1962

Evaluation and management of the patient with the headache

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EVALUATION AND MANAGEMENT OF THE PATIENT WITH HEADACHE

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Submitted in Partial Fulfillment for the Degree of
Doctor of Medicine

College of Medicine, University of Nebraska

March 5, 1962

Omaha, Nebraska

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I. Introduction

Possibly the most common complaint of modern man is headache. This symptom may be an indication of an underlying disturbance within the cranium, in other systems of the body, in the personality or environment, or in a combination of these. To establish a diagnosis and offer effective therapy, the physician must understand both the physiological and personality mechanisms responsible for the pain. He must use extreme patience and skill in eliciting a history, making a careful physical and neurological examination, and evaluating the need for ancillary studies pertinent to the problem.

The purpose of this paper is to evaluate the current literature on the management of the patient with headache; to present the mechanisms and a clinical classification of headache; to discuss the differential diagnosis of the more common types of headache; and to consider general principles in the therapy of headache.

II. Mechanisms of Headache

Pain-Sensitive Cranial Structures

Intracranial Structure

Observation in conscious patients during craniotomy by Northfield, by Ray and Wolff, and by Penfield and McNaughton [1 p 8] revealed the following intra-

-cranial structures to be sensitive to pain: the major venous sinuses and their tributaries from the surface of the brain; the meningeal arteries in the dura; the major arteries at the base of the brain leading to and emerging from the circle of Willis; the cranial and upper cervical nerves carrying fibers for pain from the head; and the dural floor of the anterior and posterior fossae. Those structures that were pain-insensitive included the parenchyma of the brain, most of the pia-arachnoid and the dura, the ependymal lining of the ventricles, the choroid plexus, and the cranium itself. In these same observation the sites on the surface in which pain is felt when any of the pain-sensitive intracranial structures in stimulated were noted. The data supports two generalizations: (1) pain arising from intracranial structures on or above the superior surface of the tentorium cerebelli is referred to frontal, temporal, or anterior parietal areas on the same side over pathways in the trigeminal nerve; and (2) pain arising from intracranial structures below the tentorium cerebelli is referred to the postauricular, occipital, suboccipital, and upper nuchal areas on the same side over pathways in the glossopharyngeal, vagus, and upper three cervical nerves. The major exception that the site of headache fairly closely overlies its intracranial

origin is that pain arising from the posterior half of the sagittal sinus or the upper surface of the transverse sinus is transmitted over a branch of the first division of the trigeminal nerve and is referred to the frontal area, a considerable distance forward.

Extracranial Structure

The surface tissues of the head are pain-sensitive. Common sources of headache are the arteries of the scalp and face, the orbital contents, the muscles of the scalp and upper neck, the linings of the nasal cavities, the external and middle ears, and the teeth. At first, pain arising from these structures is well localized, but later the pain extends upward or backward and is a direct source of headache.

Mechanisms

Traction Upon Intracranial Structures

"Jolt headache" probably arises from traction by the brain as it abruptly shifts in position within the skull case. Experiments indicate that jolt headache is due to the pull upon the major arteries anchoring the brain at its base.

Patients with expanding intracranial lesions (tumors, subdural or intracerebral hematomas, or abscesses) often complain of headache. The pain is aggravated by coughing or sudden standing, and head

movement. This headache is most likely due to traction rather than increased intracranial pressure. Often "the location of the headache can be related directly to distortion of adjacent structure, as when pain is noted in the fronto-orbital area on the side of a sphenoidal ridge meningioma, or when postauricular headache accompanies the growth of a neurinoma in the cerebellopontile angle. Headache may also extend widely whenever expanding masses produce gross displacement of the brain, leading to pressures upon the tentorium cerebelli, herniations at the incisura or foramen magnum, and distortion of multiple structures. In such situations the headache loses all localizing diagnostic value."[1 p 8]

Dilatation of Cranial Arteries

Dilatation of cranial arteries is one of the processes commonly observed in a patient with headache. The symptom complex soon becomes aggravated by edema of the arterial walls, contraction of the muscles of the neck and scalp, and local edema in the extracranial tissues. Schumacher and Wolff have shown that "the vascular headache which follows the injection of histamine can be prevented by increasing the intracranial pressure and can be temporarily relieved by subjecting the patient to a caudal centrifugal force of two or three G. Patients with spontaneous migraine,

as well as headaches secondary to hypertension, obtain no relief from increases in the intracranial pressure."[2 p 3] Since the headache of these latter two types can be promptly relieved by vasoconstrictors such as ergotamine, they must also be 'vasodilating' headaches but the vessels involved are extracranial. Direct observation of these vessels during an attack supports this hypothesis.

Symptoms of migraine headache may be the result of constriction in branches of the cerebral circulation, and that this, as part of an inefficient attempt to maintain homeostasis, then leads to reactive relaxation of arterial tone in other cranial arteries, usually extracranial. A few patients with various forms of migraine present during the headache other signs of central neural activity, such as miosis and nasal congestion ipsilateral to the headache. These features suggest an integrated parasympathetic discharge of which the vasodilator headache is only one complaint.

Inflammation

Headaches due to inflammatory lesions inside the head or on the scalp may be intense, but are usually overshadowed by other symptoms of the underlying disease. When the source is intracranial, the headache is similar to drainage headache. The headache is slightly relieved when the patient lies down, is easily augmented

by mild head movement, and is often accompanied by nuchal rigidity and a mild fever. Headache caused by an extracranial source is usually localized and is unaltered by changes in arterial pressure or by head movement. It is rarely intense and its source is usually manifest to inspection and palpation.

Contraction of Skeletal Muscles of Head and Neck

Pain produced by contraction of skeletal muscles of head and neck is often transiently made worse by rapid or slow head movements. Local massage and the application of heat usually gives some degree of relief. This type of headache "is encountered clinically as a 'secondary headache' in association with almost any of the major forms of headache,"[1 p 18] especially when the patient is anxious and tense. Postural strain may contribute to the development of muscle tension in such occupations as typing and drafting.

Headaches Spreading from Diseases of the Eyes, Nose, and Paranasal Sinuses, Ears and Teeth.

Frontal or temporal headaches are sometimes noted with disease of the eyes, nose, sinuses, and teeth, all using pain pathways over the fifth cranial nerve. An example is the "ice-cream" headache which spreads from facial and nearby structures to the upper half of the head. When pain spreads from the ear it may be noted

either frontally or in the back of the head, presumably because of the intermediate position of ear structure and their multiple somatic sensory innervation by branches of the fifth, seventh, ninth, and tenth cranial nerves.

III. Headache is a Symptom of Systemic Disease

Cardiovascular Disorders

The degree of hypertension a patient has is not directly proportional to the severity of headache, although it does seem more related to the diastolic pressure than the systolic pressure. Head pain in these patients is usually aggravated by lying down which suggests it may be due to the mechanical effect of increased intravascular pressure. The assumption that the pain of hypertensive headache is not due to excessive pulsation is born out by the experiments of Wolff[2 p 35] who showed that the hypertension headache cannot be abolished by increasing intracranial pressure which tends to compress the cerebral vessels and to reduce their pulsation. Thus, the mechanism of head pain in hypertension is not known other than the possibility of it being a direct effect of increased intravascular pressure.

Head pain in arteriosclerosis is probably produced by excessive pulsation in a torturus vessel.

These headaches are aggravated by lying down and often these patients must be propped up in a chair to sleep.

Infection

Headache is associated with nearly every febrile infection. Wolff believes this to be "a result of increased pulsation of the larger branches of the internal carotid system."[2 p 37] The head pain is usually worse in infectious mononucleosis, viral hepatitis, typhoid fever, and malaria. It usually responds well to simple analgesics.

Metabolic Diseases

Headache may be a symptom of hypothyroidism, hypoglycemia, and diabetes. Headache is commonly present in hypothyroid patients although the mechanism isn't understood. The retention of fluid, poor muscle tone, and depressed state of mind may be factors responsible for the headache. Headache is occassionally seen in patients with hypoglycemia and it is usually dull and frontal and disappears promptly when the hypoglycemia is corrected. Diabetics often complain of a dull, frontal, or vertical headache. It is usually seen in mild diabetics and is relieved by proper diabetic therapy.

IV. Differential Diagnosis and Treatment of Head Pain

Tension Headache

Tension headache is defined as the pain resulting

from sustained contraction of the skeletal muscles of the head and neck. Although the term describes a specific clinical picture, it is inadequate and confusing, for tension refers to the art of stretching or a feeling of tightness. The contraction of skeletal muscles as a part of the pathogenesis of this type of headache has been confirmed by electromyography. Prolonged contraction of muscles produces a resistance to blood flow with a resultant ischemia and pain.

The cause of tension headaches is related to psychophysiologic disturbances. The fundamental psychic factors are sometimes subconscious, although most patients are aware of their anxiety. The headache may be brought on by economic, social, or physical environmental demands. The personality of the patient who suffers tension headache is similar to the patient with migraine in that both of them are ambitious, perfectionistic, set high goals for themselves, and are sensitive to criticism.

Symptoms are described in a variety of ways; they feel discomfort all over the head, or they may speak of a fullness, drawing, or tightness as if they were constantly wearing a tight cap. A high percentage of patients locate the center of their discomfort in the back of the head and neck. Vigorous massage of the neck is comforting. They have unilateral or bilateral aching

in the occipital, parietal, temporal, or frontal regions and often have tender spots in the scalp. Dizziness or vertigo may be associated. It usually is described as a steady ache or cramp and does not pulsate. It may appear at any time and may last for weeks or months. Occassionally one has associated nausea and vomiting. Physical examination reveals no neurological findings other than muscle contraction and spots of tenderness.

The treatment of tension headaches is based on the fact that the etiology is primarily psychological rather than physiologic. The physician should offer the patient a reasonable and adequate explanation of the cause of his symptoms. Simple non-addicting analgesics in combination with muscle relaxants are usually effective in the symptomatic treatment of tension headache. Psychotherapy is often needed in the prevention of tension headache and is the only way the patient can be led to develop the necessary insights into his reaction to life.

Vascular Headaches

Vascular headaches are at times difficult to differentiate into specific entities. There is an overlay of symptomatology and a misinterpretation is easily made. They will be discussed under the following headings.

1. Migraine headache
2. Allergic headache
3. Hypertensive headache

4. Menopausal headache
5. Arteriosclerotic headache

Migraine

The word "migraine" literally means one-sided headache and is derived from the Greek hemi and kranion (meaning half a skull). Periodic unilateral, recurrent headache, often associated with nausea and vomiting, and preceded by a variety of prodromal symptoms, including visual disturbances are a part of the symptom complex of migraine headache. The incidence of migraine in the general population has been estimated to occur at five to ten per cent and in one series constituted eight per cent per eighteen thousand patients seen in the general practitioners office.[5 p 326] Migraine is more prevalent in women than in men and Alpers states[2 p 99] the ratio is 9:1 although I have found some discrepancy in this ratio. It occurs in all social and economic groups but is more prevalent in urban areas among active housewives, executives, and professional people.

One concept of the mechanism of migraine is obtained when the migraine syndrome is divided into the preheadache period and the painful period. The prodromal phenomena are probably a result of vasoconstriction in branches of certain blood vessels supplying the cerebrum and possibly vessels in the retina. The vascular changes produce visual, sensory, auto-

-nomic, and motor responses prior to the onset of headache. This suggests a neurogenic origin rather than generalized response of smooth muscle to chemical agents. The painful phase is associated with vasodilatation during which the cranial vessels have altered sensitivity and increased amplitude of pulsation. Arteries are usually extracranial and anything that diminishes the cranial arterial pressure reduces the headache which doesn't seem to be influenced by increased intracranial pressure. Recent studies show an accumulation of a substance of low molecular weight bradykinin, which may be responsible for lowering the pain threshold.[4 p 12] Continued dilatation of the blood vessels results in their becoming hard, tender, and rigid. The afferent impulses transmitting the sensation of pain are carried on the trigeminal, glossopharyngeal and vagus nerves and the upper three cervical nerves.

Emotional stress is capable of precipitating an attack of migraine. These patients are emotionally stable inbetween attacks and the emotional storm occurs only after the vascular changes have taken place. In many instances the migraine patient is a picture of calm and controlled behavior but at the time the attack occurs mood disturbances and personality expressing feelings of hostility frequently emerge.

The personality structure indicates that they are meticulous, perfectionistic, neat in their appearance, and rigid in their thinking. It seems that the basic issue stems from the arousal of hostility. That emotional disturbances are of great importance as precipitating factors can't be doubted, but that they are the underlying cause remains to be demonstrated. Many believe allergy to be the dominate factor.

Migraine usually begins in childhood or in adolesence; as age advances it tends to become less severe and finally to disappear. It is probably due to an inherited abnormality, since a strong family history of migraine is common. Goodell considers that the inheritance of the migraine trait is through a recessive gene with a penetrance of seventy per cent.[4 p 12] Most observers report an incidence of fifty per cent or more in the offspring of a patient.[1 p 209]

The symptomatology of migraine headache is characteristic. In some patients there may be no clear-cut prodromata. Visual phenomena, including scintillation, stars or hemianopsia, and photophobia are commonly present. Disturbances of vision usually clear before headache begins and are present on the contralateral side of the head pain. Other symptoms preceding the headache include lethargy, paresthesia, chilliness, flushing, weakness of extremities and facial

edema. The pain itself is usually unilateral in onset and most often localized to the orbital, frontal, or temporal regions. The headache progressively becomes more severe and may spread to involve the entire head. The throbbing quality is replaced by a steady ache and may persist for hours. Other associated symptoms include nausea, vomiting, increased perspiration and salivation, diarrhea, paresthesia, cold extremities, vertigo, tremors, pallor, and abdominal distention.

Table I

Diagnostic Criteria of Migraine Headache[2 p 103]

A. Hereditary background
B. Constitutional pattern
    1. Motion sickness in childhood
    2. "Bilious spells" in childhood
    3. Short stature, delicate build
    4. Low basal metabolic rate
    5. Flat glucose tolerance curve with late hypoglycemia
C. Personality pattern
    1. Hypersensitivity to all stimuli (smell, light, temperature change)
    2. Excessive orderliness
    3. Evening vigor and morning lassitude
    4. Negativism
D. The Attacks
    1. Preceding vigor
    2. Scotomata
    3. Gradual development of pain in the head
    4. May occur on either side
    5. Accompanied by photophobia, nausea and vomiting
    6. Response to vasoconstrictors

The etiology of migraine headache remains controversial. Psychogenic, metabolic, hormonal, genetic and autonomic nervous system roles have been investigated.

After reviewing the pros and cons on these various possibilities, I feel that the mechanism is most likely related to an inherent lability of the autonomic nervous system. There are commonly other symptoms relative to disturbances in the autonomic nervous system. These include miosis, nasal congestion, salivation, excessive perspiration, nausea and vomiting, diarrhea, pallor, abdominal distention, variations in peripheral vascular tone, and increased motility of the gastrointestinal tract. It is difficult for me to perceive that a specific personality type is characteristic of the patient with migraine headache.

Migraine may require no treatment other than an explanation of the symptoms and reassurance by the physician. Therapy is directed towards the early painful secondary stage of vasodilatation. Ergot preparations are the most effective and their effect is due to the action of the substance on the smooth muscles of the blood vessels, producing a constriction of these vessels and a decrease in amplitude of pulsation. Treatment is seldom required during the phase of the aura, but inhalation of amyl nitrite may abolish the disturbance. The milder attacks usually respond to the usual analgesics, acetylsalicylic acid (0.6 to 0.9 gm.) with or without the addition of caffeine citrate (25 mg.) and codeine (60 mg.). The more severe attacks respond only

to ergotamine and dihydroergotamine.

Allergic Headache

Allergic susceptibilities may occassionally represent a "trigger mechanism" for the precipitation of a "cluster type headache". It is Alpers belief[2 p 115] that it is this type of headache that is referred to as "histamine cephalgia" and that it is often precipitated by specific food allergies. The headache can be produced in a normal subject by rapid intravenous injections of 0.1 mg. of histamine acid phosphate. By means of experimentation with the latter the following has been established.[6 p 21]

1. Headache doesn't arise from an extracranial structure, because arrest of the circulation to the scalp before injection of histamine does not prevent the pain.
2. Headache arises from a structure innervated by the trigeminal nerve, since it does not occur on that side of the head when the sensory root has been severed or the ganglion injected with alcohol.
3. It probably arises from the meninges, as it is greatly increased by shaking the head gently, and the meninges are the structures which will be chiefly strained in this movement.
4. It arises from the territory supplied by the

internal carotid artery, since injection of histamine into the internal carotid produces headache, but injection into the external carotid does not.

The headache occurs predominately in males (9 to 1) and the patients usually give no history of childhood stigmata that are found in the migraine patient. Its highest incidence is in patients between twenty-five and forty-five years of age. These headaches usually occur in "clusters" occurring several times a day for periods of six to ten weeks. The clinical picture includes periodic, agonizing unilateral pain involving the eye or temple or both, associated with injection and tearing of the ipsilateral eye and stuffiness of the nose. The mechanism of vasodilatation during an attack is unknown, but it is felt the prime vessels are triggered by neural and chemical factors.

Table II

Differential Diagnosis[4 p 16]

	Cluster Headache	Migraine
Family history	Less than 20%	Over 60%
Average age of onset	28 yrs.	Less than 20 yrs.
Bouts	Yes	No
Paroxysms	More than 1/day	Less than 1/week
Prodromata	Shorter	Longer
Distribution	Mostly orbital	Mostly supra-orbital or cranial
Visual disorders and G. I. symptoms	Rare	Common
Lacrimation, nasal congestion	Common	Very rare

Therapy of "cluster headache" has included antihistamines, vasoconstrictors, cortisone, and dietary changes all with little success. Symptomatic treatment of an attack is best accompolished by use of ergotamine preparations as noted in migraine. Desensitization with histamine does not seem to alter the course of the disease. Proper dietary eliminations does seem to help a few patients.

Hypertensive Headache

Hypertension is the cardiovascular disorder most commonly associated with headache. Elevation of the diastolic pressure is more important as a causative factor and headache seems to be more prominent in patients with secondary renal impairment and retinal changes. At this stage the headache seems to be more related to widespread vascular damage and not entirely based on changes in mechanical or hydrodynamic changes.

Headaches secondary to hypertensive vascular changes vary in intensity from time to time. It is usually gradual in onset and is often present on awakening in the morning. It is characteristically unlike the histamine (cluster) and migraine headache. It is usually a vertical, frontal, or occipital pain lasting several hours and sometimes made worse by a change in position. If it occurs during sleep it is often relieved by propping the head and back with

several pillows or even sitting in a chair.

Usually mild episodes of hypertensive headache are relieved by simple analgesics and sedative mixtures. If the headaches are more severe and prolonged it is usually necessary to reduce the blood pressure by antihypertensive drugs and with salt restriction. Sympathectomy will usually give relief even if the hypertension is not reduced. Hexamethomium, which acts at the ganglion and is an effective blocking agent, is indicated when there is no response to other medical therapy.

Premenstrual Headache

Premenstrual headache is quite rare and its cause is unknown. Most functional symptoms of the female are usually related by her to the menstrual cycle. "Whether it be headache, hives, or pain, it is likely to be either aggravated or relieved by the approach, performance, or completion of the menstrual flow. Thus all headaches occurring in women whether they be migraine, allergic, tension, or hypertensive are likely to be worse just before the period."[2 p 121]

This headache frequently occurs only during the day or so preceding the menstrual flow or during the first two days thereof. The pain is boring in character and is limited to the retro-orbital, frontal, and midvertical areas, which suggests congestion and swell-

-ing of the pituitary gland as a possible cause. It has been observed by MacNeal and Alpers[2 p 122] that in a number of cases the anterior and posterior clinoid processes formed a nearly complete 'roof' over the sella turcica, thereby perhaps reducing the room for expansion. Pituitary suppression therapy with thyroid, estrogen, and progesterone have been followed by relief in some cases. The simple approach is to give simple sedatives and analgesics starting a day or two before the headache is expected and continue them a day or two beyond them.

Menopausal Headache

Many women are subject to periodic vascular headache at the time of menopause. It does not occur when the menstrual cycle is still in progress nor does it occur in men. It is more common among those women who have other menopausal complaints including hot flashes, palpitation, and sweats. The headache usually follows an attack of hot flashes. It is usually of sudden onset, severe, and lasts several hours, and may awaken the patient at night. It usually is not relieved by simple sedatives or analgesics.

Many theories involving estrogen deficiency, excess of gonadotrophin, fluid and salt retention, and pituitary swelling have been investigated to explain the mechanism of this type of headache, but none are

supported by good experimental evidence. In the face of these theories, the menopausal headache has been treated by gonadotrophic hormones, estrogens, androgens, progesterone, and salt and fluid restriction, with occassional success and frequent failure. However, the most satisfactory approach to this problem is by the appropriate oral administration of moderate doses of estrogenic substances in cyclic fashions.

Arteriosclerotic Headaches

Arteriosclerotic headache occurs in patients with marked tortuosity and sclerosis of visible vessels but without significant hypertension. The pain is usually worse when lying down and disappears after assuming the erect position. It is not as severe or as well localized as those headaches seen in true aneurysm. It is almost never seen in those patients under sixty years of age. Findings include tortuosity of the brachial, temporal and radial vessels with an increase in pulse pressure and the accompanying straightening of the artery with each systole. It is possible that this straightening phenomenon occurs in the cerebral vessels with each systole which tends to be greater when the head is low and the force of the cardiac thrust is therefore unopposed by the force of gravity. Simple analgesics and sedatives are of no value in these patients and the most successful therapy is simple elevation

of the head during the sleeping and resting hours.

Local Intracranial Lesions

Headaches of organic origin must be thoroughly investigated. There must be proper selection of patients. In the ambulatory patient with headache the following criteria are used by Alpers to refer a patient for a neurological survey. "(1) Patients who develop acute, disabling headache, with or without other symptoms referrable to the nervous system; (2) patients with longstanding headache for which no cause has been determined, associated with increasing disability, and refractory to treatment; (3) patients with headache of varying duration who develop neurological symptoms such as seizures, diplopia, or other bona fide neurological complaints; (4) patients not previously afflicted with headache who develop unilateral headache with neurological symptoms; (5) patients with persistent headache after head injury."[2 p 41]

The studies that are commonly used at present include complete neurological examination, roentgenogram, audiogram and Barany tests, arteriogram, pneumo-encephalogram and pneumo-ventriculogram.

Spinal fluid studies should be routine in properly selected cases of headache. The spinal fluid pressure is the most important feature, and it is often elevated when a tumor is present. The spinal fluid protein is

often increased in presence of tumors, especially gliomas lying close to the surface of the brain or the ventricle, in cerebellopontine angle tumors, and in meningiomas. The other chemical determinations are not significant.

The electroencephalogram may give useful information regarding organic disease. It is more valuable in cerebral than in subtentorial processes. The EEG findings should correlate well with the clinical findings. The usual findings consist of abnormally slow waves of high voltage which vary from one-half to seven cycles per second, delta or theta in type, and range in amplitude from forty to one-hundred and fifty microvolts. If one has a deep cerebral tumor the findings are those of bilateral cortical involvment and localization is not distinct. Infratentorial neoplasms produce diffuse abnormality. Audiogram and Barany tests are indicated in cases of headache associated with tinnitus, deafness, and vertigo and clinical findings of absent corneal reflex on one side or unsteadiness in walking and standing.

Arteriogram is a useful device for determining vascular disease as a cause of headache. It helps to determine if aneurysm or vascular anomalies are present. It is helpful in differentiating internal carotid

artery thrombosis from brain tumor and is used in the diagnosis of brain tumor per se.

Pneumograms are utilized to determine the absence or presence of a tumor. Pneumoencephalogram is used only in the absence of increased intracranial pressure. If there is an increased spinal fluid pressure a ventriculogram becomes necessary. They are of no value in the presence of vascular disease.

Headache is one of the symptoms of cerebral tumor. It at first occurs in attacks lasting only a few seconds to three hours. Frequently as the tumor grows, headache may increase in frequency, duration, and intensity. The pain may be slight but can be severe and is a deep aching pain. Most frequently the headache is bilateral and frontal but there is some relation between the sites of pain and the tumor. Thus occipital headache is more frequent with tumors below than with those above the tentorium, and when headache is unilateral the tumor is on the same side in ninety per cent of the cases. Headache is more frequent in quickly growing tumors, such as gliomas, than in the slower growing varieties, such as meningiomas. "The headache of brain tumor is explained by traction or distortion of neighboring pain-sensitive structures such as the large venous sinuses and their tributaries, the middle meningeal artery, direct pressure on the cranial nerves,

dilatation of intracranial arteries, and inflammation in or about any intracranial pain-sensitive structure. It has been suggested that the headache associated with increased intracranial pressure is due to dilation of the pial arteries. In the case of headaches associated with increased pressure posterior head pain may occur in supratentorial tumors and frontal headache with posterior fossa tumors. In these cases it is assumed that there is distant traction on pain-sensitive structures caused by distortion of the brain which accompanies the growth of the tumor."[2 p 46]

Headache is a common finding in subdural hematomas, subarachnoid hemorrhage, and meningitis. The headache of acute subdural hematomas is difficult to separate from the headache of acute head injury. The headache of chronic subdural hematomas is one that follows head injury, is generalized, constant, and severe. It tends to be persistent and is due to traction on the sagittal sinus and its tributary veins. The headache of subarachnoid hemorrhage is sudden and apoplectic in development. It may be localized or generalized and is increased on shaking the head and by jugular compression. It has been supposed that the headache arises, as does that of meningitis, from inflammation of the meninges evoked by the presence of blood, and it is true that neck rigidity

is a constant feature of subarachnoid hemorrhage, as it is in meningitis. Subarachnoid hemorrhage most commonly results from rupture of an intracranial aneurysm. In more than fifty per cent of the patients the onset of intense pain is accompanied by vomiting, grogginess, neck rigidity, and loss of consciousness.[5] The headache of meningitis is constant, generalized, usually severe, and associated with evidence of meningeal irritation such as neck stiffness and Kernigs sign.

Headache is one of the most frequent symptoms following injury to the head and occurs in approximately sixty per cent of head injuries.[3] The majority of patients with chronic post-traumatic headaches have no known intracranial abnormalities to explain their headaches. In studies by Freidman[3] there is little correlation between severity of the injury and the development or severity of the post-concussion syndrome. It has no special characteristics although many refer to it as a pressure or bandlike sensation, others describe a recurrent, periodic, throbbing pain. It varies in frequency and duration and may be precipitated by emotional stress, change in posture, fatigue or effort. Present evidence indicates that in the majority of patients it is caused by sustained contraction of the skeletal muscles of the neck resulting

from the emotional reaction to the injury. These patients are best treated with mild analgesics, muscle relaxants, and psychotherapy.

Extracranial Causes of Headaches

Mechanical

There is a certain group of headaches whose pain is produced entirely by stress on the voluntary muscles attached to the scalp. Faulty posture will produce these strains by keeping the cervical paraspinal muscles on a constant stretch. This type of headache is seen in the desk worker, the painter, the small woman driving a car, the reader in bed, or the slouching television viewer. Elevating the desk, the use of prismatic lens, raising the seat of the car, reading and watching television in a straight back chair may be helpful in the above conditions. Such simple procedures relieve the stress placed on the involved muscles.

Miscellaneous

There are several other etiologies of head pain which should be mentioned. The eye is a common source. Errors of refraction are probably a cause of headache and they respond readily to the wearing of proper lenses. Ophthalmologists state that headaches may result from muscle imbalance. Glaucoma is another

source of headache. Chronic paranasal sinus disease is rarely a cause of headache, although an acute suppuration in a sinus will cause temporary, severe pain in the involved region. Idiopathic neuralgias produce an intermittent type of paroxysmal lancinating pain limited to the area supplied by a single peripheral nerve. Examples of such neuralgias include trigeminal neuralgia, sphenopalative neuralgia, occipital neuralgia, and supra-orbital neuralgia. All of the latter etiologies of head pain are extensive subjects and I purposely have failed to discuss them in detail.

V. Summary

The purpose of this paper was to investigate the evaluation and management of the patient with headache. Since this is such a broad subject it has been limited to the mechanisms of head pain, its differential diagnosis, and basic therapy.

For one to understand the mechanism of headache one must understand the basic pathophysiology of pain. The pain-sensitive structures have been named and, of course, must be involved by some abnormal mechanism to produce pain. These include traction upon intracranial structures, dilatation of cranial arteries, inflammation, contraction of skeletal muscles of head and neck, and headaches spreading from disease of the eyes,

nose and paranasal sinuses, ears, and teeth. These mechanisms are discussed in detail.

Headache is a symptom of systemic diseases, including cardiovascular, infectious, and metabolic disorders. Head pain in hypertension seems to be more related to diastolic pressure than to systolic pressure. Headache in arteriosclerosis is probably produced by excessive pulsation in a tortuous vessel. Nearly every febrile infection has an associated headache. Headache is also a symptom of hypothyroidism, hypoglycemia, and diabetes.

The differential diagnosis and treatment of headpain is divided into four main categories: 1) Tension headache; 2) Vascular headache; 3) Local intracranial lesions; 4) Extracranial cause of headache. These categories are dealt with individually from a diagnostic and therapeutic standpoint.

Tension headache is defined as the pain resulting from sustained contraction of the skeletal muscles of the head and neck. The cause is related to psychophysiologic disturbances. The symptoms are described in various ways and the physician should be familiar with these to make a diagnosis. Physical examination reveals no neurological findings other than muscle contraction and spots of tenderness.

Vascular headaches have been divided into the various types: 1) Migraine; 2) Allergic headache; 3) Hypertensive headache; 4) Premenstrual headache; 5) Menopausal headache; 6) Arteriosclerotic headache. These entities have been discussed from an etiologic, diagnostic, and therapeutic standpoint. The vascular system seems to be related to the production of pain and the variations in it are responsible for the variations in the symptoms produced.

When a headache is secondary to specific intracranial disease a thorough investigation must be done including the use of various laboratory tools. Headache as a component of the symptom complex of brain tumors, subdural hematomas, subarachnoid hemorrhage, meningitis, and trauma is discussed. The onset, duration, location, and precipitating factors of the headache are presented.

Extracranial causes of headache are usually related to stress on the voluntary muscles attached to the scalp or to specific disease processes in closely associated structures such as eyes, ears, nasal and paranasal sinuses, and teeth. Idiopathic neuralgias produces an intermittent type of paroxysmal pain. There are other miscellaneous causes of head pain which I have failed to mention, but are not within the limits of this paper.

VI. Conclusion

Headache is a symptom of many disease processes including intracranial, extracranial, or systemic disease. When a patient is seen in a physicians office with the chief complaint of headache, this should be evaluated from an etiologic standpoint. The physician should proceed to make a sound explanation to the patient. In attempting to determine the underlying pathologic cause, it will require the physician to understand the pathogenesis and physiology of head pain. He must not only understand the mechanisms, but also must be fully aware of the differential diagnosis of headache. Whenever headache is encountered, it is a symptom which challenges accurate observation and straight thinking, for the differentiation and causes of headache are as much dependent on sound clinical observation as on laboratory aids.

For every patient that is seen with headache, the physician must decide whether it is on an organic (structural) or a functional basis. This is true with many symptoms, but for proper therapy to be given this fact must be known. Only after adequate history, physical examination, neurological examination, x-ray, and laboratory aids can this differentiation be made. Hence evaluation of the patient with headache must be comprehensive and it requires a thorough knowledge of the pathologic physiology and the basic personality of the patient.

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