pressure within the skull, or say, "I have a feeling as if there is a tight vise around my head." When you hear a complaint of that kind, you can almost be certain that you are dealing with a tension headache, a psychologically induced headache. This, of course, is subject to exceptions. Dr. Friedman will perhaps remember that we saw, some ten years ago, a nurse who had headache of this type but in whom a brain tumor developed. It is probable that the brain tumor was not the cause of the headaches, which she had been having for many years, but that we were dealing with a patient who had tension headaches and who later happened to have a brain tumor.

In summary, a great variety of morbid conditions, both intracranial and extracranial, can cause pain in the head and produce headaches, but chronic, recurring headaches are usually due to migraine or psychological tension headaches. It is possible that the mechanism of the two are closely related.

Migraine and Tension Headaches

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HEADACHE IS ONE of the most common complaints of modern man. It has been estimated that 12 per cent of the urban population of the United States have chronic headache. Because in the vast majority of these persons the symptoms are either due to migraine or are primarily associated with an emotional disturbance (tension headache), discussion in this presentation will be limited to headaches of these two types.

MIGRAINE

Migraine is a symptom complex characterized by periodic attacks of headache, with or without associated symptoms, in a person who has a background of well-being between attacks. Upon analysis of one thousand patients with migraine the following characteristics were noted: A history of migraine in the family occurred in 65 per cent. Seventy per cent were females, or a ratio of more than two to one. The headaches began before the age of 20 in over half of the patients; onset after age 40 was exceptional. The attacks occurred less than once weekly in 60 per cent of the patients. Prodromata were present in three out of four patients, and the quality of the headache was throbbing in 80 per cent.

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Cause

It is difficult to explain all cases of migraine on any single etiological basis. Most observers have emphasized heredity as an important factor in migraine. There is no evidence, however, with regard to the exact mode of this inheritance, nor is there any evidence as to what specific abnormality may be inherited. In some instances, and perhaps to a certain degree in all cases, there may be an inherent functional instability of the autonomic, endocrine, vascular or enzyme systems. There is also a real possibility that migraine occurs on a familial basis and is related to environmental rather than hereditary factors.

From their investigations, Jimenez Diaz and his associates concluded that substances of an acetylcholine type were released in excess in migraine patients during stress. They postulated that the normal choline acetylase system is different in these patients.

Many patients with migraine undergo changes in fluid balance and fluid distribution during the headache. Such changes are both local and general. A number of investigators have reported that water, sodium, potassium and creatinine are significantly decreased in excretion prior to and during the early phases of the migraine attack. Increased rates of excretion of these substances were usual as the headache subsided. Studies by Schottstaedt and Wolff, as well as studies in which I have taken part, indicate that fluid and electrolyte changes are not related causally to the onset, intensity or duration of the migraine attack, but are manifestations of bodily changes accompanying adaptation reactions during stressful periods.

It has been emphasized that the frequent association of migraine with puberty, menstruation and menopause indicates gonadal etiology. There is not, however, any valid physiologic or biochemical data to support this hypothesis.

A relationship between migraine and allergic sensitivity has been suggested for over a hundred years. There is no doubt that headaches may occasionally be precipitated by allergens in certain susceptible persons, but investigations of the last few years have indicated the role of allergic reaction as the cause of migraine has been overestimated. Theoretically allergy offers many hopeful and suggestive possibilities as an etiological agent in migraine, but substantial proofs are yet too few and inconclusive to warrant much encouragement.

The importance of psychologic factors in migraine has long been recognized. Studies indicate that in a great majority of patients with migraine there is a "personality pattern," one of the features of which

is an abnormally strong reaction to environmental stress. It is doubtful whether psychologic mechanisms alone can be responsible for the chain of physiologic events noted in the migraine attack without some underlying lability in the physiology of these patients.

From available information we may postulate that patients with migraine have inherited a neurophysiologic system which is limited in its capacity to handle a variety of stresses. Furthermore, they have an insecure personality pattern which interferes with their adjustment to their environment. This sets a pattern of function which the limited physiologic capacity of the individual is unable to stabilize, resulting in cranial vascular changes which produce headache.

Mechanism

The attack consists of three distinct vascular changes. The prodromal or aura stage is associated with vasoconstriction of the intracranial arteries and clinical phenomena, such as scotomata, hemianopsia, paresthesia and depression. The second phase is associated with vasodilatation, at which time the cranial vessels have altered sensitivity and increased amplitude of pulsation. It is hypothecated that the sensitivity of the blood vessels is due to local vascular changes, particularly in the arterioles and capillaries, which result in the accumulation of fluid and a substance in the tissues that lowers the pain threshold. The pain at this stage is usually throbbing and is aggravated by anything that raises the venous pressure, such as stooping or straining. Associated symptoms are common and include anorexia, nausea, vomiting, diarrhea, hyperhydrosis and other signs of autonomic nervous system instability. In the third phase there is edema of the affected vessels, which also become hard, tender and swollen. The head pain at this stage is a steady ache. During or following these stages there may be contraction of the neck muscles (and muscular contraction pain may develop). This spasm of the muscles is a reaction to the initial pain and may outlast it.

Considerable attention has been given to the psychological factors in migraine attack. Early reports in the literature emphasized that migraine patients were psychologically stable between attacks and that the emotional storm occurred only after vascular changes had occurred. In recent years studies have indicated that many patients with migraine are meticulous, neat in appearance, rigid in their thinking and excessively aggressive toward their environment. It is often noted that in their earlier life they had considerable insecurity with resulting tensions, which are manifested in inflexibility, overconscientiousness, meticulousness, perfectionism and resentment. My studies, however, have indicated that there is little evidence of specificity of the precipitating psychodynamic factors. Not all patients with migraine are compulsive, perfectionistic or rigid. Repressed hostility is an extremely common factor in persons who do not have migraine. Nevertheless, psychological factors play an important part in the dynamic mechanism of a migraine attack, and an understanding of these underlying psychologic mechanisms is important in the management of the problem.

Diagnosis

Diagnosis of migraine is usually made from the history. However, a detailed physical and neurological examination is necessary for each patient. In an occasional case, special studies of the eye, nose and paranasal sinuses and tests for offending allergens are indicated. Routine laboratory studies such as examination of blood and urinalysis—although in most cases unrevealing—should be a part of the work-up. Roentgenograms of the skull and cervical spine, electroencephalograms and studies of visual fields may be necessary to exclude organic causes of headaches. In my experience provocative tests, such as giving nitroglycerine and histamine, are not of much value as a diagnostic aid.

Diagnostic manifestations of migraine include:

1. Recurrent throbbing headaches, usually unilateral at onset, occurring against a background of relative well-being.
2. Nausea, vomiting, and irritability occurring at the height of an attack.
3. Temporary visual disorders preceding the headache, including scintillating scotomata, photophobia, hemianopsia or blurred vision.
4. History of migraine in the immediate family.
5. Symptoms such as paresthesias, speech disorders, dizziness, sweating and other vasomotor disorders.
6. Relief by ergotamine.
7. Personality characteristics of inflexibility and shyness in childhood, giving rise to adult perfectionism, rigidity and resentment, ambitiousness and efficiency; a constitutional predisposition to sustained emotional states.

The clinical manifestations of migraine indicate widespread bodily disturbance. Although attention is usually first called to the headache, there are numerous other symptoms to be considered as part of the entire picture.

Treatment

The most commonly employed treatment of the migraine attack is, undoubtedly, the use of ergot preparations, particularly ergotamine tartrate. The

value of this method has been confirmed by clinical evidence of its beneficial effects and experimental observations in the laboratory. Ergot was first employed for this purpose by Eulenburg as far back as 1873 and by Campbell in 1894. It was only, however, within recent years that careful studies by Wolff and others demonstrated that ergotamine acts by promoting vasoconstriction rather than vasodilatation, and that sympathetic blockade does not predominate. The benefit that follows the taking of ergotamine for an attack of migraine is probably due to the direct 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. This is particularly noticeable in the circulation of the external carotid artery.

A combination of ergotamine tartrate and caffeine, which acts as a synergist to the ergot alkaloids, is the most effective medication for the treatment of a migraine attack. Rectal use of ergotamine has proved empirically to be most efficacious, especially when oral medication cannot be retained. More recently many forms of ergot derivatives have been made available as proprietary preparations incorporating antispasmodics, sedatives, central nervous system stimulants, antiemetics and so on. These preparations can be used to suit the individual patient's need.

Patients who tolerate ergotamine tartrate poorly can be given dihydroergotamine methanesulfonate (DHE-45) parenterally. To produce an equivalent therapeutic effect dihydroergotamine must be given in doses twice as great as those of ergotamine tartrate.

It is most important to administer these drugs early in the course of the attack and in adequate doses. The optimal time of administration is the prodromal period or at least immediately after the onset of the headache.

(Information as to dosage, side effects and contraindications in the use of the ergot preparations is readily available in the literature.)

Numerous vasodilators have been used in the prodromal stage (vasoconstrictor phase) in order to abort the attack, and in the headache stage (vasodilator phase) to lower the blood pressure sufficiently to reduce the arterial pulsations. Except for a few isolated cases, these preparations are of little value.

Analgesics and sedatives are sometimes indicated if the headache has been present long enough for edema to take place and the vessels to become firm and tortuous.

Prophylactic

It is well known that in most patients with migraine, suitable psychotherapy may be of great value in reducing the intensity and frequency of attacks and may even result in disappearance of attacks for a considerable time. The details of this kind of treatment will not be a part of this discussion.

Adequate relaxation, improvement in sleep and correction of any physiologic abnormalities are aids in reducing the frequency of the attacks. In a few cases there may be evidence that the precipitating mechanism is of an allergic, endocrine or "metabolic" nature, but these factors are too rare to have significant application to therapy. However, when an allergen is the cause, it should be removed or the patient desensitized. If the onset of migraine is associated with the menstrual period, the use of progesterone or testosterone, or both, may be helpful. In some patients a premenstrual diet that restricts the daily intake of salt, in addition to giving a diuretic, seems to be beneficial.

Drugs such as histamine or nicotinic acid are of no value. Sedatives to reduce emotional tension are limited in their usefulness. More recently we have used reserpine and chlorpromazine hydrochloride (thorazine) as interim treatment. In a small number of patients, the result is encouraging, but final conclusions are yet to be made.

TENSION HEADACHE

It has long been known that sustained contraction of the skeletal muscles about the head and neck is a frequent source of headache. Such headaches occur in association with a wide variety of other sources of head pain including migraine headache, hypertensive headache, and with inflammatory, degenerative, traumatic, and neoplastic diseases about the face, head, neck and cervical vertebrae. The most common cause of muscle spasm headache, however, is tension headache. Tension headache is that type of headache occurring in relation to constant or periodic emotional conflicts concerning which patients are usually partially aware. Reviewing the histories of one thousand patients with tension headache, it was noted that the majority were females (65 per cent). A family history of headache was present in 40 per cent of the patients. Those with daily headaches composed the largest group (30 per cent). The duration and location of the headaches were extremely variable. Prodromata occurred in only 10 per cent of cases.

Cause

Although the cause of tension headache is not known, there is relatively good evidence that such headaches are related to psychologic disturbances. Frequently in patients that I observed the fundamental psychic factors were largely unconscious, although most patients were aware of their anxiety.

Headache may also be brought on by environmental demands of an economic, social or physical nature that are beyond the capacity of the patient's personality. The most frequently observed conflicts in cases of tension headaches were those concerned with hostile and aggressive impulses of an intense and destructive nature.

Mechanism

In tension headaches muscular or vascular mechanisms may act independently or concomitantly. With muscle tension, sustained contraction of the skeletal muscles of the head and neck causes pain or dysesthesia in the neck and scalp. Associated with these muscular spasms may be ischemia, which could be a contributory or primary factor in the induction of pain. It has also been hypothecated that excessive concentration of potassium in muscle, from ischemia or sustained contraction, stimulates the chemoreceptors in the tissues. Another factor leading to the head pain may be a central spread of the excitatory effect of noxious stimulation of the soft tissues of the neck. This spread of pain is carried by the upper cervical nerves and may produce painful sensation in the forehead and face.

Two possibilities have been suggested to explain the mechanism of the pain projection from the cervical spine to the forehead and face. One involves irritation of the descending or spinal root of the trigeminal nerve with transmission of pain to the head and face, and the other pertains to pain which is sympathetic, caused by reflex stimulation.

Diagnostic manifestations of tension headache include:

1. No prodroma.
2. Bilateral nature of the headache (commonly occipital and frontal).
3. Variable in character (burning-pressing-throbbing).
4. Variable in frequency and duration (more constant than migraine).
5. Associated symptoms — various — including anxiety, nausea, and fatigue.

Tension headache must be differentiated from migraine and from headaches associated with intracranial lesions, cervical disc disease, osteoarthritis and spinal cord tumor.

Treatment

1. *Symptomatic.* Symptomatic relief from tension headache is secured by drug therapy. The treatment of tension headache is nonspecific, but the purpose of therapy is to relieve tension and raise the pain threshold. This is best accomplished by use of an analgesic-sedative combination. Such a combination should be one that acts with minimal side effects, allowing the patient to maintain environmental relationships, and does not become a problem because of possible addiction or the development of tolerance.

2. *Prophylactic.* Control of tension headaches can best be accomplished by use of psychotherapy, for this is the only method in which the patient's emotional conflicts can be resolved.

Sedatives, such as the barbiturates, and analgesics afford only temporary relief. Furthermore, the side effects curtail prolonged use. More recently we have used reserpine, chlorpromazine and meprobamate in patients with tension headaches. Improvement has been noted in some cases, but the results are preliminary and not conclusive.

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