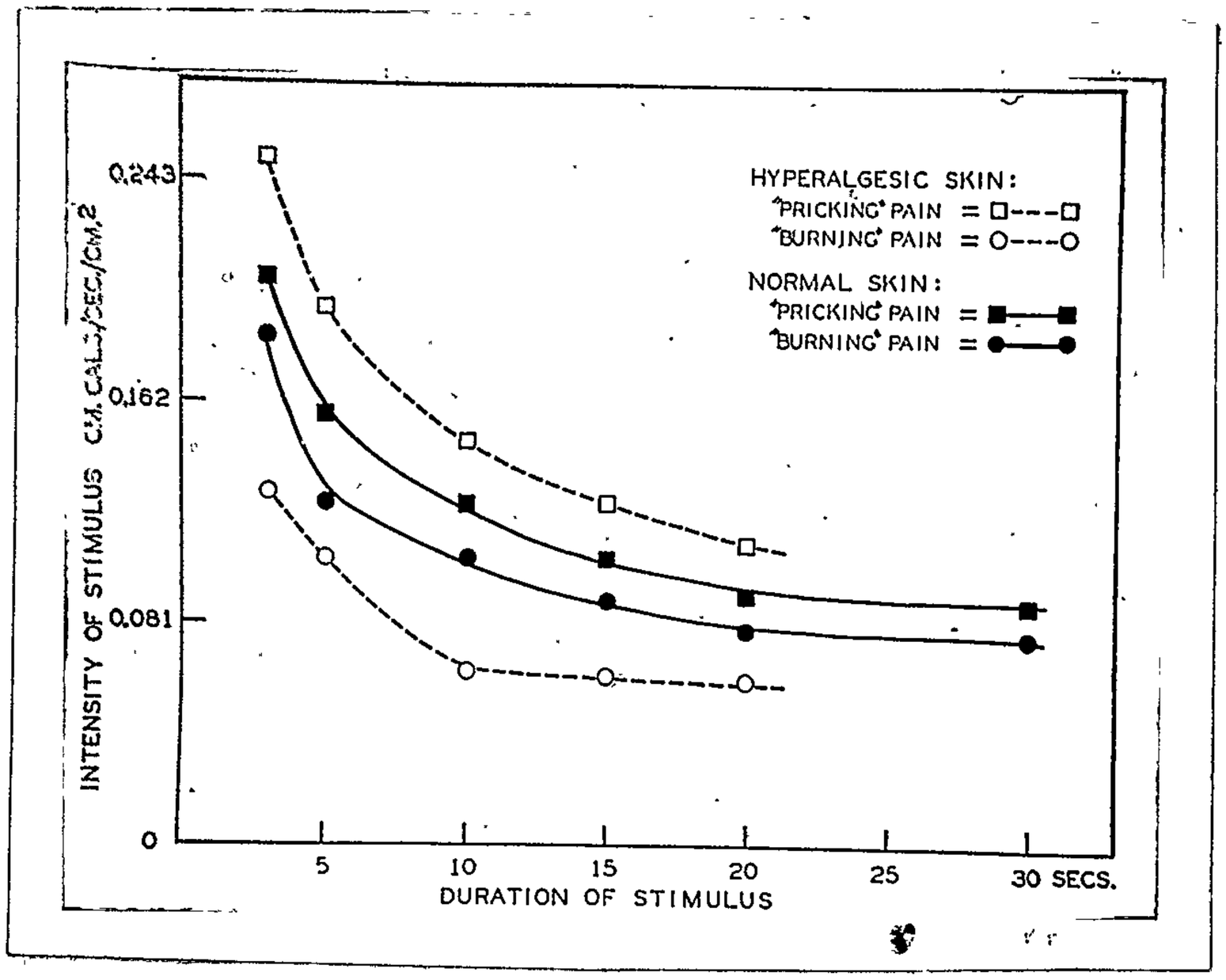


Fig. 43 - Comparison of thresholds of 'burning' pain to 'pricking' pain in a patient with hyperalgesia as a result of radiculitis.

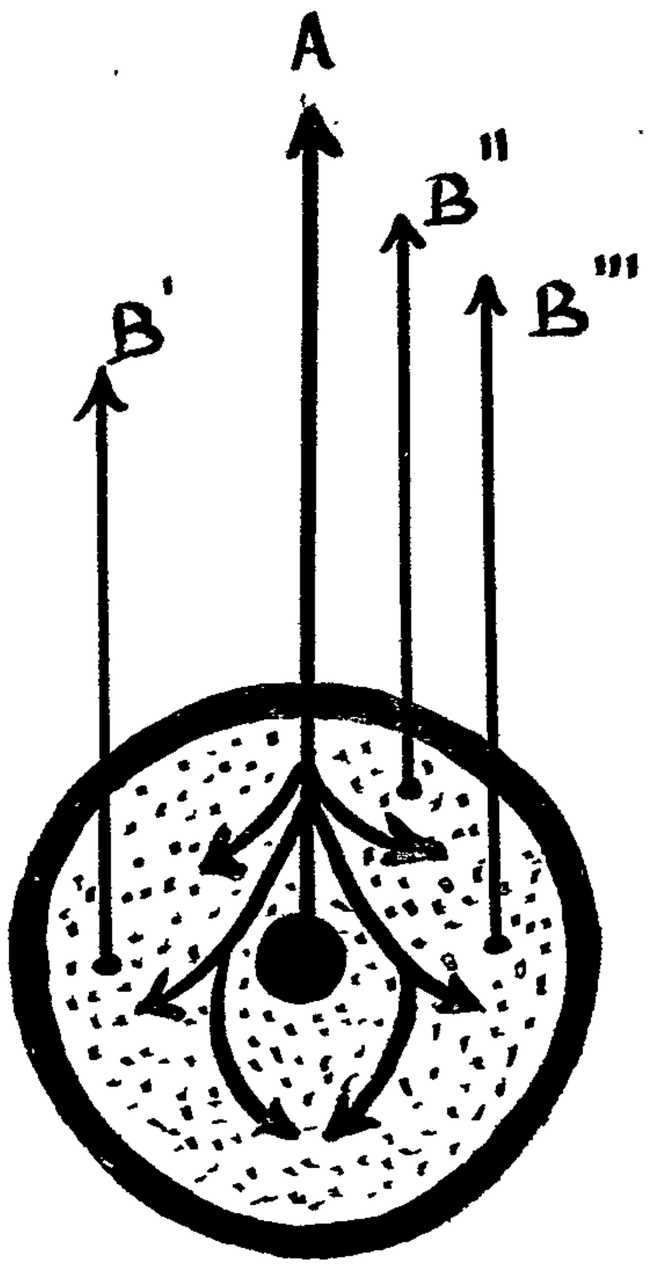


Fig. 44. Possible neural basis of cutaneous hyperalgesia. (p160)

In such cases ordinary innocuous stimuli are perceived as painful; even contact with bedclothes may result in burning pain whereas sensibility to prick is impaired (171). The same phenomenon has been shown to occur with progressive asphyxia of nerves induced by interruption of the circulation to an extremity prior to the development of complete anaesthesia; also in disease of the thalamus and spinal cord e.g. syringomyelia.

This effect also apparently occurs in other types of neuritis and following injury to nerves (180). Lewis confirms that stimulation of a nerve trunk produces hyperalgesia which may be maintained for hours, a day or longer. This hyperalgesia may be less obvious than the erythralgic tenderness of the injured skin.

Again, after section of a cutaneous nerve, the skin around the anaesthetic area shows increased sensitivity, to friction, i.e. it shows true hyperalgesia. The response is rather diffuse and lasting, but not referred. Trotter and Davies (165) stated that this hyperalgesia lies outside the line of anaesthesia, and assumes a patchy distribution in the midst of the analgesia, "curiously tending to be distributed in the neighbourhood of subcutaneous veins". It lasts a few weeks but gradually vanishes. As long ago as 1909 these writers attributed this phenomenon to the presence of some irritating substance produced as the result of division and degeneration of the nerve.

Hyperalgesia may be due to <u>irritating chemical products of injury</u> from the tissues. Lewis (100) states that "when a tissue (in a state of hyperalgesia) is injured, the immediate stimulus is direct, while the second, more prolonged pain, is due to the action of certain tissue substances upon the pain nerve endings, the latter being rendered hyperexcitable". The presence of these chemical irritants seems borne out by the observation that the recurrent pain is prolonged by circulatory arrest.

Samsom Wright (183) elaborates on this theory - "When the

skin is injured, chemical substances are released which stimulate the local pain endings. Impulses consequently pass up along afferent nerves to the central nervous system and give rise to the initial sensation of localised pain. It is known that the cutaneous afferent nerves branch in the skin to supply blood vessels in the vicinity. It is suggested that in addition, other branches are given off which ramify over a considerable area ending throughout in the skin cells. It is thought that when impulses along these filaments reach the skin cells they release some stable and unidentified chemical products which modifies the behaviour of the local cutaneous nerve endings, e.g. those connected with B_1, B_2 and B_3, so giving rise to abnormal states of skin sensibility". (Fig. 44).

"We seem to be dealing with a neural transmission of a chemical disturbance. That is, a chemical change set up in a discrete area of skin by an injury, setsup nervous impulses, which in their turn produce a long lasting chemical disturbance in a related wide skin area. In the latter zone, the local pain nerves register a skin condition which is not directly produced by local external stimulation".

Spontaneous Pain:

As has been stated, in hyperalgesic skin, pain may be felt in response to a stimulus which ordinarily gives none. For instance, when skin is inflamed and the pain nerves are hyper-excitable, stretching is a most effective stimulus for pain.

The effect of pressure of tension in producing spontaneous pain can also be seen in an injured limb, where increased tension in the tissues caused by hydrostatic increase in the pressure within the vessels increases the painful response.

Again, in injured tissue, a temperature of 29 - 32° and above may produce spontaneous pain.

The reason why a slightly inflamed tooth pulp gives rise to severe pain spontaneously or in response to slight stimuli is now clear.

The tooth temperature is normally 32° and consequently

if injury to a tooth by trauma or caries is severe enough, hyperalgesia may develop and reach such an intensity that normal body temperature will produce spontaneous pain. Again, it can be seen that the build up of pressure due to oedema or inflammation in the dental pulp can cause considerable pain due to the rigid confining walls of the pulp chamber. This is similar to the intense pain accompanying pressure on a boil before pus has issued from it.

In such cases, pain is frequently intensified by the pressure and increased tension induced by each systolic beat of the heart. Thus we have the pulsating toothache, or pulsating headache, which may become more dominant in the reclining position.

In summary it may therefore be stated that injury or inflammation alters the threshold to pain and slight degrees of tension and grades of heat within the range of normal body temperature becomes adequate in producing pain. Hyperexcitability is probably produced by released substances playing upon the nerve endings. Whether these irritants substances diffuse widely from the area of injury or whether hyperalgesic spread is the result of neural transmission of the chemical disturbance locally is a matter of speculation.

An Analysis of Deep Pain:

It is now necessary to consider the differences between cutaneous and deep pain. Superficial pain is pricking or burning whereas pain primarily from mesodermal tissues, e.g. muscle, fascia, periosteum, blood vessels or joints is aching in quality (34). Deep pain is generally poorly localised (35, 50), and characteristically spreads to be felt in areas other than those stimulated. This tendency of deep aching pain to spread to associated neural segments is described as 'referred' pain.

Deep pain is non-specific in quality and this is illustrated in the following experiments (174).

Attempts were made to differentiate in the same person qualities of pain (a) in headache resulting from intravenous histamine causing noxious impulses from cerebral artery distension; (b) in migraine headache emanating chiefly from distended branches of the external carotid; (c) in headaches due to venous traction arising after spinal fluid drainage and (d) in headache resulting from injection into the frontalis of a hypertonic salt solution. Although these headaches varied as regards site, pulsatile or non-pulsatile nature the quality of the pain was reported to be same in all. It was of a deep aching, diffuse nature, quick to arouse reaction in muscles and in glands such as lacrimation and injection of the eyes, and was associated with feelings of nausea. The pain was indistinguishable in quality from that which results from painful stimulation of the teeth, periosteum or the muscles of the eyes.

Although aching pain implies deep pain and pricking and burning pain implies superficial pain, Cohen points out that all pain cannot be clearly divided into these two groups. Many mixed types may occur, for a lesion may involve both superficial and deep structures.

The phenomenon of pain reference has been clearly illustrated by Lewis and also be Goodell and Wolff who stimulated the ostium and mucous membrane of the maxallary antrum.

Both investigators showed that following brief stimulation nothing could be felt except a brief intra nasal pain. The sensation in the skin of the face was quite normal. Following prolonged stimulation however, pain spread over the side of the nose and cheek along the zygoma. This hyperalgesic area then spread into the temporal region, into the upper teeth and the skin of the upper lip (Fig. 45). The hyperalgesic state is at its height after 40 - 60 mins., and may continue until the next day. The effect is noted whether the ostium, the mucous membrane of the antrum or the nerves to the incisor or molar teeth are stimulated. The area becoming hyperalgesic is the 2nd division of the 5th nerve in this case, and while Lewis did not observe spread beyond this midline or invasion of the 1st and 3rd division territories, Goodell and Woolf found involvement of adjacent portions of the other divisions. This widespread area of pain had a deep aching quality which became so intense as to minimise the pain from the nose. This was associated with flushing, lacrimation and photophobia and upon rolling the eyes, a little unilateral pain was felt momentarily in the orbit (174, 100).

Deep pain therefore, may become falsely localised, being experienced far from the site of stimulation. That is, depending upon the intensity and duration of the painful stimulation, the pain may be localised within the same division or it may spread so that it seems to emanate from peripheral structures innervated by adjacent divisions or segments.

Associated with deep pain are the following phenomena:-

i) Deep tenderness.

ii) Faulty localisation and spread of pain to deep and superficial structures in areas remote from the site of stimulation.

iii) Surface hyperalgesia and hyperaesthesia.

iv) Skeltal muscle spasm and smooth muscle and glandular effects.

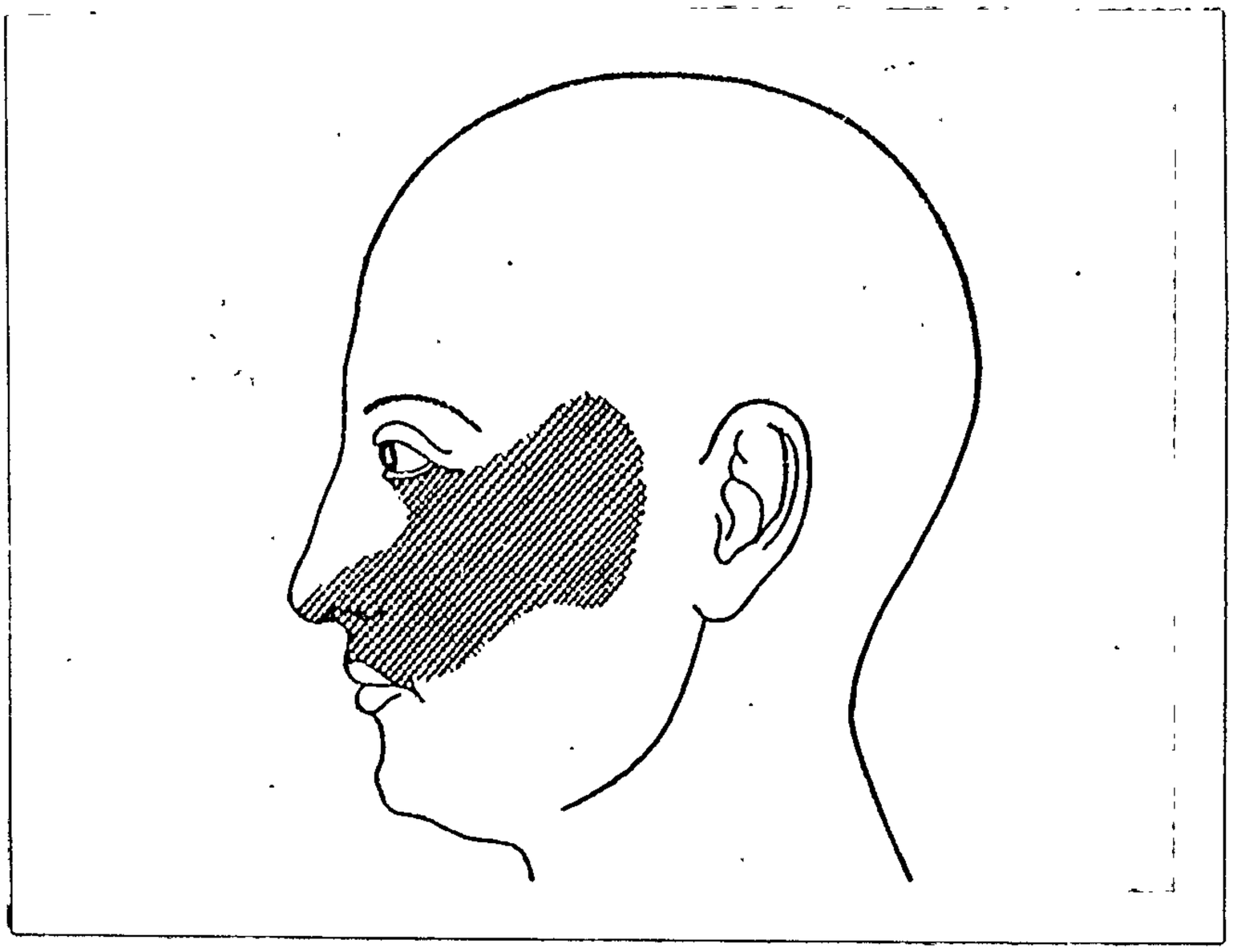


Fig. 45. Area of hyperalgesia developing on stimulation of the mucous membrane or dental nerves within the maxillary antrum (Lewis).

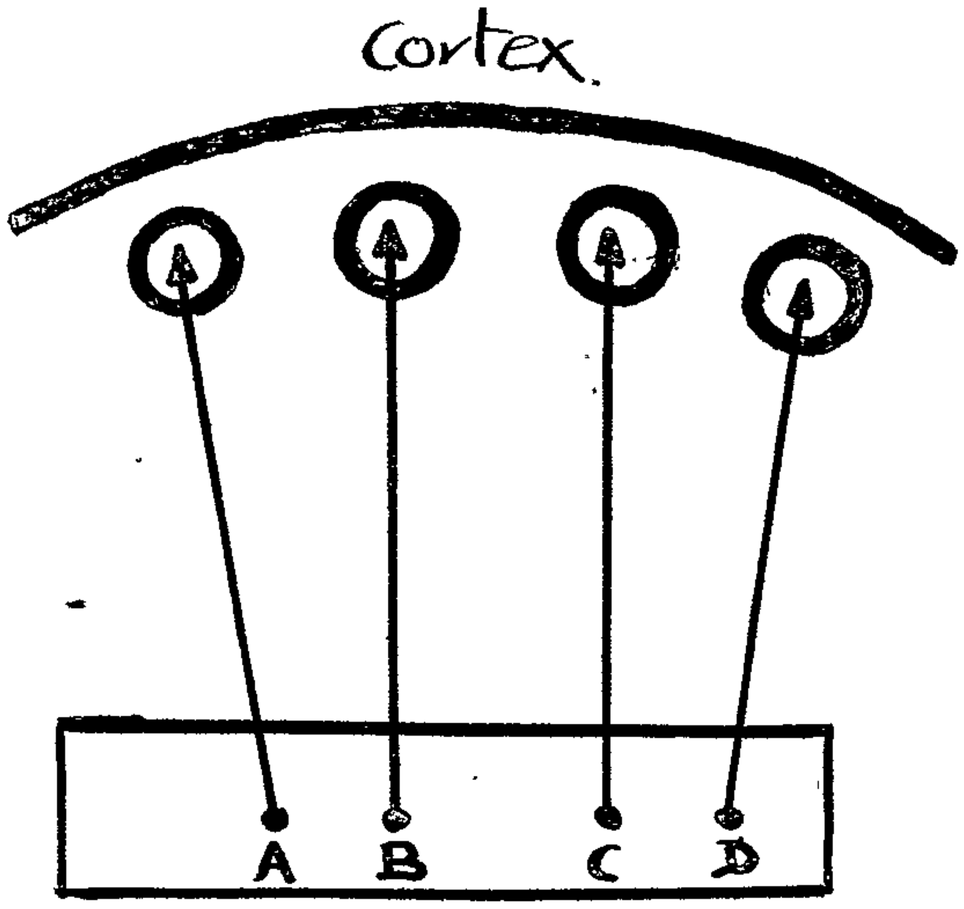
The following sections will be devoted to an analysis of the above phenomena.

Referred Pain:

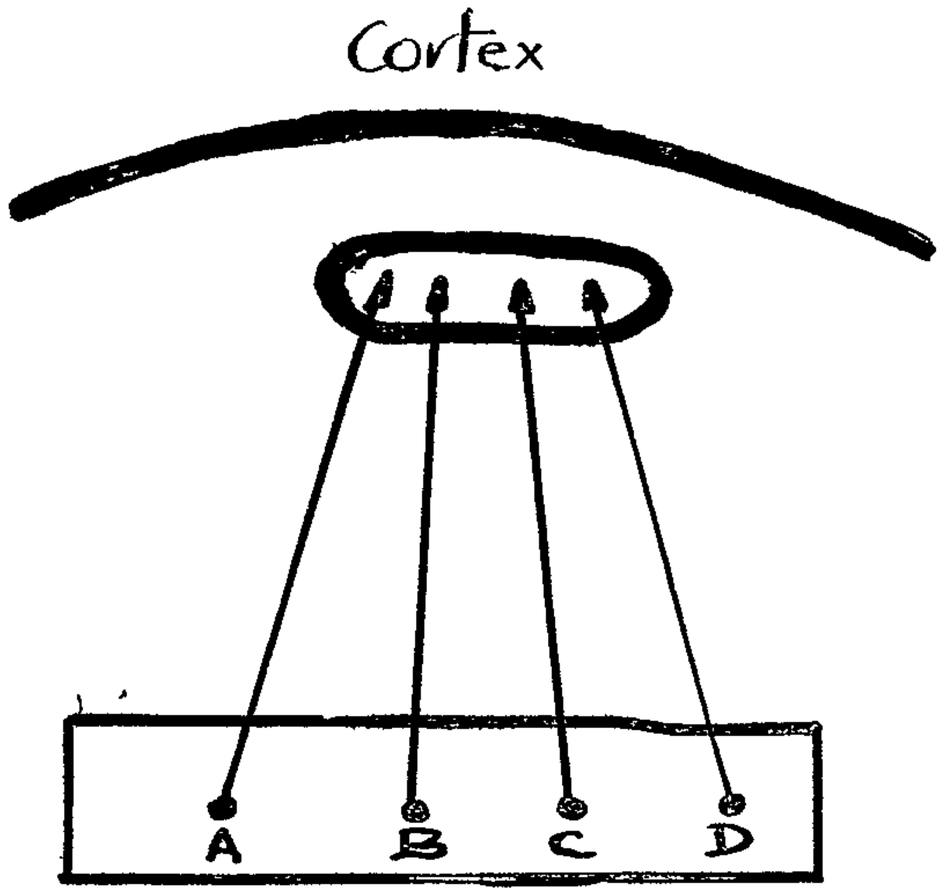
Deep pain is essentially diffuse and poorly localised and arises from structures of which we are ordinarily little conscious. The reason for such poor localisation is not difficult to explain. It has been stated that the degree of accuracy of localisation of a stimulus depends upon the wealth of innervation of the tissues concerned, the extent of the cortical area receiving impulses from the region, and the degree of topographical projection of the receptor surface onto the discrete areas of the sensory cortex (183). If the sensory surface is sparcely innervated or if many receptors from an area converge upon a single cortical point, localisation is correspondingly impaired (Fig. 46). Ability to localise somatic sensation seems to depend upon the development of a cortical pattern of the body image in the parietal lobes. Each person on the basis of his past sensory experience creates in his cortex a sensory image of himself. Perhaps through lack of experience with internal stimuli, together with the relative paucity of nerve endings in deep somatic structures no such visceral representation is ever built up and the individual is prone to misinterpret the source of impulses from these areas (171) (205). Oliver feels that in the persistence of this 'body image' lies the explanation for 'phantom limb' pain. Perhaps a similar phenomenon occurs when a patient is convinced that pain comes from the region of a previous extraction.

Diffuse pain from deep structures tends to be projected to a region where pain is usually well localised and innervated by the same spinal segment as the structures being stimulated i.e. deep pain tends to be "referred".

The term "referred pain" was first used by Head in 1893 in relation to visceral disease. Dana preferred the term "reflex" or "transferred pain" while Waterston suggested the terms "homotopic" and "hetrotopic" to indicate pain felt locally or at a distance (100).



Accurate localisation possible.



Accurate localisation impossible.

Fig. 46. Neural Basis of Accurate localisation and diffuse sensation.

Accurate localisation of a stimulis depends on the connection of peripheral nerve endings in a point to point manner with 'private' groups of cortical cells. If however, the peripheral receptors A, B, C and D are connected with a common single group of cortical cells no distinction between them is possible, and localisation is poor.

Referred pain may be described as pain resulting from stimulation of a somatic sensory nerve and referred to a remote part of the distribution of that nerve or of the segmental sensory distribution with which it is connected.

Many theories have been forwarded in an endeavour to explain the mechanism of referred pain. Certain facts are worth repeating before outlining these theories.

When pain is falsely projected superficially, it is to an area which has a somatic sensory nerve supply common with the stimulated deep somatic structure or which are closely associated anatomically in their central connections.

Referred pain may be present when pain is absent from the site of noxious stimulation, but it may be eliminated by local anaesthetic infiltration at the site of noxious stimulation (35).

Some examples of pain reference will be considered:

When ice is applied to the roof of the mouth, frontal headache develops, i.e. noxious stimulation of structures supplied by one division of the 5th nerve causes pain to be experienced in structures supplied by another division. Also when ice is applied to the fossa of Rosenmuhler and the posterior wall of the pharynx pain is experienced in and behind the ear-areas all connected with the 9th and 10th cranial nerves.

In coronary artery occlusion, or angina, the pain at first seems to emanate from one or more of the anterior portions of the first 4 - 5 thoracic segments and spreads headwards so as to seem to arise within the structures supplied by the lower and upper cervical segments. Ultimately it may seem to emanate from the lower jaw and teeth. (It is interesting to note at this stage that the descending nucleus of the 5th nerve and the cervical dorsal horn are contiguous in the upper cervical cord (174).

A further observation is the hyperalgesia and surface hyperaesthesia which often accompanies referred pain, i.e. noxious stimulation of an area of referred pain is often

productive of more discomfort than similar excitation of an uninvolved region. For example, angina may leave behind it tenderness in the skin of the chest over the left side.

Toothache may similarly produce hyperalgesia of the skin of the cheek. Robertson et al (126) stimulated an upper tooth electrically with a 60 cycle alternating current which was briefly interrupted every few seconds. Stimulation at a strength of 1 volt for 10 mins., caused very severe pain, apprehension, lacrimation, tachycardia and sweating. The severe pain subsided almost immediately at the end of the stimulation, but 5 mins. later there was moderate disconfort over the upper half of the face with a sensation of fullness, numbness and stiffness, of the jaw. This in turn died away, but was succeeded by headache in the same area which was associated with reddening of the conjunctiva, photophobia and tenderness of the masseter and temporal muscles. At this state there was marked hyperalgesia to pin prick applied to the temple. Electromyograph recordings showed that there was no abnormity in tension or spasm of these muscles, i.e. painful muscular spasm was not in this case the primary cause of the pain.

Not only the mechanism of referred pain must be considered but also the manner in which hyperalgesia in the areas of reference is produced.

Theories of Referred Pain:

1. Referred pain has been attributed to the branching of the peripheral axons of the dorsal roots which convey the impulses set up by noxious stimuli (Sinclair quoted by 183, 171).

It is known that axons branch extensively in the skin mediating the vascular axon reflex which is responsible for the flare. A similar ramification has been cited as responsible for the cutaneous hyperalgesia which develops round an injured area of skin (page 160).

It has been suggested by some writers that dorsal root axons divide more proximally with branches to visceral as well

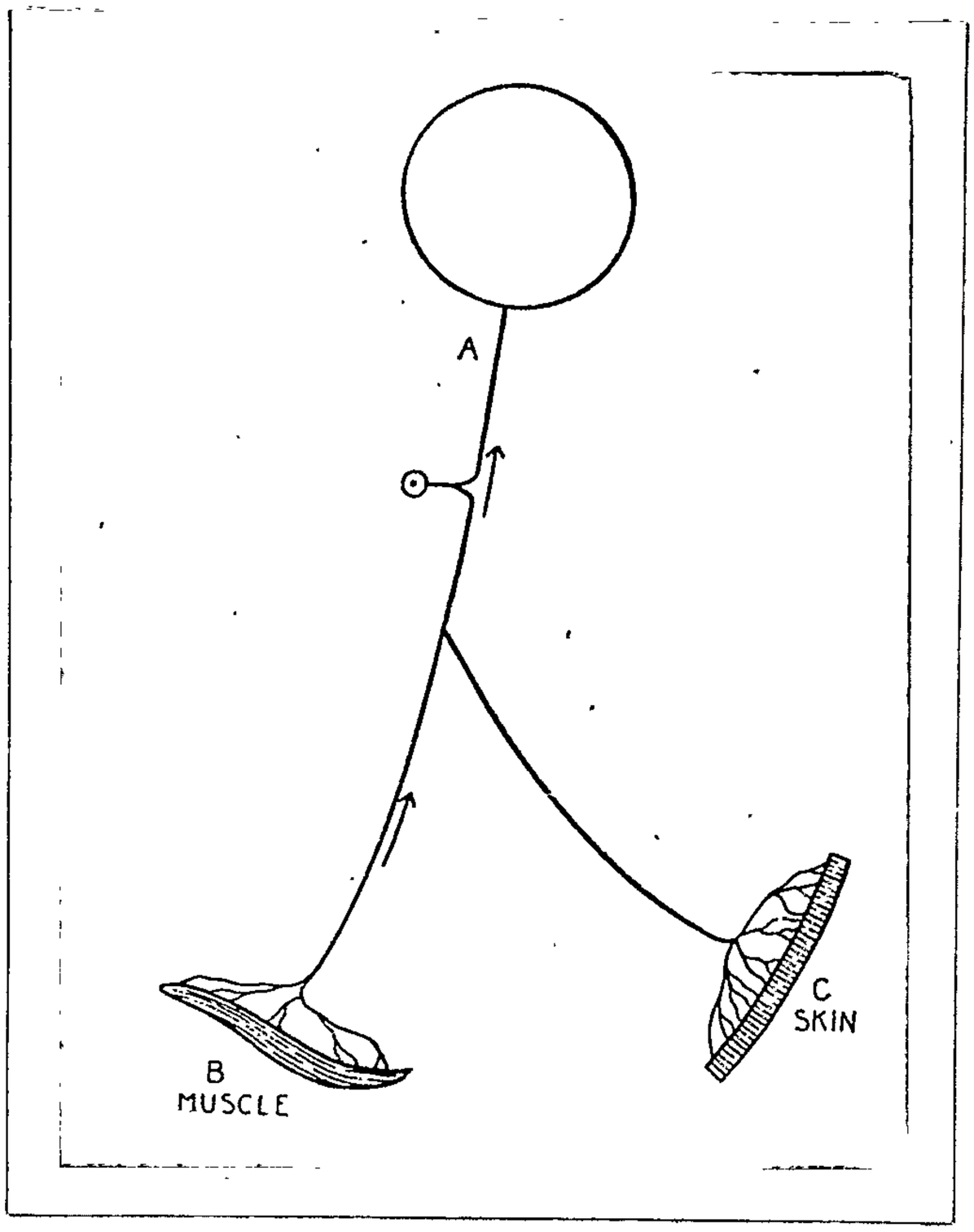


Fig. 47. Sinclair, Weddell & Feindel's concept of central misinterpretation of visceral pain: The "branched axon" theory.

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Impulses passing up the common stem A of the branched axon from the muscle B, are interpreted by the receiving centre as coming from the area of skin C.

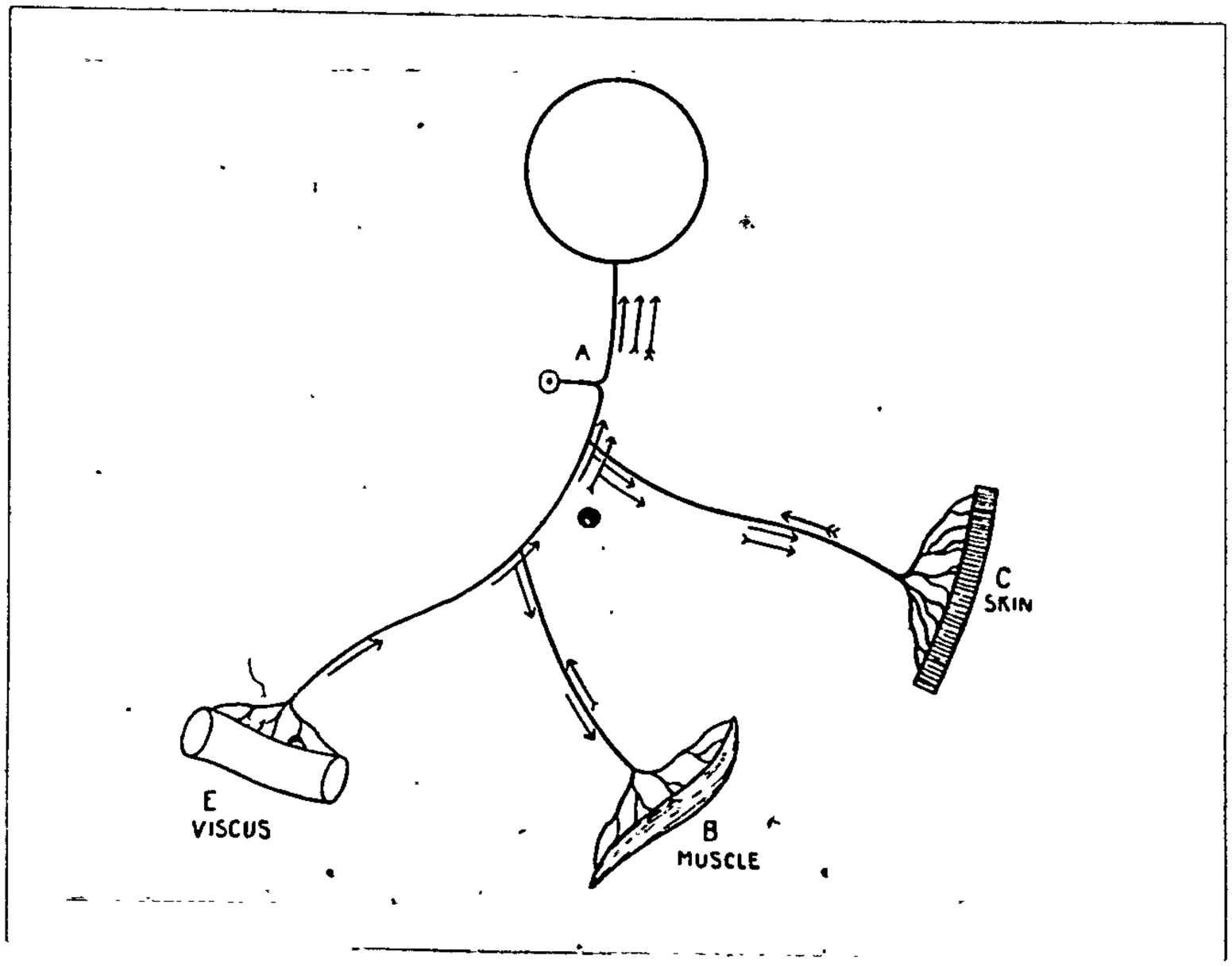

Fig. 48. Multiple reference from a viscus: The arrows indicate the pathways traversed by the impulses set in train from a focus in the viscus E. The different flighting of the arrows indicates the different origins of the impulses they represent.

(Quoted by White and Sweet)

as to somatic structures (Fig. 47, 48). Although branching axons have been demonstrated supplying skin and gut in fishes by Wernoe (1925), this view requires anatomical verification in man.

It is nevertheless an attractive hypothesis, for it could explain not only the feasibility of impulses arising from a viscus being referred to somatic structures, but also the fact that such referred pain may sometimes be felt in the skin, or in deeper structures or in both.

2. Weisengreen (169) forwards the view that constantly irritated and stimulated nerve fibres may stimulate other fibres in close proximity ....."we are justified in assuming that where sensory nerve fibres meet whether en route or in ganglia, they may stimulate one another". In this way he explains the phenomenon of pain reference to divisions of the 5th nerve via the gasserian ganglion.

Although experimentally it has been demonstrated that one fibre may, under certain conditions induce an impulse in another (Winton and Bayliss) this does not seem to be a physiological occurrence.

Sicher (142) most emphatically refutes this theory. "Whatever the cause of referred pain, it is not a result of peripheral nerve connections ..... the function of the entire nervous system rests on the independence, isolation and insulation of all nerve fibres, and induction of impulses to the neighbouring fibres is entirely impossible. The mechanism of referred pain has to be sought in central neuronal connections".

3. The 'painful scar' theory was elaborated by Hutchins and Reynolds (125) on the basis of Crile's writings in 1916 (82) These authors were originally concerned with the pain of aerodontalgia, and sought to explain the reason why pain was commonly experienced in teeth which had previously received trauma or were in a subclinical diseased condition, when associated structures or the nerve trunk to the area was stimulated.

While this hypothesis deals specifically with poor localisation of dental pain, it is interesting to mention it under this section.

Hutchins and Reynolds state: "The afferent nerve of a tooth, after once being painfully stimulated by deep caries, cavity preparation or infection, continues to react by means of a constant series of excitation waves for a considerable period (even years). After the original external stimulus has disappeared, the excitation subsides to a subliminal level. No pain is experienced in the tooth until stimulation of an associated structure, innervated from the same segment produces a facilitation of the excitation existing in the afferent nerve, sufficient to carry this excitation to the central nervous system where the subjective sensation of pain arises".

While the concept of excitation waves continuing from teeth for years may be questioned, this theory bears many similarities to the following theories.

4. <u>The dominant receptor zone theory</u> (Fig. 49)

It is supposed that receptor zone R1, R2 both terminate in the same cortical area C. Zone R1 is frequently stimulated and the mind has consequently learnt to project the sensation to the region actually stimulated. Zone R2 on the other hand, is rarely or never stimulated. Whether zone R1 or R2 is stimulated, it is supposed that the mind always projects the sensation to zone R1. That is, impulses from zone R1 are correctly localised but impulses from R2 are falsely projected or referred to zone R1.

5. A similar explanation to account for the misrepresentation of painful impulses which reach the spinal cord from deep somatic structures and are erroneously referred to the skin has been suggested by Ruch in 1949. This theory states that there is a <u>convergence of visceral and cutaneous afferents</u> on the same spinothalamic (or spinal tract) neurones shares in common by both sets of fibres (Fig. 50). Sicher (142) tends to support this view ".....groups of neurones activated by visceral and

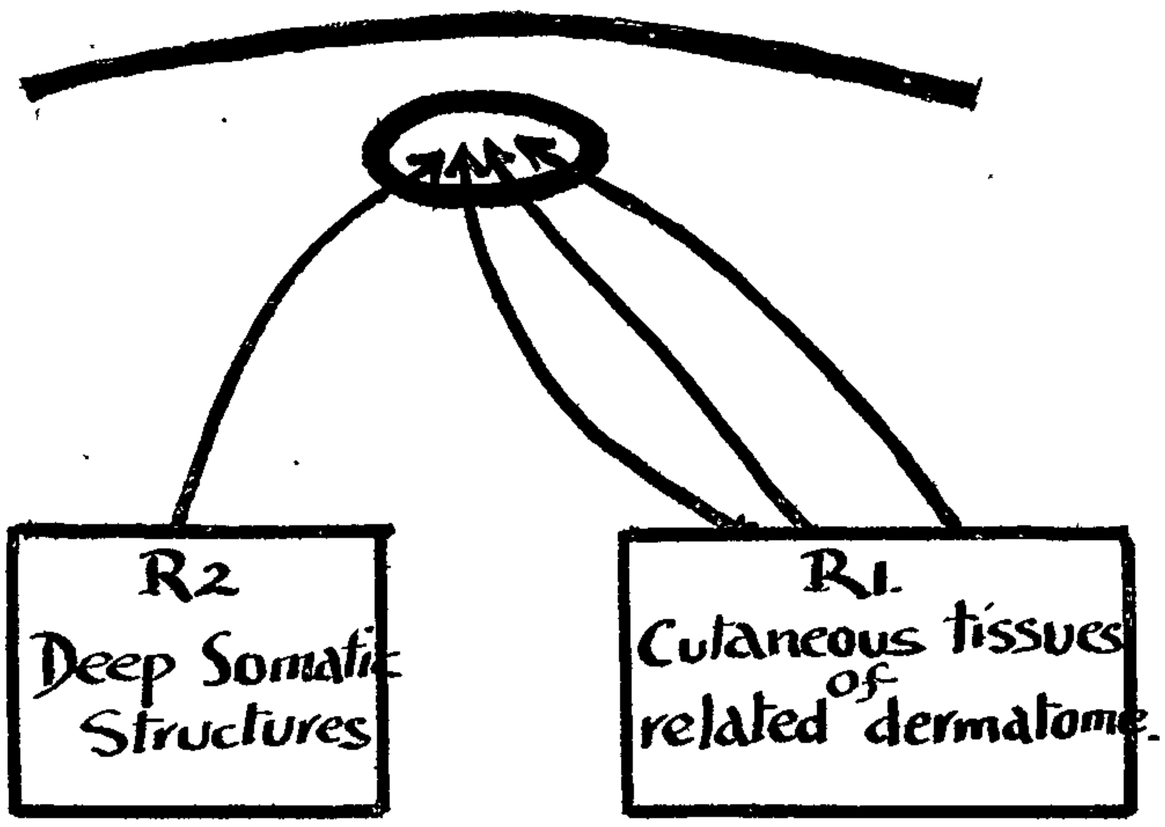

Fig. 49. Dominant Receptors Zone Theory

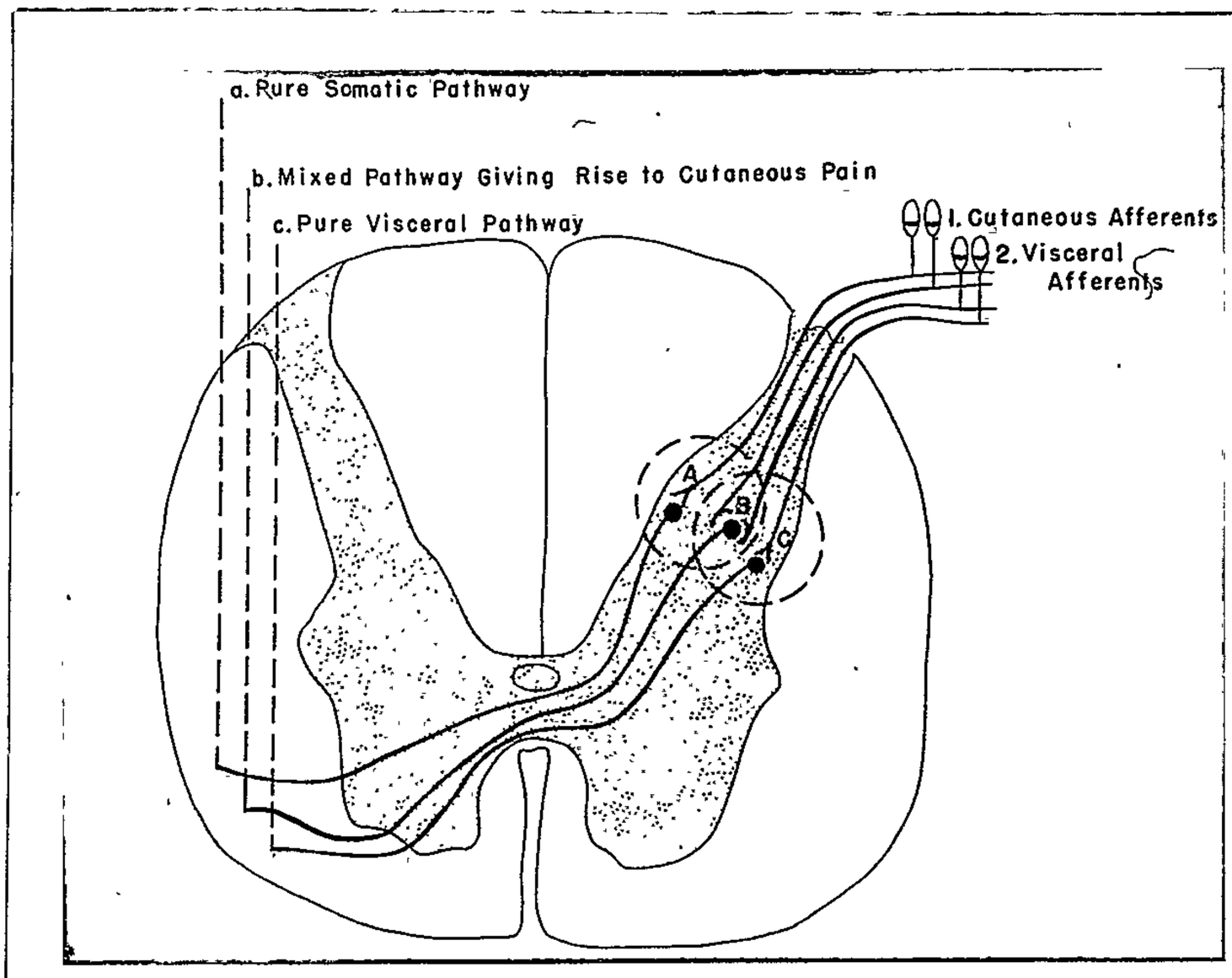

Fig. 21. Ruch's concept of central misrepresentation of visceral pain:
"Convergence-projection mechanism of referred visceral and somatic pain based upon Sherrington's neuron-pool concept. A, B, C represent a neuron pool consisting of all the spinothalamic tract fibres originating in one segment of the spinal cord. A is the field of neurons having connections only with afferent fibres from cutaneous sense organs. B is the field of overlap constituted by neurons which receive impulses from *both* visceral and cutaneous afferents, and impulses in b will give rise to pain referred to the skin. C are those neurons of the pool which connect only with afferent fibres from the visceral cavities, and give rise to unreferred or true splanchnic pain. Only one neuron in each category is represented . . . a, b, c, are fibres in the spinothalamic tract having cell bodies in fields A, B, and C respectively."
Drawing adapted and legend quoted from Ruch in Fulton's *Textbook of Physiology*, 1946.
Courtesy, W. B. Saunders Co., Philadelphia.

Fig. 50. The theory of convergence of visceral and cutaneous afferents.

cutaneous pain fibres may overlap. Thus in visceral pain, neurones of the cutaneous group are subliminally stimulated and their firing by other subthreshold stimuli is facilitated".

Wright (202) agrees with Sicher and feels that more than one sensory fibre can affect an individual neurone ([illegible]). He reasons that intense irritation of a viscus causes pain referred to cutaneous areas by reason of these common neurones. He also feels that in some cases, impulses from the irritated viscus do not set up sufficient stimulation of the central nervous system to cause a feeling of pain, but that the reinforcement of these impulses by the normal impulses going from the skin, bring the degree of stimulation up to the level of perception of pain. In such cases, local anaesthesia of the skin to which pain is referred, will cause a disappearance of pain, while in cases of more severe irritation from the deep tissue, such an effect is not observed.

There are indeed many more pain fibres in the posterior roots than axons in the spinothalamic tract, but whether both visceral and cutaneous pain fibres converge upon common groups of cells and whether this common 2nd neurone ends cortically in an area which serves both zones (183, 171) is a matter of some speculation.

6. Mackenzie's theory of <u>hyper-irritable foci</u> within the spinal cord is one of the best known theories of visceral pain. This hypothesis states that impulses from a diseased insensitive biscus bombard the posterior horn and there set up an irritable focus, so that normal subthreshold cutaneous impulses break across the synapses to the secondary neurones, which traverse the spinothalamic tract and give rise to the sensation of superficial pain (Fig. 51).

If Mackenzie's theory were the entire explanation procaine infiltration of the cutaneous surface to which visceral pain is referred, would relieve it. This is not often the case. However, under certain conditions pain (see page 183) or hyperalgesia referred to cutaneous areas can be relieved by local anaesthesia. Also it is known that impulses coming in from the

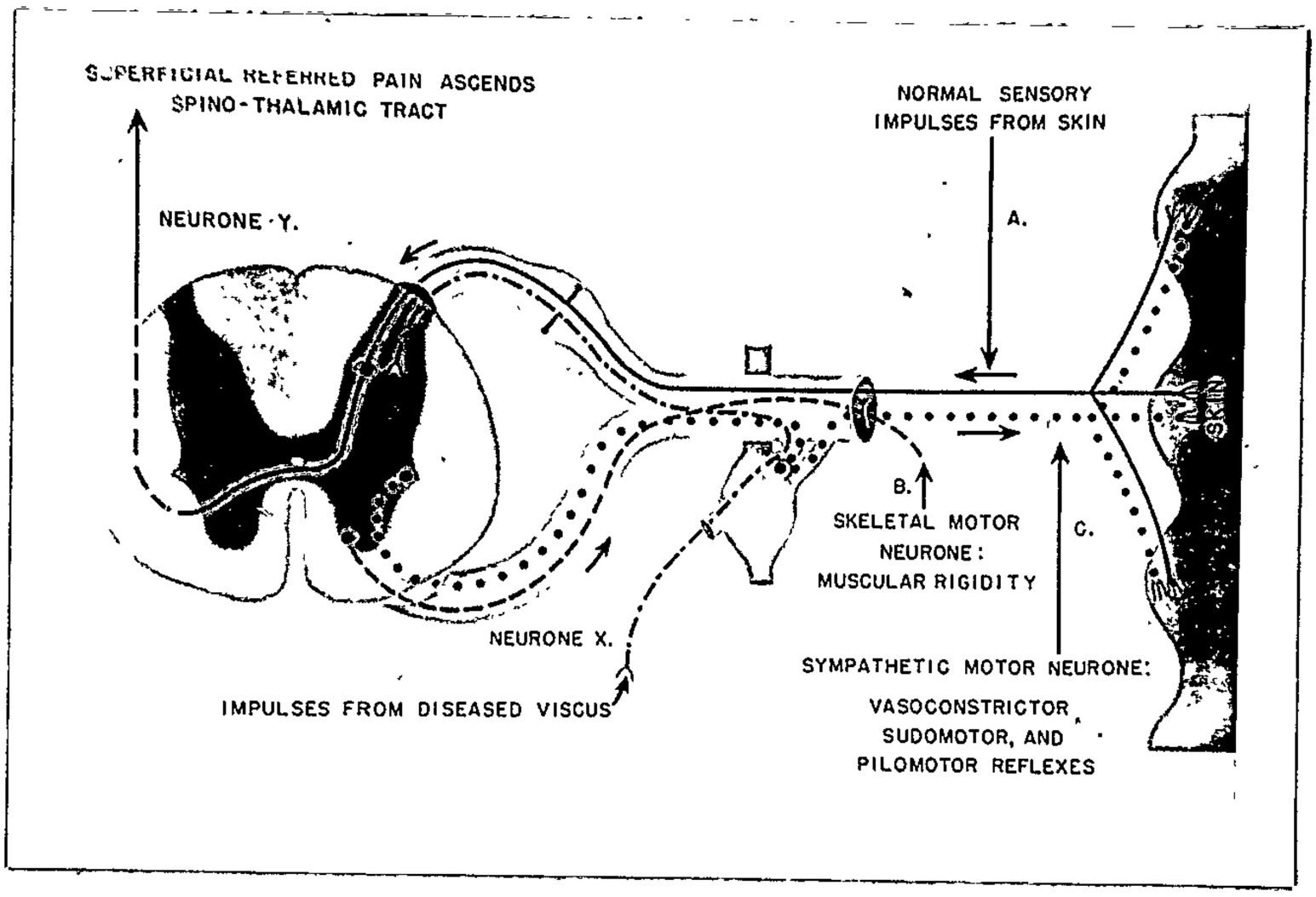

Fig. 51. Mackenzie's concept of central misinterpretation of visceral pain.

Impulses from a diseased viscus propagated over neurone x reach the posterior spinal horn via a white ramus communicans, spinal nerve, and its posterior root. On arrival in the posterior grey matter there is no synapse with a secondary spinothalamic neurone, but the visceral discharge sets up an "irritable focus". This lowers synaptic resistance between the cutaneous afferent (A) and its secondary fibre (Y) to such a degree that ordinarily subthreshold cutaneous impulses reach the thalamus and are appreciated as pain in the skin of the same spinal segment.

surface of the body may increase certain types of visceral pain.

Therefore, although Mackenzie's hypothesis is not the complete answer, it has provided a basis for further investigation.

7. It seems clear that referred pain in view of its reference to tissues supplied by the same or adjacent neural segments, is a central rather than a peripheral effect and the studies of Woolf and Hardy (80) in1947 seem more acceptable. They prefer the theory of a central excitatory state in the spinal cord evoked by a barrage of afferent nervous stimuli. They state that referred pain is "attributable to the spread of excitation in the neuraxis to the other portions of the same segment or segments adjacent to those into which the noxious impulses are conducted.

This gives rise to pain experienced in parts innervated by deep and superficial branches of the affected segments and a variety of motor effects".

These investigators postulated a network of internuncial neurones in the spinal grey matter which are intercalated amongst the neurones mediating impulses from visceral, deep somatic and cutaneous tissues. Intense bombardment of such a network from any of these sources may result in a spread of the disturbance to neighbouring spinal segments.

That extension, spread, or reference of deep pain is in all probability due to this spread of the central excitatory state within the posterior horn of the spinal cord or its cranial equivalent, seems to be borne out in the following observations.

In an individual who had undergone a unilateral interruption of the spinothalamic pathways below T_1, noxious stimulation of the anaesthetic half of his body induced pain felt diffusely on the opposite side. This illustrates how spread of excitation occurs within the cord from noxious impulses entering it even though the pathways which bring to consciousness the pain from the site stimulated have been severed (Fig. 52) (180).

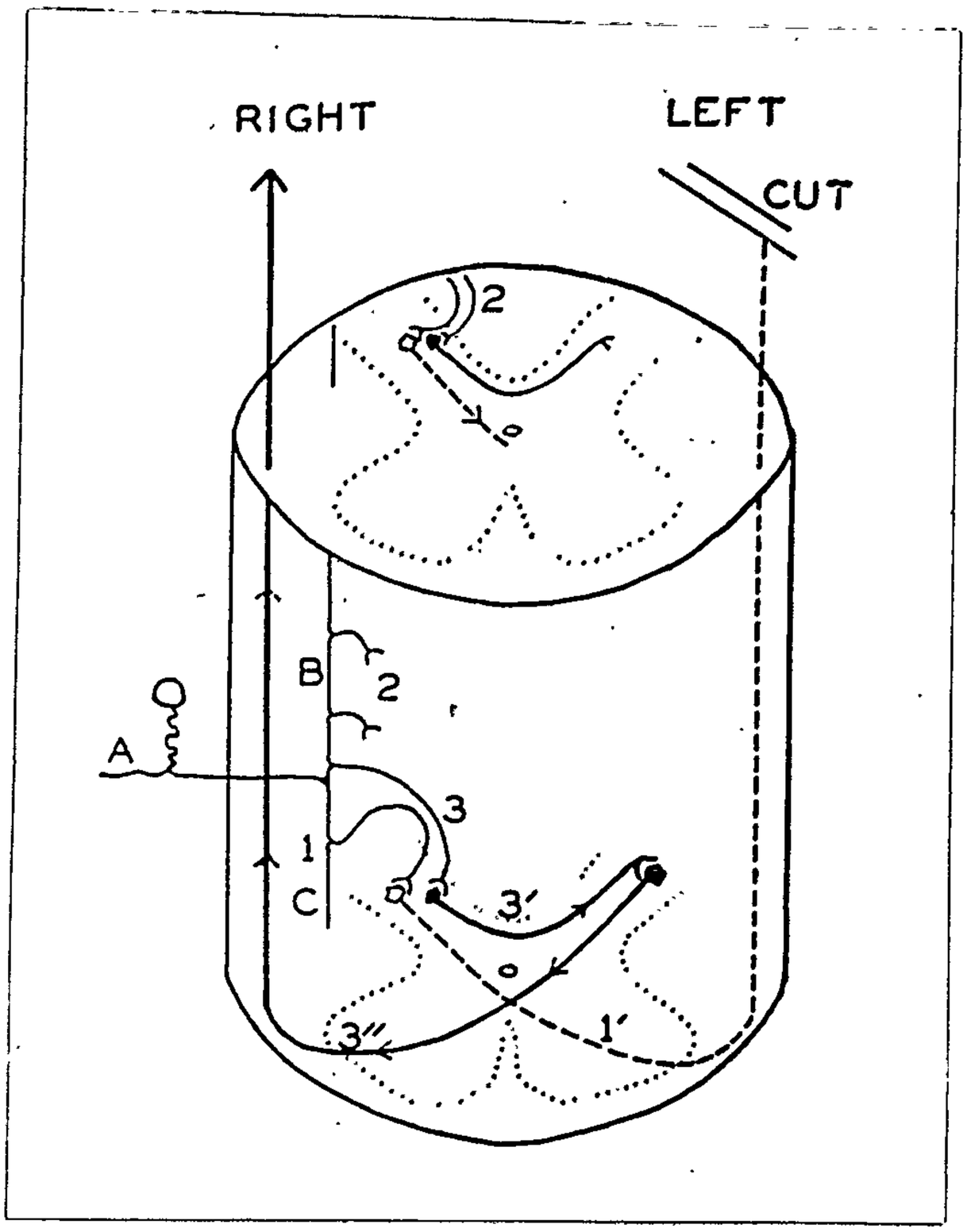

Fig. 52. Schematic representation of the pathways for the spread of pain within the spinal cord (Wolff and Wolf (180)).

A. represents the primary sensory neurone;

B. the ascending branch of the sensory radicle; and

C. the descending branch of the sensory radicle.

1. is a collateral to the posterior horn of the nearest spinal segment. This connects with neurone 1' which crosses to ascend in the opposite spinothalamic tract.

2. indicates collaterals to the adjacent segment; and

3. collateralls to the posterior horn, connecting with neurone 3', which forms part of the posterior commissure in crossing to the opposite posterior horn, where it connects with 3". This crosses back to ascend the spinothalamic tract on the side of entry of the noxious impulses.

In general Wolf found that the more intense the noxious stimulation the more widespread is the area of reference.

Again, spread of 'cold pain' - induced by immersing one digit for 10 minutes in water at 0^{o}c., to the neighbouring fingers, tends to support the above view. Procainisation of the digital nerves in the secondarily involved fingers did not eliminate pain. This should be the case if a metabolite were produced under the stimulus of antidromic conduction. Further, the pain does not appear to have developed quickly enough in other fingers to be accounted for on the theory of misrepresentation through branching of sensory fibres.

This phenomenon then, cannot be explained by MacKenzie's theory or by Sinclair's hypothesis of branching axons and antidromic metabolites. Spread of pain is apparently a central effect and does not depend in any way upon afferent impulses from the tissues into which the spread occurs (174).

Referred Pain and Associated Hyperalgesia:

The observations that surface hyperalgesia often accompanies deep pain reference was noted many years ago. Head in 1894 gave an elaborate description of areas of hyperalgesia appearing on the head and neck in diseases of the eye, ear, tongue, nose and other parts.

In areas exhibiting this surface hyperalgesia, pin prick may be experienced as sharper and of longer duration, and tactile and thermal sensations seem more intense than those induced by the same stimuli in other areas (180, 174). Such skin areas are innervated by the same or adjacent neural segments which supply the deep tissues from which the noxious and painful impulses are originated.

It has been stated that a possible cause of cutaneous hyperalgesia produced by painful skin stimuli is a neural transmission of the chemical disturbance locally and this concept has been applied by Sinclair to explain the surface hyperalgesia accompanying deep pain. Accepting the hypothesis of branching axons supplying both viscera, muscle and skin (183), it is

suggested that impulses pass centrally from the deep focus of irritation, along one branch of an axon and then antidromic impulses pass along a second branch to the skin. These set up a long enduring chemical change which enhances the sensitivity of the local nerve endings.

In explanation of the fact that surface tenderness and hyperalgesia does not always accompany referred pain, the above theory assumes that only in the later stages, when the stimuli have been acting some time, does the chemical factor (i.e. metabolites released as a result of antidromic impulses) become operable with production of hyperalgesia in the areas of reference.

This theory implies a lowering of threshold of the cutaneous nerve endings and the veracity of this assumption has recently been questioned. The anatomical illustration of branching axons supplying superficial and deep structures in man is also lacking.

Hyperalgesia in a tissue may be produced by lowering of the pain threshold locally or it may be due to other mechanisms whereby sensory impulses are exaggerated and intensified while a normal pain threshold exists (180).

Wolff et al (180, 174) state that it is extremely doubtful that there is liberated in the skin of the hyperalgesic areas associated with referred pain, a chemical which lowers the pain threshold. Observations on patients with such hyperaesthesia have shown that there is no significant lowering of the pain threshold. For example, a man with hyperalgesia on the right side of the face secondary to noxious impulses from Tic tissues was found to have the same pain threshold on the two sides of his face which was within normal limits.

However, a stimulus inducing a sensation which was reported as 1+ on the control cheek and forehead, induced a sensation which was reported as a 3+ pain on the hyperalgesic side of the face. This seems to be an intensification of the pain received rather than a lowered threshold (79).

Lewis (100) seems to have made this observation when distinguishing between tenderness of the skin resulting from changes in the skin itself and tenderness resulting from pressure on the nerve roots.

He noted in the former case that nerve endings are rendered hyperexcitable to stimulation. The skin responds painfully to warmth at 40°c., and friction produces not only immediate pain but recurrent pain after an interval of many seconds which lasts.

In areas of pain reference from deep stimulation however, the tender skin is not painful to warmth at 40°, and while pain to friction does occur, recurrent pain is not observed.

While Lewis tended to regard these hyperalgesic states as due basically to the same mechanism (page 159) differing essentially in degree, it may also be reasoned that in the former case the threshold is lowered while in the latter case the threshold to pain is normal the response to a threshold stimulus merely being exaggerated.

The two hyperalgesic states therefore appear to be produced by different mechanisms.

Wolff et al (180, 174) consider that this intensification of sensation in the hyperalgesia of referred pain results from a change within the nervous system. It is suggested that the central excitatory effects due to a pre-existing barrage of noxious impulses in the segmental or suprasegmental neural apparatus alter the situation so as to make impulses originating at the usual threshold in the skin seem to be more than usually intense and persistent.

This theory seems quite acceptable in explaining the phenomenon of referred pain and associated hyperalgesia and hyperaesthesia. It explains why, in an area of skin involved in a process of referred pain, not only does pain sensation arising from noxious stimulation appear intensified, but so do all sensations arising from other sensory stimuli (Fig. 53) (180).

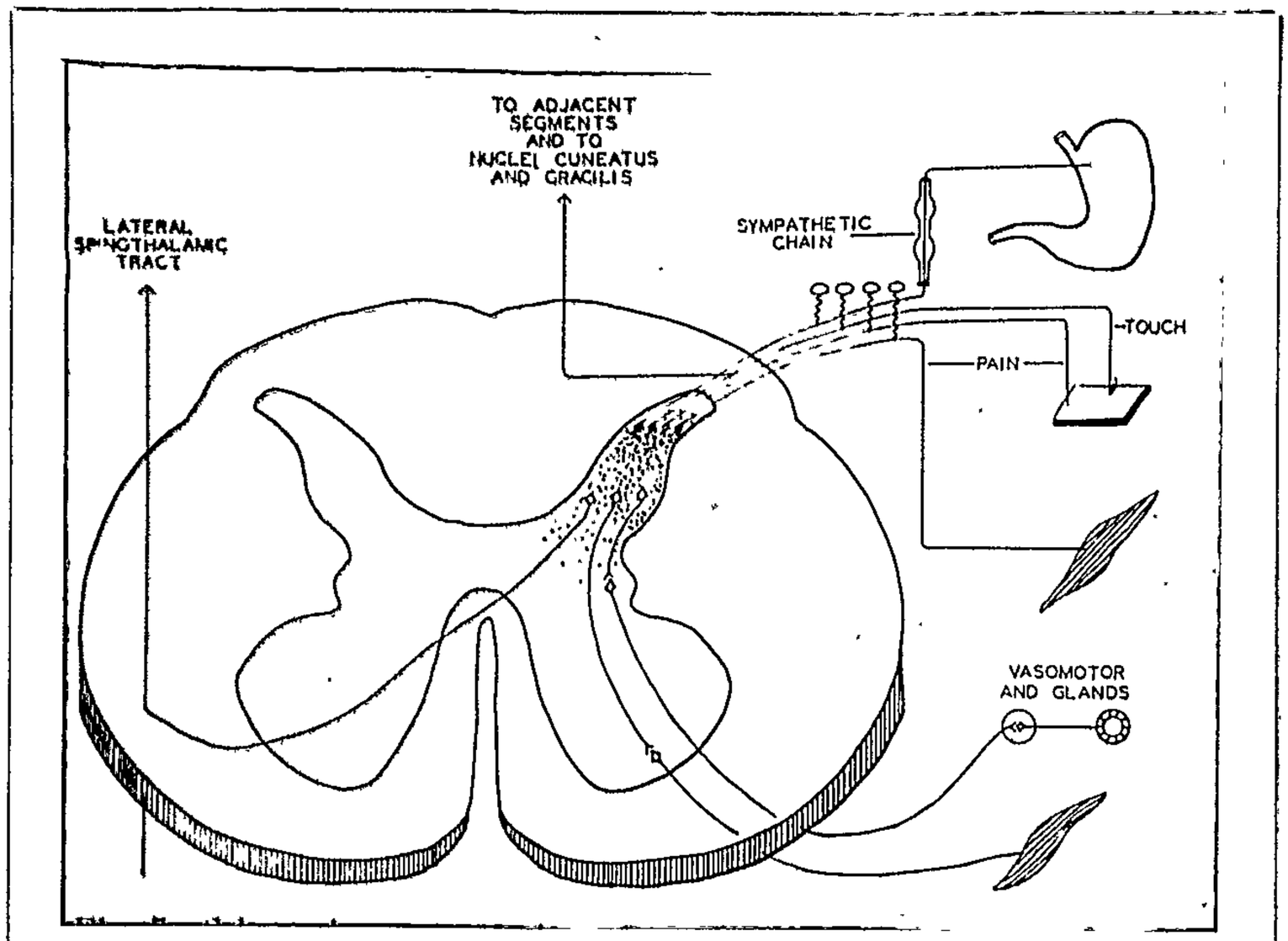

Fig. 53. Diagram of a spinal segment showing various effects of excitatory spread of noxious impulses from a focus in the stomach. The skin becomes hyperalgesic and hyperaesthetic and muscle becomes tender. Effector structures innervating blood vessels and glands (sweat and sebaceous), are involved as well as those innervating skeletal muscle causing contractions which may in themselves become painful (page 183).

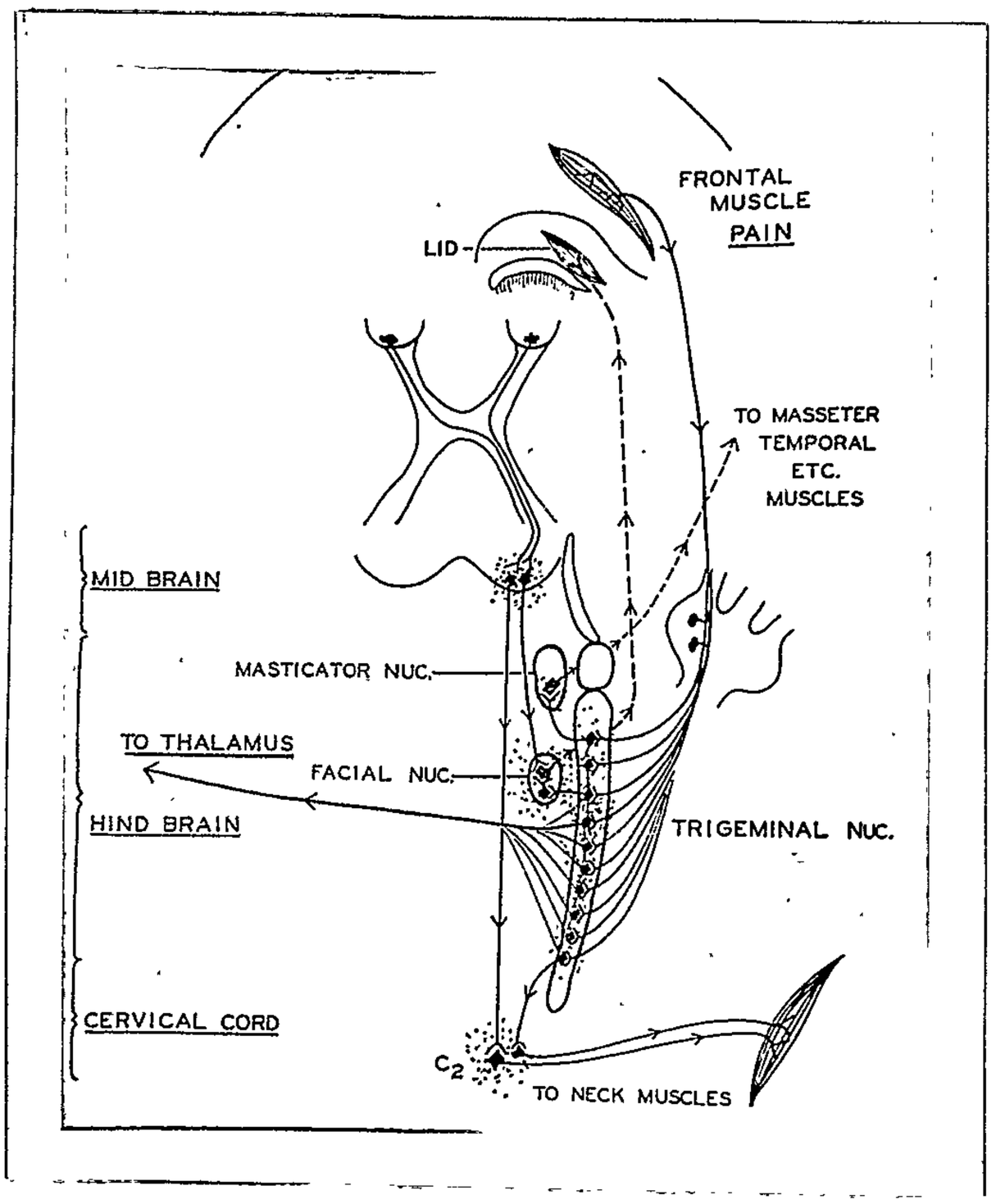

Fig. 54. Diagram of segmental structures involved in motor and sensory effects from noxious stimuli at the segmental level (page 183) - Wolf & Wolff.

The hyperaesthesia due to accentuation of the effects of sensory impulses arising in the tissue of a segment involved in referred pain may constitute the principal element of discomfort from a given visceral disease. In such cases local anaesthesia of the superficial tissues greatly reduce the patients discomfort by blocking impulses arising from the skin. Such procain infiltration cannot interrupt the central spread of excitation within the cord, however, and will not abolish referred pain as such - only the hyperalgesia associated with it.

For example, when a tooth is noxiously stimulated causing headache and superficial and deep hyperalgesia of the temporal region, infiltration of the hyperalgesic skin reduced but did not eliminate the headache. All headache was eliminated however, by infiltration about the tooth. This illustrates the fact that pain referred from deep noxious stimulation can only be completely eliminated by blocking the primary afferent impulses at their source.

As mentioned on page 159, Lewis ascribed the hyperalgesic state to the release of chemical pain producing substances in the skin mediated by nervous activity and this, he asserted, occurred whether hyperalgesia was due to local injury of the skin, stimulation of the trunk of a cutaneous nerve, or due to reference from deep somatic structures. He refuted the theory of hyperexcitable areas in the central nervous system or the spread of central excitation as being responsible for the hyperalgesia of referred pain.

Lewis postulated the presence of a special system of "Nocifensor nerves" which were common to superficial and deep tissues and which are involved in the development of hyperalgesia both cutaneous and referred. He felt that pain producing substances were released by the nocifensor nerves in areas of reference from deep somatic tissues.

The factual evidence against this "nocifensor system" lies mainly in the inability to anatomically provide evidence of its existence. Tower describes it as an "anatomical ... mstrosity".

Whereas the hyperalgesia due to local injury is probably due to pain producing substances, (possibly mediated by neural elements) it is unnecessary to postulate a new system of nerves responsible for this. The most important factor which led Lewis to reject the role of ordinary pain fibres in the mediation of cutaneous hyperalgesia was his evidence that maximal areas of hyperalgesic spread are far larger than the largest area covered by pain terminals histologically demonstrated to arise from a single fibre (171).

The Work of Tower (162) however offers an acceptable hypothesis to account for these large hyperalgesic areas, namely that the hyperalgesia producing substances presumed to be liberated, may activate and thus recruit many additional interlocking neurone units.

Lewis' theory that the erythralgic hyperalgesia of injured skin and the surface hyperalgesia arising from deep somatic tissues are "manifestations of states differing from each other merely in degree", that both states are due to pain producing substances released in the skin, and that in each case there is a lowering of the pain threshold, seems to have been disproved conclusively by Wolff et al (180, 174, 179).

The two hyperalgesic states are dissimilar and the theory of special nocifensor fibres connecting cutaneous nerves one with another and deep somatic structures with superficial tissues in untenable.

The fact that surface hyperaesthesia in areas of pain reference develops without any change in the peripheral threshold of stimulation is of great importance for it places such hyperalgesic states in the centralnnervous system.

Rigidity Associated with Deep Pain:

It is a common observation that deep pain on occasions is followed by local and sometimes distant contraction of skeletal muscle together with smooth muscle and gland effects (183, 100, 180, 174). When pain sensitive deep structures of the head are stimulated muscles of the head contract. If the stimulus

is short lived, muscle effects are also brief. If however, the painful stimulus persists, longlasting contractions of the head and neck and even the jaws and face, take place - presumably due to reflex contraction of muscle set up by the afferent impulses from the affected area (184).

Deep noxious impulses from the head region produce contractions especially in the frontalis, masseter and temporal muscles. Accentuation of winking, and ultimately the contraction of occipital and cervical muscles may occur together with local vaso-motor changes - nasal congestion, oedema of the eyelids, sweating, pupillary dilatation, nausea and vomiting (Fig. 54).

The local tenderness attributable to the central modification of sensory impulses from an area of referred pain may be increased by the rigidity from such secondarily contracted muscles. Indeed, the pain resulting from such muscle spasm may overshadow the original pain and complicate the proper localisation of the site of the deep pain.

If rigidity and tenderness, induced by secondary muscle spasm occurs, then the pain maybe modified by local anaesthetic infiltration of the muscles concerned. When such effects predominate in the pain experience, local anaesthesia of such muscles may be effective in virtually eliminating the discomfort of visceral disease.

However, when the referred pain and hyperalgesia are induced centrally, and prlonged reflex contraction of the muscles locally is not evidenced, then local anaesthetic infiltration of the muscles could not be expected to modify the central effects although it would block fresh impulses arising in the periphery whose effects would become enhanced upon reaching the cord (180).

Robertson et al (126) illustrated this latter point by prolonged stimulation of maxillary posterior teeth thereby producing deep headache and tenderness together with surface hyperalgesia and hyperaesthesia in the temporal region. Procaine infiltration intracutaneously at the site of most

intense temporal headache and tenderness caused surface analgesia, but the temporal muscle still remained tender to palpation. When procaine was injected into the belly of the temporalis, pain still persisted though local tenderness was eliminated. Only when tissues around the noxiously stimulated tooth were anaesthetised was there complete elimination of all pain.

As a summary of the characteristics of deep pain, Wolff and his co-workers (180,174) may be freely quoted. Below is an analysis of their investigations, which describe deep pain in three categories.

1) <u>True visceral and deep somatic pain</u>:

Such pain is felt at the site of primary stimulation and may or may not be associated with referred pain. It is eliminated by infiltration of procaine into the site of noxious stimulation or by blocking of afferent nerves, but it is not altered by infiltration of procaine into other structures supplied by the same or adjacent neural segments.

2) <u>Referred Pain</u>:

Such pain may occur in addition to, or in the absence of the true visceral and deep somatic pain described above, and is experienced at a site other than that of stimulation but in tissues supplied by the same or adjacent neural segments. It may occur either with or without associated hyperalgesia and hyperaesthesia.

a) <u>Without superficial or deep hyperalgesia</u>:

In this case, pain depends only upon the central effects of the spread of excitation of the original noxious impulses to the same and adjacent segments of the cord, whence they are relayed to higher centres for perception and interpretation. Injection of procaine into superficial or deep regions of referred pain does not reduce the intensity of pain due to this mechanism.

b) <u>With superficial and/or deep hyperalgesia</u>:

Referred pain may be accentuated in intensity by virtue of ordinarily non-noxious stimuli from zones of reference. Impulses from such sources, normally inadequate to produce pain, may do so upon reaching the cord in a segment involved in the central spread of excitation. Procaine injected into superficial or deep hyperalgesic structures will abolish this element of referred pain phenomenon, resulting in more or less reduction in the patient's discomfort depending upon the amount of hyperalgesia.

3) Pains due to the secondary skeletal muscular contractions which provide a fresh source of noxious impulses:

Pain may result from secondary effects of the central spread of excitation on the effector structures including painful contractions of skeletal muscles. Such disturbances may be widespread and the pains may be experienced in situations remote from the original source of noxious stimulation. Local infiltration of the contracted muscles with procaine abolishes this type of pain by disrupting its peripheral mechanism.

THE SEPARATENESS OF SUPERFICIAL AND DEEP PAIN

Lewis (100) draws attention to the essential differences between superficial and deep pain and maintains that the two systems are quite separate in character and establish different connections in the central nervous system. For instance, painful stimulation of the skin awakens quick protective reflexes and a rise in pulse rate, whereas painful stimulation of deeper structures does not possess this association. Rather it results in quiescence and withdrawal reactions, often associated with slowing of the pulse, a fall in blood pressure, sweating and nausea. Nauseating pain is never derived from cutaneous pain.

"There is a reason to think that all nerve impulses are fundamentally alike, and that nerves are mere conductors of a common pattern of excitation thrown into them at the end apparatus. The central connections which ultimately receive these impulses alone determines the form of sensation, be it sound, light, or pain. The difference in the qualities of superficial and deep

pain is so clear as to make it unsafe to regard both types as represented in a common centre.

The difference in the quality of pain derived from skin and deep structures leads one to suppose that it is probable that these are separate forms of sensation, as separate, for instance, as touch, warm and cold .....establishing different connections with the central nervous system".

CHAPTER 7

THE AETIOLOGY OF FACIAL PAIN

The complex innervation of the maxillo facial region, the variations in pain reaction and the phenomen of referred pain often obscures the aetiology of facial pain. Disease distant from the oral cavity may set up pain symptoms in the areas of the mouth, palate, throat and tonsils and conversely, oral manifestations may engender generalised pain in the head and neck. (169, 152)

The aetiology of facial pain is so varied as to make precise classification difficult. Various outlines have been adopted by writers on this subject, many of which are unwieldy and confused.

Agnew (2) describes three categories of pain in the head and neck:- the first related primarily to the strictly odontal tissues and the environmental tissues adjacent to the dental organs; the second grouping of pain patterns relates to pain referred from the dental or associated tissues to other nearby or remote region; the third category includes pain referred to the odontal and related structures from adjacent or more remote locations.

Taverner (156) finds it convenient to start peripherally and work centrally along the ramifications of the 5th cranial nerve while Harrigan (81) tends to describe pain emanating from specific structures and specific pain syndromes in order of relative frequency.

The approach of Stones (151), Cohen (33) and Monica (111) while differing slightly in outline serves as a basis for the classification used in this thesis.

Some confusion exists with regard to the terminology used in describing pain states. The terms 'neuritis' and 'neuralgia' are often used with a lack of semantic precision, and it is necessary to clarify these.

Neuritis is defined as "inflammation of a nerve or nerves

marked by neuralgia, hyperaesthesia, anaesthesia or parasthesia, paralysis, muscular atrophy in the region supplied by the affected nerves, and by abolition of reflexes" (Stedman's Medical Dictionary). "Lesions of a nerve or nerves, either degenerative or inflammatory" (New Gould Medical Dictionary).

Neuralgia is described as "severe paroxysmal pain along the course of a nerve, not associated with demonstrable structural changes in the nerve". (New Gould Medical Dictionary)

"Nerve pain: pain of severe throbbing or stabbing character in the course or distribution of a nerve".

The two terms are therefore quite distinct. Neuritis refers to pathological involvement of the nerve itself whether inflammatory, toxic, degenerative or traumatic (2). Neuritis may give rise to neuralgia or aching of various intensity but it frequently results in hypoaesthesia due to degenerative change. An attempt to assign neuritis with a definite pain pattern is therefore quite unproductive.

Neuralgia on the other hand refers to a symptom. Cohen points out, however, that neuralgia should not be synonymous with 'pain' as such. It more precisely denotes pain corresponding to the known anatomical distribution of a nerve and implies pain along the course of a nerve (151, 33). This tends to exclude the whole range of local pain states related to disease of peripheral structures such as the teeth, antra, ears etc.

Two types of neuralgia are recognised. The first is due to a gross pathological lesion, such as new growth, scar, injury, infection etc., directly involving the nerve and this type is best termed 'symptomatic' or 'secondary' neuralgia. There is also a type of neuralgia which occurs in the absence of any obvious gross pathology and is often called 'tic' or 'primary' neuralgia.

Both neuralgias give pain in the recognised anatomical distribution of a nerve, but certain features distinguish the two. In 'primary' neuralgia, the pain is paroxysmal - shocklike,

stabbing, or lightning pain - lasting seconds with complete or almost complete remissions between the spasms.

Pain of symptomatic neuralgia is usually more constant and is a mixture of superficial and deep pain. Sometimes it is pricking and burning, but usually it is boring and aching; it is usually continuous but very rarely there are remissions.

The aetiology of primary neuralgia is obscure whereas secondary neuralgia may always be relieved by removal of the cause. With secondary neuralgia there may be associated objective signs of interruption of continuity of the nerves. via. anaesthesia, paresis and muscle wasting, absent reflexes or trophic changes. These signs tend gradually to spread to contiguous areas (111, 33).

A method for considering the aetiology of facial pain is now proposed.

A. <u>Peripheral structures which may give rise to facial pain</u>:

(i) Teeth
(ii) Periodontium
(iii) Jaws
(iv) Nasal and Paranasal structures
(v) Temporo mandibular joint
(vi) Ear
(vii) Eye
(viii) Muscles of the head and neck
(ix) Styloid Process
(x) Heart
(xi) Blood vessels

B. <u>Primary Neuralgias</u>

(i) Tic Douloureux
(ii) Glossopharyngeal
(iii) Geniculate
(iv) Superior Laryngeal
(v) Post Herpetic

C. Secondary Neuralgias caused by:

(i) Intracranial lesions

(ii) Intracerebral lesions

(iii) Extracranial lesions

D. Psychogenic Pain

A. PERIPHERAL STRUCTURES WHICH MAY GIVE RISE TO FACIAL PAIN.

(i) The teeth as a source of pain

Among the most common causes of facial pain is pulpitis and its sequelae i.e. extension into the periodontal tissues with abscess formation. The diagnosis of pulpitic pain is often difficult because of the inability of the patient precisely to localise the source of pain.

Noyes states (115) that "if several pulps on the same side are exposed including those in both the maxillary and mandibular arches, so that they can be stimulated without perception from the periodontal membrane, it will be impossible for the patient to localise the tooth which has been stimulated. The pain from a single tooth may be referred to almost any point on the same side supplied by the trigeminal nerve".

This inability to localise pulpal pain is in striking contrast to the clear localisation of periodontal pain. It has been shown that in the skin, muscles and periodontal ligaments, sensory nerves of different modalities abound and it is impossible to stimulate pain receptors without at the same time stimulating receptors of touch, pressure, heat, cold or proprioceptive sensations. It is through the ability of exact localisation of these sensations that we localise the impact of pain. Where, however, as with the dental pulp, pain receptors are the only sensory nerve endings and there are no proprioceptive fibres, pain at the outset is difficult to localise (115, 142) (page 101).

The pain may be referred to another division - involvement of the upper teeth may simulate pulpitis in the lower teeth and vice versa. Authors have attempted to compile tables of pain reference from pulpitis involving various teeth (72, 158, 81) (Fig. 55). It is really only possible to generalise, for depending on the severity and duration of pain, so the area of reference becomes more remote.

Pulpitis in upper centrals, laterals and canines tends to cause pain in the fronto nasal and naso labial regions while the pain reference from upper premolars and the first molar tends

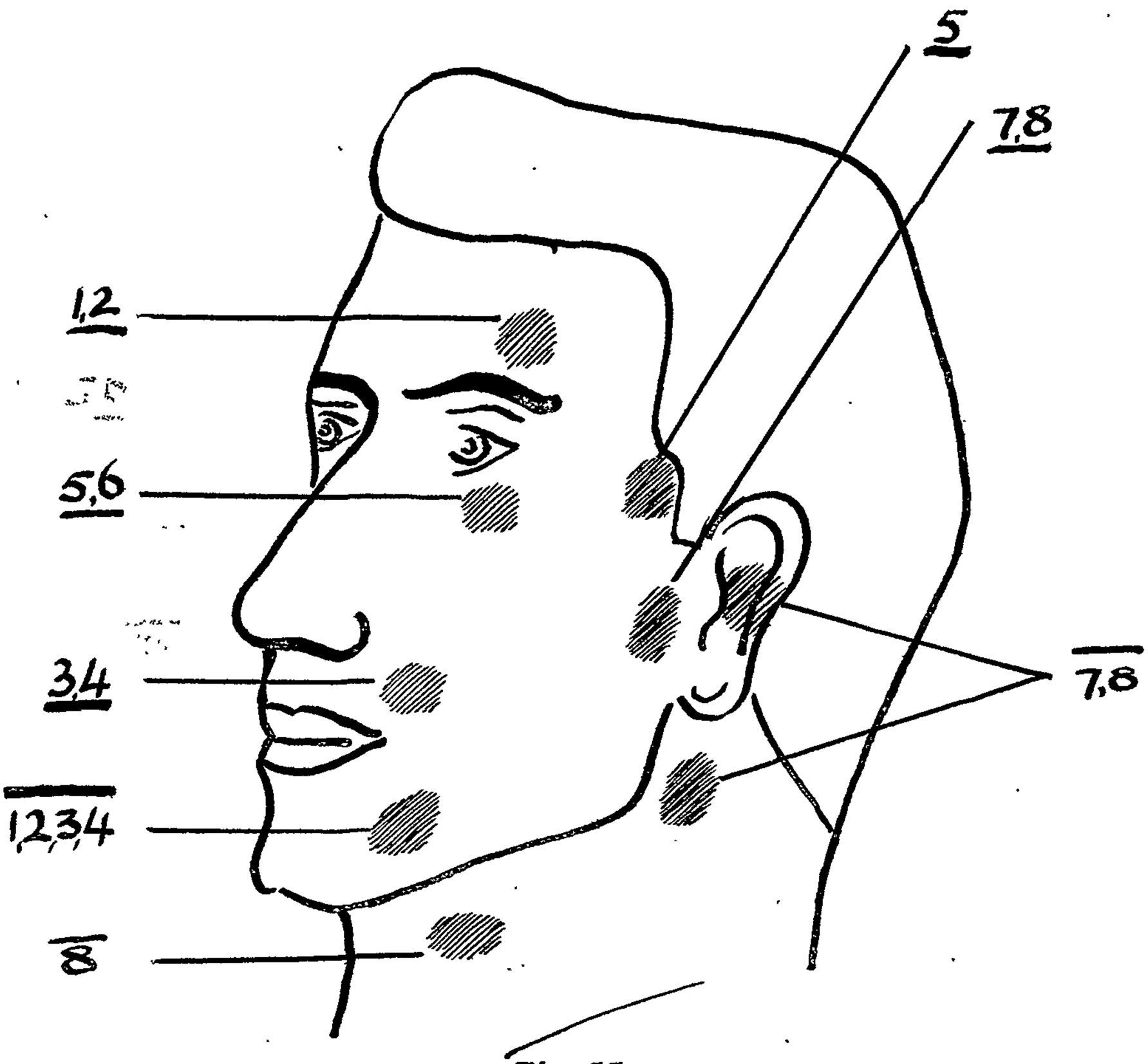

Fig 55.

Head's areas of Referred pain from the teeth

Head in 1884 called attention to certain surface areas of the face and neck to which sensations of pain, originating from specific teeth might be referred. Such definite correlations between specific teeth and definite surface areas, will not always be found to exist, however.

to be maxillary and temporal in distribution. Pain emanating from the $\frac{78}{78}$ may be mandibular and/or maxillary and such reference may be remote - as far as the premolars in some cases.

Pain from mandibular posterior teeth may be referred close to the angle of the jaw and upward behind the ear. Sometimes known as '<u>otalgia dentalis</u>' (157) this pain reference to the ear may occur by way of the tympanic plexus, which is connected with the second division of the fifth nerve by means of the spheno-palatine ganglion via the greater superficial petrosal nerve. It is probably referred in most cases from the third division through the otic ganglion which anastomoses with the chorda tympani.

It is only after careful study of the patient's history and searching clinical examinations together with adequate X-rays and pulp testing that the causative tooth be isolated.

Severe pulpitis can give rise to extensive referred pain, headache and hyperalgesia. Robertson, Goodell and Wolff (126) stimulated upper and lower teeth using electrical methods.

Severe toothache was induced for ten minutes using a gradually increasing voltage and interrupted current to overcome accommodation effects.

When stimulating upper molar and premolar teeth they found that pain tended to spread into adjacent teeth and along the maxilla with occasional jabs of more intense pain spreading into the eye, orbital ridge and temple. Autonomic effects were noted. After termination of the stimulation, the pain in the tooth decreased but a steady aching, profuse pain was experienced in the homolateral temporal region, along the zygomatic ridge and for a short distance over the eye. This headache and pain persisted for up to eight hours. During this time the temporal muscles and overlying tissues were tender to palpation and the skin over the painful area was hyperalgesic. In such cases local anaesthetic infiltration intracutaneously over the site of most intense temporal headache caused only partial diminution of pain, the temporal muscle remaining tender and pain continuing from areas other than the analgesic areas. Local anaesthetic

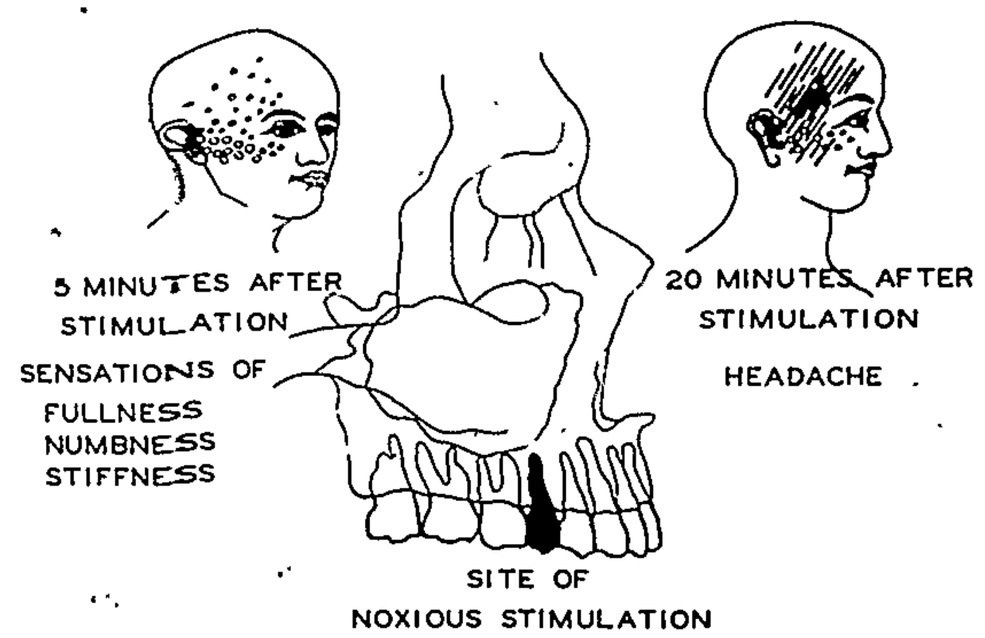

Fig. 2.—Distribution of sensations of fulness, numbness and stiffness and distribution of headache following noxious stimulation of a tooth on the right side of the upper jaw.

Fig. 56.

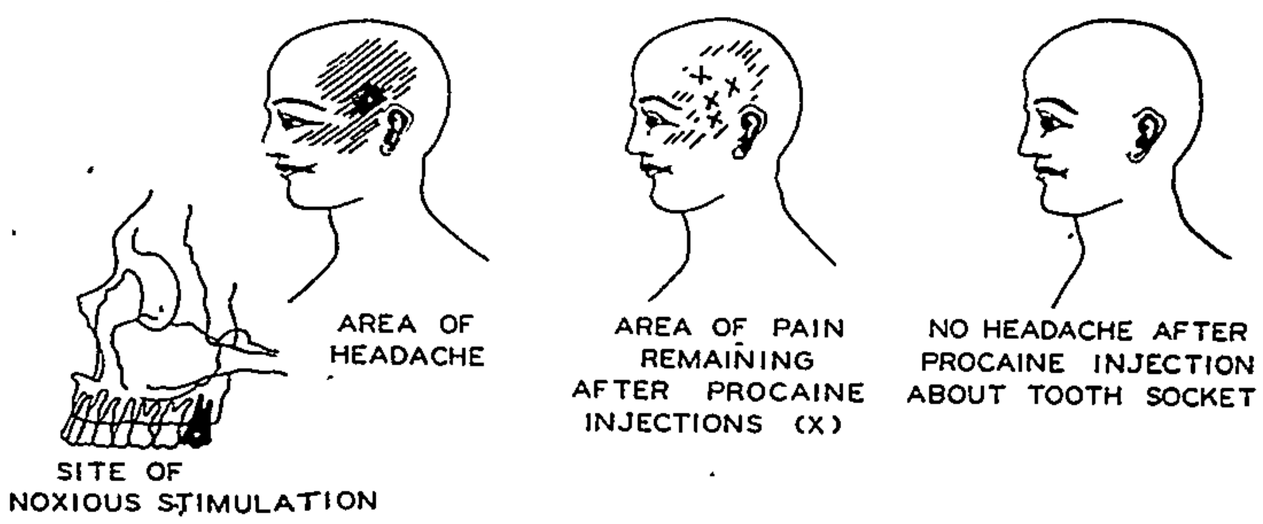

Fig. 5.—Area of headache following noxious stimulation of a tooth and the effect of injections of procaine into the painful area, as compared with the effect of injection of monocaine into the site of noxious impulses.

Fig 57.

of the stimulated tooth however, administered 20 minutes after the period of noxious stimulation, eliminated all pain and sensations of tightness in the head remote from the site (Fig. 56, 57). Such an experiment was duplicated in a case where an upper third molar tooth had been extracted under local anaesthetic. Postoperative headache and spread of pain were similar in all respects to the experimental stimulation and these symptoms were again speedily relieved by blocking the posterior superior dental nerves three hours afterwards.

These experiments bear out the fact that diffuse headache and referred pain is caused by noxious impulses arising from the injured pulp or periodontal membrane. These impulses give rise to excitatory processes in the brain stem which spread to exert their effects on many trigeminal structures (Chapter 6). It is interesting to note that whereas the pain threshold was substantially lowered in the zone of noxious stimulation, the sites of remote hyperalgesia was not associated with a lowered threshold - the effect being exaggerated reaction rather than reaction at a lower level of stimulation.

These workers stimulated a lower premolar in a similar manner and found that during the period of stimulation intense pain was experienced locally and throughout the lower jaw which usually extended into the anterior wall of the auditory canal. Following cessation of stimulation, pain developed which extended throughout the upper and lower jaw into the area over the zygoma and the temple to the top of the ear. There was also a sense of fullness and aching in the ear. This discomfort was increased on biting, and stiffness of the masseter was apparent (Fig. 58).

They concluded that noxious impulses arising from one or more of the upper teeth resulted in pain which was experienced at first locally and then diffusely in tissues supplied by the second division of the fifth nerve. Noxious impulses from lower teeth also caused pain to be experienced at first locally and then widely in the tissues supplied by the third division of the 5th nerve. Moreover, the site at which pain was felt was often remote from the primary sources of noxious impulses. If the

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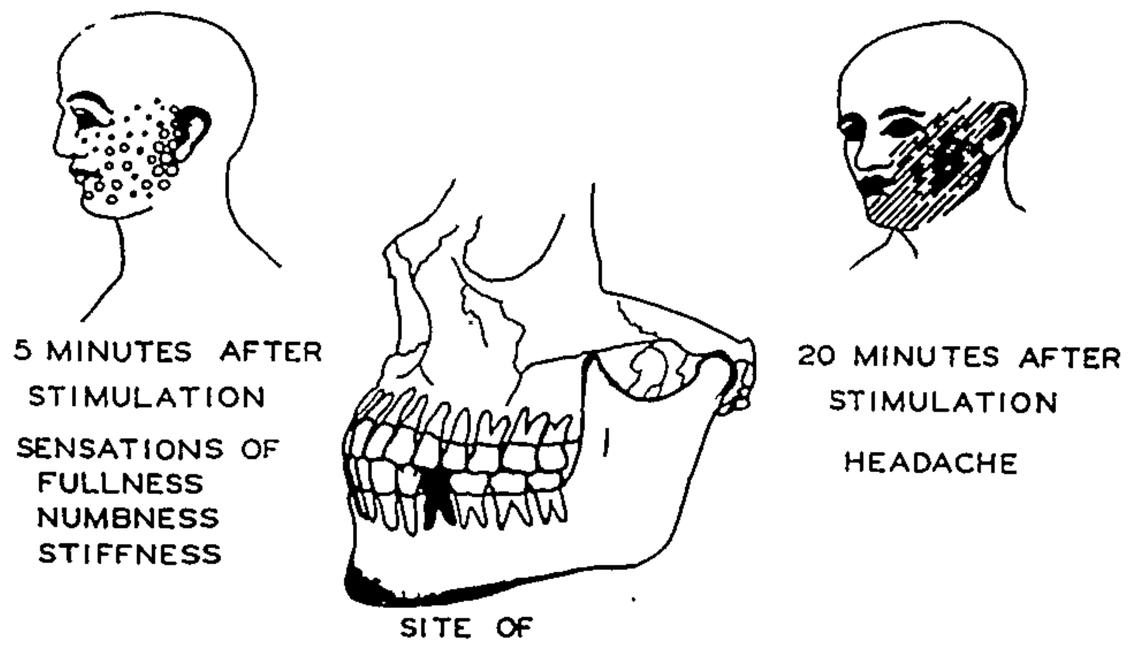

Fig. 3.—Distribution of sensations of fulness, numbness and stiffness and distribution of headache following noxious stimulation of a tooth in the left side of the lower jaw.

Fig 58.

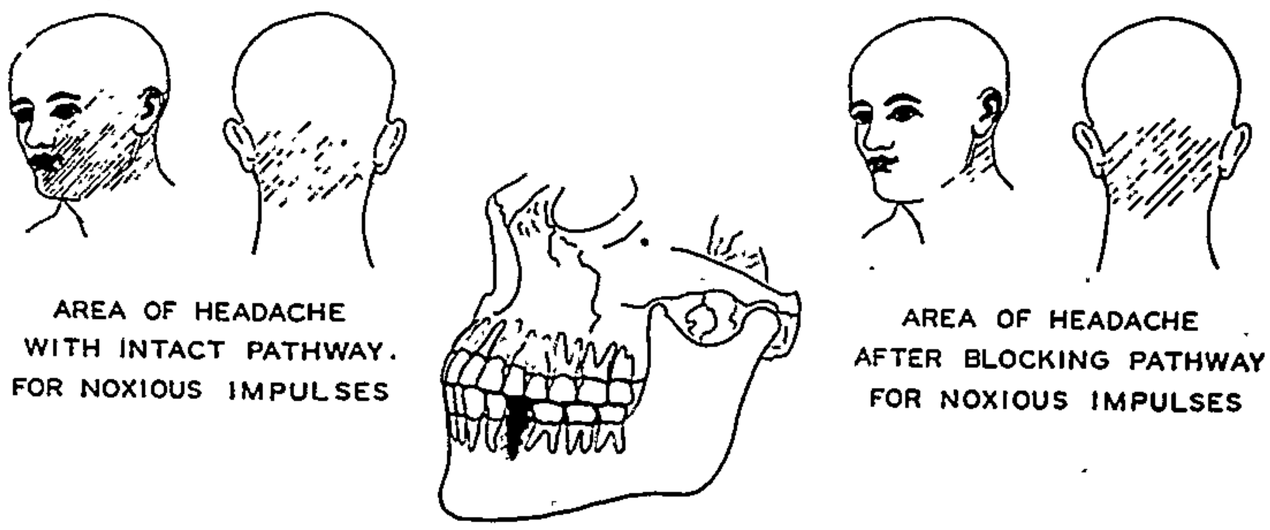

Fig. 6.—Area of distribution of headache accompanying prolonged noxious stimulation in the lower jaw (osteomyelitis), showing headache primarily due to the central spread of the effects of noxious impulses arising in the diseased tooth and headache secondarily arising from contraction of muscles in the head and neck. The former was abolished by blocking the pathway for noxious impulses, whereas the latter persisted for several hours.

Fig. 59.

noxious stimulation of the teeth was sufficiently intense and prolonged, regardless of the site of origin, the pain usually spread and was experienced also in tissues supplied by the first as well as the second and third division of the 5th nerve. The size of the painful area bore no close relation to the size or location of the noxious stimulation, but prolonged disturbances were more likely to give rise to distant pain than were short lived disturbances.

Further more, homolateral hyperalgesia, tenderness and vaso-motor reaction of tissues remote from the source of noxious impulses in the teeth were common.

The above examples deal with the effects of relatively brief stimulation of the teeth but when a patient is subjected to very prolonged periods of noxious impulses from the teeth, more severe symptoms may present due to secondary spasm of the masseter or temporal muscles.

In such cases pain from sustained muscular contraction may be a dominant feature of discomfit.

Robertson (126) quotes the case of a patient who experienced excruciating toothache of two weeks duration associated with osteomyelitis on the right side of the lower jaw. The headache and pain extended from the midline of the chin along the lower jaw, into the ear, into the upper jaw and into the neck below the jaw and across the back of the head. All the lower teeth on the right ached. There was intense pain at the angle of the jaw and the jaw could not be opened more than 0.5 cm. because of spasm and extreme tenderness of the masseter. Monocaine block of the mandibular nerve eliminated all pain in the face but tenderness and pain in the right side of the neck, and both superficial and deep tenderness and headache in both sides of the back of the head and neck persisted due to sustained muscle contraction. (Fig. 59).

Pain from the dental pulp varies greatly in degree depending upon the aetiologic agency and the reaction of the pulp tissue.

It is not proposed to deal exhaustively with the pathology of the pulp. This aspect will be approached in a general way and an effort made to simplify the complicated expositions found in many texts.

We rightly accept the fact that sufficiently intense stimuli injure the pulp and precipitate an inflammatory response with resultant pain, and we also concede that such a response may be acute or chronic. The attempt to subdivide such states into acute serous, acute suppurative pulpitis, or chronic ulcerative pulpitis, chronic pulpitis aperta etc. tends to be confusing and perhaps unnecessary from a clinical stand-point. It is impossible to diagnose from the patients symptoms and existing tests the precise pathological state of an inflamed pulp.

Thoma (157) supports this view and emphasises that acute inflammation is a progressive disease.

A variety of miscroscopic stages are met with but the process is a continuous one resulting either in total involvement of the pulp or if the defensive mechanism is effective, a transition to a more chronic response.

The most important aspect of the problem is to consider whether the pulp is irreversibly inflamed or whether it is in a state of irritation from which it may recover.

The term hyperaemia is loosely applied to a pulp in a transitional state and the precise meaning of this term must be examined.

Maurice (108) endeavours to differentiate between the physiologic hyperaemic pulp and the pathologic hyperaemic pulp and the point is well made. The former term describes a pulp which is otherwise normal, but which reacts as one would expect to an irritant in close proximity to it. If this irritant persists or progresses to involve the pulp, then hyperaemia as a prelude to exudation and emigration of leucocytes eventually occurs.

The patient's history of a sharp pain as a result of applying a stimulus, which ceases within seconds on removal of the stimulus is indicative of a pulp which is not as yet pathologically diseased. The common agents cited as responsible for this state are chemical irritants - (cements); physical trauma (fracture of the crown); operative trauma; and a deep carious lesion. Such transitory pain is often elicited by stimuli which are in fact ordinarily innocuous to tissues e.g. warm water or sweets, and the conclusion must be that the pulp must be in a state of exaggerated sensitivity as a result of the initial irritation i.e. the threshold must be lowered.

Robertson et al (126) showed that the pain threshold in a tooth which have been painfully stimulated electrically for 10 minutes was lowered 35% below its control level 30 minutes after cessation of the experiment when the tooth itself was no longer aching. Fig 60. The experimental tooth was also painful when cold air was sucked through the mouth or when the subject drank cold water. This lowering of pain threshold of the pulp following noxious stimulation is in accord with the observations of Lewis and Schumacher on the skin. It is postulated that noxious stimulation or irritation of a tooth results in a local and transitory inflammatory reaction which probably results in the local liberation of a substance which lowers the pain threshold.

Because of this, minor tissue changes such as vasodilatation and oedema become capable of evoking impulses.

Such noxious stimulation with lowering of the pain threshold occurs following operative trauma or following tooth fracture with resultant free communication between pulp and oral fluids.

In many cases however, a pulp remains asymptomatic under a large carious lesion until actually exposed. This, as Fish has so adequately pointed out (55) is due to the insulating effect of eburnoid and secondary dentine depositions under the area of penetration. "Caries would be a most painful disease without the 'eburnoid' reaction and it would be possible to get quite severe toxic absorption from widespread caries if it were not for the impenetrable barrier which this calcific deposit

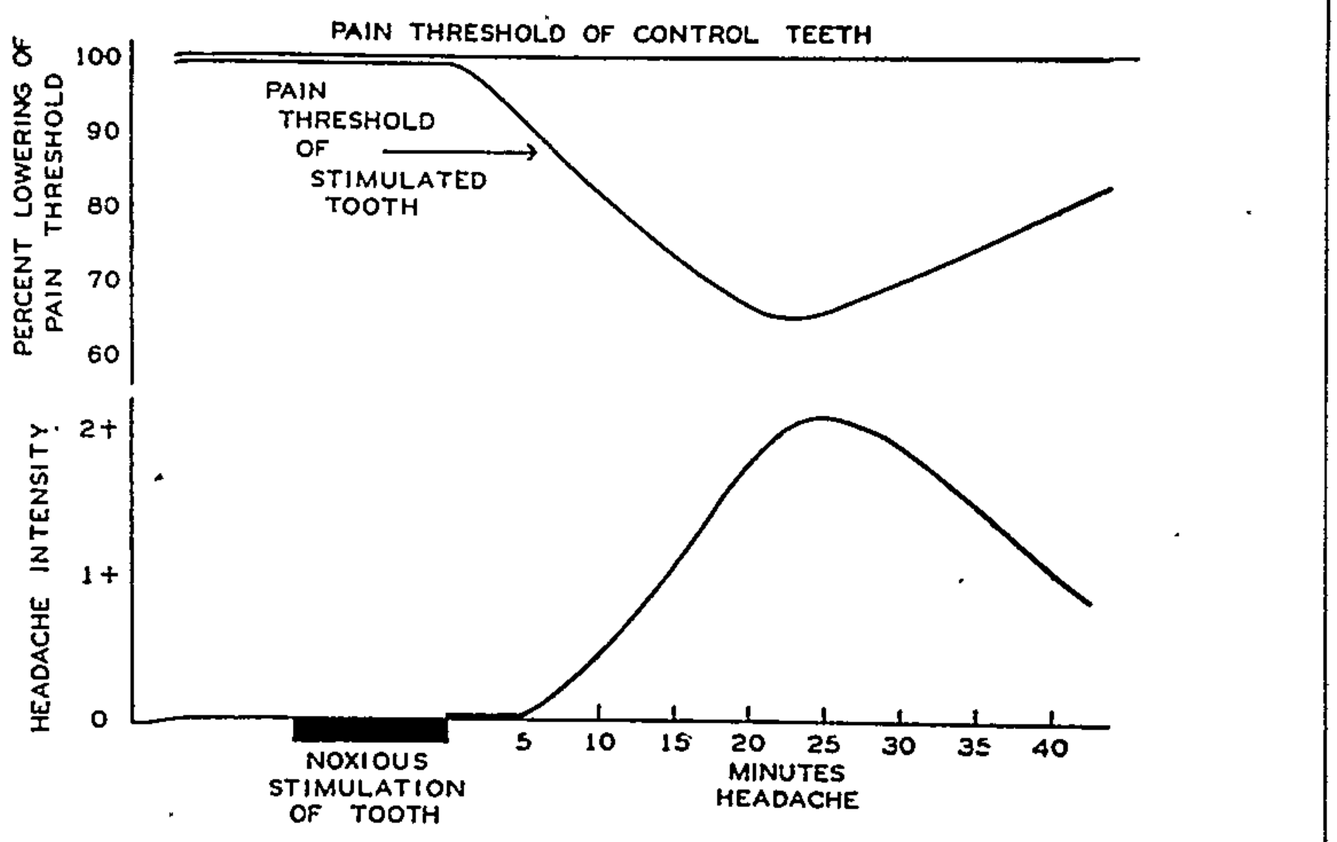

Fig. 4.—Lowering of pain threshold as measured by a "vitalometer" in a tooth which had been noxiously stimulated. The lowering of the pain threshold in the tooth was accompanied with headache on the homolateral side. The pain thresholds of the two adjacent teeth were not altered.

FIG. 60.

provides". Fish continues to discuss the extreme tenderness of carious cavities in the molar teeth of children and considers that soft leathery masses of carious dentine are of such rapid development that the pulp has had insufficient time to protect itself with calcific tissue.

Teeth which are hypersensitive as a result of such causes as mentioned above, and which give rise to sharp but very brief pain in response to heat and cold, are amenable to conservative treatment.

If, however, the pain is severe and lingers for some minutes after application of the stimulus then it is probable that the hyperaemic condition is associated with excessive exudation and oedema and the development of frank inflammation is inevitable. Apart from heat, cold and electric tests, an indication of pulp exposure under a carious lesion in a tooth presenting such symptoms is pain elicited following pressure on the base of the lesion with a pellet of cotton wool.

Two clinical entities must be considered at this stage before proceeding to a discussion of pulpitis - cervical sensitivity and internal haemorrhage of the pulp (Fish 55).

The pain which accompanies these conditions is often so severe as to indicate irreversible pathology of the pulp tissue.

Cervical sensitivity whether due to caries, abrasion or erosion, or gingival recession and abrasion, of the cementum can give rise to a persistently painful condition which is inordinately sensitive to touch and to sweet, hot and cold.

Histologically, Fish has shown that below such a lesion, the odontoblasts have disappeared without producing the usual eburnoid barrier. The usual reaction may have been prevented by contamination and chronic irritation. Such a condition is usually readily diagnosed, for the patient is acutely conscious of the pain when brushing the teeth.

A further condition which is met with clinically is that following operative trauma. In most cases postoperative

sensitivity to thermal changes may be a transient affair or may be so severe as to develop into frank pulpitis. Occasionally however a case presents when the pulp remains sensitive to thermal changes for some weeks or months. The author has noticed many such cases which have in time become quiescent with pulp vitality maintained. Fish ascribes this to haemorrhage within the pulp. "The blood vessels of the pulp are thin walled, though relatively large, vessels and rely on the fact that they are enclosed in the pulp chamber for hydrostatic support. The sudden opening of a large number of tubules withdraws this support to some extent and it is found experimentally that haemorrhage is common if large areas of live dentine are suddenly opened up".

In cases where caries has progressed to involve the pulp, where chemical irritation has caused severe damage to pulp tissue with inflammation or when trauma has been excessive, pulpitis will develop.

In the case where caries has involved the pulp, the reaction, i.e. whether acute or chronic, depends on the numbers and virulence of the invading organisms and the resistance of the tissue to invasion.

Virulent pathogenic organisms, or severe injury due to other factors, result in the development of an acute inflammatory reaction. The vessels in the part of the pulp affected dilate at the expense of the more remote vessels which later collapse to make room in the rigidly confined space of the pulp chamber. There is an exudation of plasma and polymorphs heavily infiltrate the area. If the cause of the inflammatory process is caries, the exposure is liable to become blocked up by fibrin so that exudate from the vessels raises the pressure in the pulp. This, togehter with toxic irritation of the nerve endings, causes intense pain.

Leucocytes are killed, pus is formed, and a pulp abscess develops. In acute pulpitis, spontaneously occurring paroxysms of pain are experienced (157). These are often pulsating in character and are felt with each systole of the heart. Hot food

intensifies the pain when pus has formed. Oedema, dilatation of the blood vessels, and cardiac systole are adequate stimuli at this stage.

Either due to strangulation of the pulp or pathogenicity of the organisms, diffuse suppurative pulpitis may develop with complete necrosis of the pulp. If this invasion proceeds into the apical periodontium, the radiating, poorly defined pain becomes readily localisable, and tenderness to percussion is diagnostic of apical periodontitis.

If the pus from the pulp abscess escapes into the carious cavity through the exposure, the reaction may settle down into an acute or even a chronic state. Thus after a period of intense throbbing pain symptoms may subside almost completely, the organisms having been localised by the leucocytes of the debris in the abscess cavity.

The preceding paragraphs briefly describe the sequelae of acute inflammation of the pulp, but many causes of pulp death occur which are clinically silent. Herbert (88) found that chronically inflamed pulps may easily pass unsuspected, some giving no symptoms, some causing pain for various periods following stimulation, and others causing aching at intervals without stimuli.

Chronic inflammation is due to diffusion of toxic products from the organisms rather than the actual penetration of organisms throughout the pulp tissue. We are therefore dealing with organisms of low pathogenicity and invasiveness. It can be visualised that all degrees of reaction from extreme chronicity to acute inflammation with varying symptomatology can be inaugurated. Thoma (157) lists the various organisms which may be found in chronic pulp abscesses, each capable of causing slow invasion and necrosis.

Apart from bacterial agencies, irritant medicaments and chemicals may be the cause of a persistent chronic response with gradual pulp necrosis e.g. silicate cement.

Histologically, the four classic zones may be discernible around a localised chronic abscess in the coronal pulp. The zone of infection or intoxication; the zone of contamination characterised by lymphocytic infiltration; the zone of irritation, characterised by histiocytic resorption of the collagenous framework of the fibrous tissue, and the zone of stimulation where reparative processes are in operation attempting to localise and wall off the irritant.

In other cases, bacteria may be widely distributed and multiple small focal abscesses may develop; alternatively diffuse infiltration of the pulp by mononuclear cells may occur.

Symptomatology, as stated, is very variable and in many cases is not diagnosed until operative proceedures reveal an exposure of a necrotic pulp or an x-ray shows changes around the apex of a discoloured tooth.

With regard to diagnosis of pain emanating from the pulp certain points are important. The history will generally differentiate pulpal pain from other types of facial pain; localisation is usually impossible and exaggerated response to thermal change is present. Heat and cold will aggravate or relieve pain depending upon the condition of the pulp at the time of testing. Such stimuli have no affect on other types of facial pain.(81, 151, 152). One usually finds that as true pulpitis develops, response to cold decreases, and eventually disappears, while heat causes increasingly severe aching pain that persists for longer and longer periods.

In cases of obscure pulpal pain, the electric pulp tester furnishes valuable information, since the relative ability of a pulp to react to the electric current denotes in some measure the vitality of the pulp (37). It is not, however, an absolute criterion of the state of the dental pulp, but is purely an index of the presence or absence of gross pathology (108). Failure of a tooth to respond usually indicates a non vital pulp. Response does not always imply the pulp is vital however, for liquified pulp material may conduct the electrical stimulus to

the periapical region and give rise to pain.

All too often a case presents where vague pulpal symptoms may be attributed to a number of possible causes in different teeth. The procedure must always be the elimination of each possibility, for precise diagnosis in such instances is often impossible.

A word might be said about the tendency for obscure pulpal pain to be assigned to the presence of pulp stones. Although many cases of pain possibly produced by pulp stones have been reported, the fact that pulp stones are of such common occurrence, with few complaints of pain makes any such diagnoses suspect. Histologically the tissues surrounding such stones are normal, and it is difficult to envisage pressure effects in view of their very gradual development.

Pain emanating from the pulp as a result of galvanism has been recognised for many years, but a clear understanding of its mechanism has only recently been elucidated by Mumford (112).

Itseems that when dissimilar metals come into contact during occlusion of the teeth, an external circuit is made via the tissue fluids and a current of 0.5 to 50 microamps generated. In the soft tissues, current can spread out, but in the tooth its path is restricted and current density may be great enough to stimulate the pain fibres of the pulp. This does not invariably occur however. Stimulation is characteristically brief, for rapid polarisation occurs.

Most cases of galvanic pain cease to occur within a few hours or a few days after placing of the restoration and this is due either to the ageing of the cell i.e. formation of surface metallic sulphides decreasing E.M.F., or deposition of colloidal film on the surface considerably increasing the resistance.

The problem of galvanic pain seems to have been greatly exaggerated in the past and transient symptoms which sometimes occur should never be a contraindication to the rise of dissimilar metals in the mouth.

The role of impacted or unerupted teeth in the aetiology of facial pain is discussed by many writers on this subject (159, 108, 37, 151, 81). Thoma asserts that the pressure caused by root development of impacted third molars on the mandibular nerve is a frequent cause of referred pain. He lists many case reports which illustrate the relief of pain, local and referred, gained by removal of impacted mandibular molars. Many unerupted teeth, however, remain clinically unnoticeable, especially canines and mesiodens and the emphasis placed on the pressure on nerve roots during attempted eruption of impacted teeth may be unduly stressed.

It is a clinical fact however, that many impactions - especially lower third molars - are associated with and responsible for pain symptoms. Association with pericoronitis and folliculitis is frequently observed and it is probable that such inflammatory states are responsible for pain in the majority of cases. Although the tooth may be unerupted clinically, there are many cases where some communication exists between the tooth follicle and the oral cavity, and this may well be responsible for a recurrent and transient folliculitis.

In other cases, resorption of teeth adjacent to the impaction may become apparent and if caries, with pulp exposure, supervenes, pain of varying severity will result.

In conclusion it may be stated that valid instances of obscure facial pain being produced by apparently quiescent impacted teeth are rare.

(ii) Pain from the Periodontium

Periodontal pain, especially that resulting from an irritation of the apical periodontium, may sometimes be confused with pulpal pain, but there are usually several distinguishing features. Since the periodontal membrane possesses receptors for pain and touch, the patient can readily locate the affected tooth which is sensitive (151). Again, whereas pulpal pain gives rise to sharp, shooting, lancinating or throbbing pain, which tends to be intermittent in character and often more severe in the reclining position, the periodontal abscess results in a dull continuous, pulsating pain, the position of the body having no influence on it.

The periodontal membrane is most commonly injured by traumatic factors, chemical irritants or bacteria (108) and periodontal pain may conveniently be classed in two divisions - that due to non infective injury and that which results from infective injury.

Traumatic injury to the periodontal membrane may result from an actual blow, a high restoration, biting into a hard object or forceful separation of teeth. The injury may be mild or severe varying from slight concussion to actual sub-luxation or fracture of the tooth concerned.

The haemorrhage and oedema consequent on such an injury tends to raise the tooth in its socket. The pain and soreness of the periodontal membrane is therefore extreme and tenderness to biting and percussion is very apparent. The tooth itself may be mobile, and due to damage to the apical vessels, may feel numb.

A common cause of injury to the apical periodontium is that following instrumentation during root-canal therapy. After pulp extirpation, there is inevitably apical oedema and haemorrhage and the symptoms vary from a slight awareness to frank tenderness. In view of this, it is inadvisable at any time to carry out immediate root canal filling, for unless the apical exudate has a means of draining into the canal and being absorbed, serious symptoms may develop. Instrumentation beyond the apex and the

diffusion of irritant drugs from a root canal dressing are capable of causing extreme discomfort. The tooth should be relieved from occlusal stress in all cases, for the transient apical oedema may readily develop into acute periodontitis with pus formation.

Bacteria may gain access to the periodontal membrane either by way of a periodontal pocket or via the root canal. In the latter case it may be the direct sequel to acute pulpitis, - the bacteria invading the periapical tissues and initiating an acute periapical abscess. In other cases, the defence mechanisms, constituted by the granuloma, may faill and an acute exacerbation become superimposed on a tooth which has been non vital and symptomless for some time. Frequently this occurs following root therapy instrumentation on a non vital infected root canal. If the organisms which are confined within the root canal by the granulomatous reaction are precipitated into the apical tissues by instruments, an acute exacerbation is usually inevitable. In all cases a rapidly developing inflammatory reaction occurs, the periodontal membrane becomes oedematous, the tooth is extruded, loose and very sore. When pus forms, the ache is extremely severe while it is confined to the bone and the patient complains of constant pulsating pain which may become excruciating when the tooth is touched or percussed.

Such an abscess will spread in the direction of least resistence, involving the marrow spaces and then finally perforating the cortex. The consequent progress from subperiostal abscess to submucous or subcutaneous abscess depends on the site of perforation of the cortex i.e. the relation to muscle attachments. A spreading cellulitis with invasion of the fascial spaces characteristically occurs and the patient at this stage exhibits general systemic symptoms.

A lateral periodontal abscess or an inter-radicular abscess may develop as a result of chronic periodontal disease and periodontal pocketing. In acute cases there is usually throbbing pain, tenderness of the gingiva to palpation and sensitivity of the tooth to percussion. The pain is readily

localisable and usually some oedematous, hyperaemic elevation of the overlying gingiva is present in the region of the abscess. In chronic cases, there may be a variable amount of bone destruction in relation to the lateral aspect of the root, associated with intermittent exudation from a sinus orifice along the lateral gingival mucosa. While symptom free in many cases, the patient may report episodes characterised by a dull gnawing pain, slight elevation of the tooth and a desire to bite and grind the tooth (71). Clinical findings such as the presence of extensive caries, pocket formation, tooth vitality as well as radiographic evidence aids the differentiation between periodontal and apical abscess.