ORIGINAL CONTRIBUTION

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Head Pain

Introduction

Pain is defined in many ways: the sensation received or resulting from the stimulation of specialized nerve endings;[1] more poetically, a punishment or penalty, for crime; acute discomfort of body or mind; bodily or mental suffering or distress; a distressing sensation, as in a particular part of the body; trouble experienced in doing something.[2] The experience of pain may be colored by rather diverse circumstances or, as we may describe in more scientific language, feedback. From the therapeutic standpoint, head pain is most usually interpreted by the clinical view (i.e., What do we do to stop it?) rather than from the view of pathophysiology.

In much of our analysis of head pain, we prefer to look at it as a phenomenon or as resulting from the stimulation of specialized nerve endings. In reality, pain may be an interpretation of bodily or mental distress. Boshes and Arieff state that:

> Certain aspects of pain are dedicated exclusively on a neural substrate. Here the basis is an event or an alteration in the nervous system per se, as contrasted to pain caused by malignant disease, infected tissue, fractures or the like. Various divisions of the nervous system may be implicated and a description of the disability or the manner of posture and movement is often sufficient to enable the trained observer to gain an impression as to whether the pain is genuine or functional. Such involvement may be at the receptive, the conductive, the perceptive or the apperceptive level, or combinations thereof.[3]

Purpose

This would appear to be a generally accepted concept: yet, head pain is described and interpreted on the basis of symptom complex rather than the anatomic and physiologic organization of the CNS [central nervous system]. It is the purpose of this paper to attempt to describe some of the mechanisms involved in head pain and to, again, attempt to osteopathically orient them.

Neural Pathways of Pain

Most of the sensory nerve distribution to the head and face occurs through the trigeminal nerve (CN V) and fibers of cervical nerves C 1, 2, and 3. These trigeminal fibers have their neurons in the semilunar ganglion which lies in a cave of dura mater in the middle cranial fossa just anterior to the apex of the petrous temporal bone. The peripheral branches of the trigeminal nerve, the

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Keywords

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ophthalmic, maxillary and mandibular nerves supply a fairly well defined cutaneous area, and the deeper structures underlying it. There is little overlap with the adjoining cervical nerves.

The glossopharyngeal nerve supplies common sensibility to the posterior third of the tongue, pharynx, soft palate, tonsils, auditory tube, tympanic cavity and mastoid air cells, and the inner lining of the eardrum.

The vagus nerve supplies the general somatic afferent fibers to the posterior portion of the external auditory canal, part of the eardrum, and the skin of the cranial surface of the auricle adjoining the scalp.

The pain fibers of the glossopharyngeal and vagus nerves relay impulses to the nucleus of the descending trigeminal tract (spinal nucleus).

Cutaneous distribution of C1 is not consistent. Occasionally it gives a cutaneous branch to the skin of the upper part of the back of the neck and the lower part of the scalp.[4]

The second cervical nerve chiefly supplies the area of the head and neck adjoining the trigeminal territory, with the third cervical nerve also contributing fibers.[5]

The nerve fibers supplying the cranial dural mater are derived from the trigeminal nerve, the upper three cervical nerves and the sympathetic trunk.

Nerve branches from the upper three cervical nerves and the superior cervical ganglion supply the dura mater of the posterior cranial fossa. The dural nerves, derived from the three divisions of the trigeminal nerve and from the sympathetic plexuses on the internal carotid and middle meningeal arteries, supply the remainder of the cranial dura mater.[6]

The first division of the trigeminal nerve supplies the dura mater in the anterior fossa, the diaphragma sella, nearly all of the cerebral falx, tentorium cerebelli, superior sagittal sinus, straight sinus, superior wall of the transverse sinus, and terminal parts of the cerebral veins entering these sinuses.

The maxillary division of the trigeminal nerve supplies the dura mater in the anterior part of the middle cranial fossa. Branches of the third or mandibular division of the trigeminal nerve supply the dura mater in the posterior and lateral parts of the middle cranial fossa and most of the dura lining of the calvaria.[7]

Perhaps the more important aspect of pain is that there is not a single identifiable factor. It may be represented by vastly complicated and intricate processes or by the mere experiencing of the touch of a sharp object. The integration of actual pain reception and perception represents an area of widely diverse opinion. Gooddy[8] was impressed by the observation that successive surgical interruptions of peripheral nerves, posterior roots, spinal cord, and thalamus, and ablations of portions of the cerebral hemispheres, may all fail to give permanent relief from pain. He concluded that "any nervous pathways are potential pain pathways."

The main nuclei involved with the reception of stimuli arising from the spinal cord (i.e., cervical segments 1, 2, and 3) pass to the nucleus cuneatus (homolateral) synapse, cross at this level, and ascend to the ventrolateral nucleus of the thalamus.[9]

The function of the thalamus is to pass impulses on to the cerebral cortex and it is presumed that these impulses are integrated by the association nuclei in the thalamus before being relayed. The portion of thalamus that projects impulses to a specific cortical area receives in return corticothalamic projection fibers from that area, forming a circuit between thalamus and cortex.[10]

Pain fibers of the great auricular nerve synapse in the substantis gelatinosa (Rolandi) and second order neurons ascend in the lateral spinothalamic tract to the posteroventral nucleus of the thalamus.

Pain fibers from the trigeminal nerve have their cell bodies in the semilunar ganglion. Their central processes descend as the spinal tract of the trigeminal nerve, in the lateral brain stem from the upper pons to the C-2 level of the cord to terminate in the associated spinal trigeminal nucleus which lies adjacent and deep to the tract. The spinal tract and the spinal nucleus correspond to and are continuous with the dorsilateral fasciculus of the cord and the substantis gelatinosa, respectively.

Pain afferents from the face, arriving via the trigeminal, glossopharyngeal, and vagal routes, relay to the portion of the spinal nucleus lying below the inferior limit of the fourth ventricle.[11]

Second order neurons from the spinal trigeminal nucleus cross the midline as the ventral secondary tract to ascend on the medial aspect of the lateral spinothalamic tract to gain the thalamus. There is doubt as to the thalamic termination of these fibers. The classic view is that the trigeminal lemniscus (combining the ventral and dorsal secondary trigeminal tracts) projects to the medial portion (arcuate nucleus) of the posteroventral nucleus of the thalamus. From the posteroventral nucleus of the thalamus, third order neurons pass in the sensory radiation via the posterior limb of the internal capsule to the somatic sensory area of the cortex in the lowest portion of the postcentral gyrus (Brodmann's area 3, 1, 2) just above the fissure of Sylvius. There is evidence of the face being represented bilaterally in the thalamus and the cortex. It is likely that the thalamus is responsible for the recognition of pain: however, the perception of pain as a mental event requires cortical participation, and probably includes diffuse and generalized cortical participation.

There is also evidence that pain pathways from both cord and medulla relay bilaterally in the reticular formation of the brain stem and ascend by slow multisynaptic routes to the medial thalamic nuclei and become part of the diffuse thalamic system. The latter system, which is thought to control the general level of direction of attention, may also be responsible for the affective coloring of pain.[12]

Vascular Elements of Pain

The sensitivity of the vascular elements is discussed by Wolff.[13] In his investigation, greater sensitivity to compression, stretching, and faradic current stimulation was observed in the arterial system. The great venous sinuses were less sensitive to these stimuli. The lesser sinuses and veins lost sensitivity in proportion to their distance from the greater sinuses.

The blood vessels of the head receive their preganglionic sympathetic innervation from T-1 to T-2, but C-8 and T-3 and even T-4 may also contribute.[14] The axons pass out into the sympathetic chain and ascend to synapse in the stellate and the superior cervical sympathetic ganglia. The postganglionic fibers distribute from the superior cervical sympathetic ganglion with the external and internal carotid arteries to the head.[15] The intracranial postganglionic fibers follow along the internal carotid artery to the circle of Willis and along branches of the external carotid and distribute to the adventitia and the smooth muscle of intracranial vessels, including arterioles of the pia mater, but not to the blood vessels in the brain substance. Postganglionic fibers also distribute to the middle meningeal artery. The plexuses along the common carotid and the internal carotid are not continuous with those on the external carotid: stripping the plexuses from the common and internal carotid will not destroy the sympathetic supply to the blood vessels of the face and hand. Postganglionic fibers from the stellate ganglion ascend along the vertebral arteries and the basilar artery.[16]

A parasympathetic innervation to some of the blood vessels of the head likewise has been demonstrated. Preganglionic parasympathetic fibers of the facial nerve turn off in the region of geniculate ganglion to run in the greater superficial petrosal nerve to the plexus on the internal as vasodilator of the vessels.[17]

The vascular tone (sympathetic parasympathetic influence) appears to be mediated through the forebrain with connection in the hypothalamic nuclei. These tracts constitute the various corticohypothalamic systems and the cortico-thalamo-hypothalamic tracts by way of the dorsomedial thalamic nucleus.[18] That is to say, the thalamic tract system is a study unto itself. It seem probable, as others have suggested, that the cortical paths are regulatory over the thalamic systems. The pathways in general provide for emotional accompaniments to cortically-initiated motor responses carried over the pyramidal and extrapyramidal systems. Evidence has been presented that pyramidal as well as extrapyramidal systems carry corticofugal fibers for autonomic centers of the spinal cord.

Before passing into a discussion of the types of stimuli that may be interpreted as pain, a discussion of the character of nerve endings present in the meninges and associated structures of the head and neck will clarify the types of stimuli that may give rise to pain.

The sensory terminations in the dura have been studied by various observers. The nerve endings at the base of the skull are less numerous than on the convexity. They are in the form of end branches (bare nerve endings), knob- or club-shaped terminations (Golgi-Mazzoni, Pacinian, & Meissner corpuscles), or are like balls of twine (Ruffini end organs).[19] Meissner corpuscles are found to be associated with the finest tactile sensation. [20] The Golgi-Mazzoni receptor is said to be a pressure receptor, of similar function to the Pacinian corpuscle. The Krause corpuscle has been associated with low temperature discrimination. It has been suggested that it may function in distinguishing cool rather than cold.[21] Ruffini end organs appear to serve in more than one type of receptor. The larger Ruffini endings serve as pressure endings while smaller endings of this type are present in the subcutaneous connective tissue and are regarded as receptors of warmth.[22]

Golgi, Meissner and Pacinian corpuscles have been described as receptors of discrimination in joint motion. They are credited with reporting motion characteristics in regard to rate of position change, direction of motion and force required to product position change.[23]

Characteristics of Pain

Having described the circuitry involved, let us return to pain, which may result directly from factors originating outside the body (i.e.; sharp object, excessive heat, etc.), pathophysiological changes within the body (i.e., sustained muscle tension, tumor, etc.), or by abnormally mediated psychological factors through autonomic response. Pain may result from mechanical stimulation, psychological stimulation, or as a reaction to a combination of these. It might be described then, as a response to stimuli that threaten tissue integrity or organizational integrity of the body unit.

Various authors classify pain according to the particular portion of the nervous system immediately responsible for the transmission of the stimulus to CNS. Thus we may refer again to the classification of pain as being at the receptive, the conductive, the perceptive or the apperceptive levels, or combinations thereof.[24]

Pain must also be discerned as a local, projected, or preferred phenomena. Localized pain is restricted to the immediate area of reception: i.e., pain in a tooth from an apical abscess. Projected pain in the head may be exemplified by trigeminal neuralgia, apparently due to restriction in dural investiture of the root as it passes over the petrous ridge, in Meckel's cave housing the ganglion or in the sleeves around the three branches as they exist from the skull.[25] Pain is projected at times over the entire hemiface served by the nerve. Referred pain may be exemplified by thrombosis of the posterior inferior cerebellar artery in its pain reference to the face.[26]

Although these classifications of pain overlap to some degree, using the combination of classifications helps to explain various phenomena of pain production. The patient waiting for the attention of the dentist or surgeon may mentally suppress pain and say, "It doesn't hurt like it did yesterday," until the approach of the time for local anesthetic preparation. Then a touch by any object may produce a highly unique response in the area of attention. The apperceptive mechanisms, mediated through the nuclei of the thalamus and modified through the cortifugal control systems of the cerebellum[27,28,29,30] and the pituitary-adrenal hyperstimulation by fear, cause pain that is uniquely individualized by the patient's level of apprehension. The corticofugal controls, exerted through the cerebellum, modify the intensity of activity occurring both on a motor level and through the thalamic nuclei. It appears that damage to or suppression of this system may be responsible for the rigidity, hyperactivity, dysmetria, ataxia, and epileptiform activity exhibited by patients with brain damage or trauma.[31] W. G. Sutherland[32] refers to his observations and conclusions in reference to stress mechanisms involving the dura mater and cranial sutures. The observations of the various types of nerve endings in the leptomeninges makes the information supplied by stress on the dura mater and pia mater available to the centers of perception, apperception, and motor activity. The demonstration of the recurrent meningeal nerve in the spinal area (most especially the branches that enter through the foramen magnum and with internal carotid artery–sympathetic–and supply the dura mater of the posterior cranial fossa) makes available the same information from the spinal cord meninges and supporting ligaments.

Ray and Wolff in 1940 studied the probable cause of headache or head pain in relation to the dura mater from observations made on many patients during surgical procedures on the head. They concluded that the pain results primarily from inflammation, traction, displacement and distention of cranial vascular structures. Unfortunately they failed to mention until much later[35] that the actual pain-sensitive nerve endings were located in the dura mater, arachnoid mater, and pia mater supporting the fascicular structures. These factors throw new light on the observations of Sutherland, especially since the dura mater on the internal surface of the cranium is continuous with the periosteum of the head.

There is no information, in print, to support the possibility of a "strain gauge" type of reporting across the sutures, but the observation of the sensory distribution to the internal and external surfaces of the cranial vault would appear to make such an arrangement feasible.[36,37]

The information available indicates that essentially the same types of stimuli elicit painful reactions whether arising internally or externally to the cranium. Psychological modification, through mechanisms mentioned, are most likely to affect those areas of reception most easily observed through the special senses (sight, hearing, etc.)

Since involvement of the special senses introduces the possibility of modification of afferent stimuli by influence of the limbic system, neurophysiological evidence has suggested that this portion of the nervous system (the limbic system) is concerned with smell, taste, and other special senses, the gastrointestinal system and other automatic functions, and behavioral patterns.[38]

This brings pain into the area of the psycho-neuro-physiological processes of reception, conduction, and perception to the stage of apperception or total integration of the process of interpreting pain, and a possible introduction of the subject of pain threshold (which is not a subject for discussion on this paper).

It should be mentioned that there are definite interrelationships between the corticofugal system, mentioned earlier, and the limbic system, which have yet are not clearly defined.

An Osteopathic Approach to Head Pain

The foregoing discussion has described the circuitry necessary for the identification and response to head pain. Feedback mechanisms necessary to establish a cybernetic model have been outlined. From this description it should not be difficult for the knowledgeable physician to apply therapeutic measures. The knowledgeable osteopathic physician possesses the palpatory skills to directly intervene into the pathophysiologic process. Pain in the head, through the mechanisms described, produces palpable reflex area or tissue responses in the superficial tissues (skin, etc.) and/or the muscles and deep connective tissues. By discriminatory palpation, the relative age or stage of chronicity may be determined and therapy applied.

H. V. Hoover has written extensively and descriptively in regard to the application of technique to the various ages or stage of the process involved in stress.[39,40,41,42] In the use of functional technique, the physician may affect the established cybernetic system by entering as a service aid. In this mode of therapy, enough force is exerted, through the various planes of motion of accommodation to the tissue or articulation, to bring the structures involved to a point of "dynamic reciprocal balance."[43] In this way the operator establishes a new state of equilibrium within the limits of its ability to accommodate physiologically. "Treatment by functional technic depends upon and is directed by the reaction of a part of the patient to demands for activity made upon that part."[44]

By the recruitment of the demonstrable tissue changes and changes in tissue activity, it is possible for the palpating hand to discern the cybernetic mechanisms involved in the origin of head pain. The basis of cybernetic mechanisms is "feedback." This is the process of transferring energy or information from the output of a circuit to its input and its generally accepted control mechanism in all types of self-regulating systems that use a closed-loop, negative feedback network.[47]

The author has not found the employment of active and passive joint motion palpation to be of satisfactory discrimination in the analysis of such cybernetic mechanisms, to allow him to enter into a "servocybernetic" relationship with the patient on a therapeutic level.* After observing several highly skilled osteopathic physicians in their approaches to palpation and treatment of a wide variety of pathophysiological processes and syndromes, a method of diagnostic palpation became apparent. In working with this newly found method, its applications and uses began to clearly reveal themselves, and continue to do so. At this time, a series of articles were published, separately, by two of the highly skilled physicians the author had observed.[45,46] The use of the principles presented by Drs. Becker and Frymann allows the physician to determine the area or areas of stress, the character of the assault involved, and to counteract their deleterious effects.

The previous discussion of CNS mechanisms demonstrated what is presently known of the circuitry, involved in the feedback mechanisms of the human body in relation to head pain. After the physician has determined the areas of stress and the character of assault, treatment is based on the counter-balancing of the stress forces: that is, changing the characteristics of the input and feedback, thus creating a servocybernetic system through establishing controlled input that alters the level of control influence exerted by the negative feedback network. The completion of treatment for any particular time is signaled by a more physiologic reaction of the tissues

*Editor's Note: "servocybernetic" is a portmanteau term that is modified from mechanical engineering. A servomechanism is a control system that responds and correct dynamic input about position and other variables, particularly in a closed loop system. Cybernetics refers to a closed loop causal system.

involved: an increase in activity of hypoactive tissue and/or a decrease in the activity in hyperactive tissue, and a synchronous motion (internal or external rotation, flexion or extension) with the basal respiratory cycle or pulmonary respiratory mechanism as described by Magoun.[48] This allows the individual to establish a new level of homeostasis, compatible with his or her ability at any particular time to recover from the original assault.

Stress patterns of considerable age and complicated by numerous overlying injuries have responded in surprising manners to therapy applied in this manner.

Case History

Perhaps this may be illustrated by a case history. Mrs. W. E., a 44-year-old white female was admitted to the hospital with a chief complain of severe headaches occurring in the left occipital area and radiating to the left temporal bone and vertex of the skull. The headaches were associated with nausea and vomiting. The onset of the headaches were associated with an automobile accident that had occurred 6 years previous to this hospital admission. Following the accident the patient developed hemianesthesia of the left arm, leg, and left side of the face.

At this time the patient was hospitalized for a period of 22 days. She did improve with bedrest but was not freed of the pain, and retained paresthesias of the left arm, leg, and left side of the face. She was unable to turn from a supine position to a left lateral recumbent position. It was not clear whether this was due to weakness, loss of proprioception, or loss of motor control. The patient spent a total of 66 days in the hospital over the next 2 years for paresthesias of the left side of the body and headache (left hemicephalgia). The patient denied having been unconscious at the time of or following the accident.

There was no familial history of neurological disease or headache. The surgical history included an appendectomy, [caesarean] section, and a total hysterectomy. Neurological examination failed to demonstrate any abnormal findings and the cerebrospinal fluid cellular morphology, and chemistries were not remarkable. The CSF pressure was in the mid-range of normal and the Quackenstedt test elicited no abnormal findings. Laboratory tests included CBC, FBS, creatinine, UA, and VDRL at the time of admission and discharge were within the accepted norms. X-ray examination at the time of admission revealed an apparent articulation between the posterior tubercle of posterior arch of the atlas with the occiput, and a decrease of the normal lordotic curvature of the cervical spine.

Following a week's hospitalization, the author was called in consultation and elicited the following additional findings: decreased backward bending of the cervical spine, a flattening of the cervical lordosis, and bilateral compressive mechanism through the sacroiliac articulations. The cranial mechanism demonstrated sphenobasilar compression with vertical strain (sphenoid high), left side bending with rotation, and a slight right torsion. The entire paravertebral mass was under extreme tension from occiput to sacrum.

Very light treatment was given to the sphenobasilar vertical strain, suboccipital area and sacrum due to the hyper-irritability of these tissues and their inability to react. The patient was not treated again for 48 hours due to the consultant's attendance at a two-day meeting. At the second treatment, the tissue reaction was much improved and the patient could endure deeper treatment to the involved areas. Following this treatment, the patient's cervical spine was again x-rayed and the films showed that the posterior arch of the atlas was no longer in contact with the occiput and there was improvement in the cervical curvature.

The patient's pain decreased over the next 24 hours and she was released from the hospital to be seen by the consultant in his office within 48 hours. The patient was seen at regular intervals for the next 3 weeks (twice weekly). At the end of this time, the patient had been pain free for approximately 10 days, and the length of time between treatments was lengthened to weekly intervals. As the patient's tissue response improved, the interval between treatments was correspondingly lengthened, without recurrence of severe headaches—until her daughter, who had a congenital cardiac valvular lesion, told her parents of being pregnant. These headaches responded quite well to therapy but recurred frequently and were terminated by the arrival of a normal healthy granddaughter. The patient is still seen on occasion for maintenance and preventative therapy.

Discussion of Therapy

The treatment of this patient was carried out accordingly to the principles already described.

Following routine physical examination, a thorough palpatory examination was carried out. Palpation began at the sacral area: the sacrum was cupped in the left hand, with the patient in the supine position, with the first finger extending to contact the right iliolumbar ligament (lower portion), the little finger at the left sacroiliac articulation, the second and third fingertips placed just lateral to the top of spinous process of the fifth lumbar segment of the spine. Light palpation demonstrated relatively little activity of the tissues. Carrying the palpation deeper demonstrated a rigidity of the ligamentous structures supporting the sacroiliac articulations both anteriorly and posteriorly and extreme tension throughout the ilio-lumbar ligaments bilaterally.

Light palpation is carried out by a very light contact with skin. The depth of palpation is increased by establishing a fulcrum and gently increasing the tension or pressure distal to the fulcrum so that the palpating hand may remain relaxed and be used as a palpating instrument rather than attempting to constantly monitor its own proprioceptive phenomena. The pressure is gently increased until reaction is stimulated in the layer of tissue the examiner wishes to palpate. The resulting tissue reaction will demonstrate to the examiner more details about the summation of the various forces exerted at the time of injury that elicited the protective reaction of the tissues under examination.

The transition from examination to treatment is a matter of following the summative force to the point of dynamic reciprocal balance and maintaining this balance until the issues complete their accommodation. This accommodation is accompanied by great tissue relaxation and a feeling of increased tissue vitality, and a longitudinal to-and-fro motion, corresponding to the primary respiratory cycle. If continued force is applied to the injured tissues after the immediate response, the ensuing fatigue may result in adverse or over-reaction of the treated tissues, which appears to create a type of kinesthetic shock (a disassociation of the proprioceptive motor feedback mechanism) resulting in a loss of coordinated, previously programmed, or learned motion patterns with an increase in sensitivity, possibly pain, in the particular ligaments and connective tissues. This can cause gait or motion aberration that is not typical of the individual, usually demonstrated in a single member or limb or segment of such member or limb.

Each area found to be involved in the total stress mechanism was treated in a similar manner, the only differences being in the method of application of the testing or treating forces to accommodate to the peculiarities of anatomical structure of the region under study and treatment. In the cervical area, palpation is performed along the lateral margin of the paravertebral mass which is located over the articular pillar. Thus we are able to palpate the paravertebral mass, periarticular ligaments, and the reaction of the musculature attaching on the anterior aspects of these vertebral segments. In palpation of the cranium, the index finger approximates the lateral aspect of the great wing of the sphenoid, the second finger is placed posterior to the sphenosquamal articulation (asterion) and the little finger is placed on the occiput.

This contact is often altered to suit unusual injury patterns but, in any case, the application of treatment follows the same basic principles. The fulcrum is usually established, by crossing the thumbs. The flexor pollicis longus muscles are utilized to maintain good contact and allow the hands to remain as relaxed as possible. Thus the hands may be free to move with the demonstrated force mechanisms and establish the dynamic reciprocal tension necessary to allow the tissues to overcome injury force mechanisms. The mastering of this type of therapeutic and diagnostic approach is not difficult but requires studious concentration not to hinder the activity of the tissues so that they may reveal the stress patterns to which they have been subjected. The physician must remain relaxed and observant so he may participate in assisting the tissues to reach the point of reciprocal dynamic tension and maintaining this variable fulcrum (point of dynamic reciprocal tension).

The material covered demonstrated the possibility of pain arising from the neck and possibly lower levels. In many cases the involvement of arthrodial articulation may require more stringent or forceful modes of therapy. In Hoover's article on "The Use of Shock as Alternative Therapy,"[50] the author describes the use of high velocity to accomplish a "popping" of the joint whereby the involved levels of discrimination must reorganize their synaptic organization in response to the shock produced by the forceful articulatory motion, thus establishing a new level or at least a different degree of function. Thus a major part of the training, pre- and post-graduate, of the osteopathic physician entails the responsibility of understanding the mechanisms involved and discriminately developing his diagnostic skills, especially palpation.

The little understood mechanisms of the central nervous system are slowly revealing their intricacies through the devoted efforts of many dedicated and curious researchers. Although these people may divulge this information, it becomes the responsibility of the physician to be aware of their discoveries and to adequately analyze the information and discreetly apply it to the clinical situation. The information presented may give the osteopathic physician a slightly different view of the application of osteopathic manipulative therapy to his patient.

Summary

In summary, we have introduced and discussed the neuroanatomy and physiology involved with head pain. Various types of input that may be characterized as pain have been mentioned and mechanisms demonstrated as to the process of their apperception as pain. An attempt has been to correlate the wide varieties of osteopathic manipulative approach to the particular situation in which pain is expressed in the head. A case history, exemplifying the author's approach to such cases has been discussed and the principles of treatment described.

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