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Headache

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Abstract
From a dissertation read before the Society on February 4th, 1966.

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HEADACHE

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The first recorded description of headache comes from the 'Book of Prognoses' which is a series of tablets inscribed by the Physician-Priests of Mesopotamia probably before 2,500 B.C. These writings contain a mixture of clinical observations, which are often very astute, with statements on crude drug therapy, magic and religion.[1] One section on headache has been translated thus:

'when his brow pains a man and he vomits and is sick, his eyes being inflamed, it is the hand of a ghost; then reduce to ashes human bones and bray them; and anoint him with them in cedar oil and he will recover.'

It was serious enough having the 'hand of a ghost' causing these afflictions but worse still

'not only the hand of a ghost but the hatred of a goddess against his life causes a man's right temple to hurt, his right eye to swell and tears to flow.'

While it is clear from the remains of neolithic skulls that trephing was performed at that time, and perhaps more surprising, that patients recovered, the precise indications for trephing are unknown. Perhaps an indirect indication may be considered from the finding that primitive groups in the South Sea Islands were trephing skulls during the last century. Possession by evil spirits was considered the cause of headache, epilepsy and mental disorders; these spirits were released by trephining.

The contribution of Greek medicine to our understanding of headache is the recognition that a particular group of clinical features constitute a distinct entity, the migraine syndrome. No advance in knowledge of the actiology of headache occurred with the Greeks, nor has it occurred until the present era.

Now, in our age of 'scientific enlightenment' we seek a more precise and objective description of the pathophysiological mechanisms than

demon possession, the hands of ghosts, and the hatred of goddesses.

In an article of this length the central mechanisms of pain perception cannot be discussed. Our concern here is with the disturbances which give rise to abnormal patterns of sensory input to the brain, and not with discussion of the fibres involved, the abnormal patterns themselves, and the central interpretation of these changes.

Sources of Headaches

Knowledge of which structures in the head may give rise to pain comes from two main sources. The first of these is the correlation of clinical observations, e.g. an occipital headache, with pathological findings, e.g. an infra-tentorial tumour. The second source is from neuro-surgical operations under local anaesthesia in which records are made of the site of referred pain which is elicited by such crude methods as crushing, stretching, distending, burning and electrically stimulating various structures of the head.[2] There is, however, a great difference between eliciting pain in an operation and demonstrating the mechanisms which operate in the clinically occurring headache. The following results, therefore, are but crude pointers to the sites at which pain may arise. The scalp, as expected, is pain-sensitive while the cranial bones are insensitive. The dura appears to be insensitive to pain-producing stimuli except in the vicinity of arteries, venous sinuses and their tributary veins, and the floor of the anterior and posterior fossae. Pain is particularly easily elicited from the large arteries at the base of the brain. Cranial nerves V, VII, IX and X, all nerves with a sensory component, and also CI, 2 and 3 are pain sensitive. No pain was elicited from the pia and arachnoid mater, the parenchyma of the brain or the linings of the ventricles. As the supra-tentorial dura is innervated by branches from the Trigeminal Nerve, pain arising in this region is usually referred in the distribution of the nerve producing peri-orbital, frontal and temporal headaches. Infra-tentoral disease, on the other hand, is largely referred via Cranial nerves IX and X to the auricular region and via the upper cervical nerves to the occipital region and the upper part of the neck.

The causes of headache have, inevitably, been classified. One simple and useful approach is to consider that headaches may arise in any, or a combination of five general ways;

(1) traction upon intra-cranial structures
(2) intra-cranial inflammation
(3) vascular changes
(4) sustained contraction of scalp and neck muscles
(5) spread of pain from diseases of the eyes, ears, nose and throat.

Traction Upon Intra-Cranial Structures

Headache may occur with expanding intra-cranial lesions whether these be neoplasms, sub-dural or intra-cerebral haematomas, or abscesses. It is almost always a presenting symptom in infra-tentorial lesions presumably because expansion occurs within a confined space in a region which contains a number of pain-sensitive structures. With expansion of the lesion distortion of the normal anatomy occurs, and pain arises from traction on and displacement of nerves and vessels. In supra-tentorial lesions headache occurs as a presenting feature in only about a third of all cases, presumbaly because greater expansion may occur before pressure and traction effects become prominent.

Intra-Cranial Inflammation

The intra-cranial inflammation that occurs in meningitis or sub-arachnoid haemorrhage is associated with severe headaches. The pain probably arises in part from vascular changes presently to be discussed, partly from traction and pressure effects, and perhaps from direct stimulation of nerves by the ill-defined entity 'toxins' and the breakdown products of affected cells.

Diseases of Eyes, Ears, Nose, Throat and Teeth

Diseases of the eyes and orbit, E.N.T. conditions like sinusitis and acute otitis media, as well as dental abscesses may produce pain. In general the pain is at first localised at the site of the lesions, but with progress of the disease process it may radiate in the distribution of the nerve involved.

Headaches of Vascular Origin

The vascular changes which occur in a number of conditions, for example, hypertension, migraine, uraemia, and febrile illnesses appear to be related to the headaches. The subject is perhaps best approached through an experimental model, the headache induced by the intravenous injection of histamine. Within a few seconds of injecting histamine there is a flushing of the skin, hypotension and a rise in C.S.F. pressure. In about 30 seconds the blood and the C.S.F. pressures return to nor-

mal, and it is at this time that the headache begins. It is a bilateral, throbbing headache which usually lasts between 10 and 20 minutes. There is only indirect evidence that the headache is related to intracranial vasodilatation and the evidence is as follows. The oscillations in C.S.F. pressure that are in phase with the arterial pulse are increased during the headache. The headaches are reduced by manoeuvres which reduce the intracranial arterial pressure. The headaches are intensified or diminished by lowering or elevating the C.S.F. pressure respectively.

Wolff has applied these methods to patients with headaches and finds that a number of headaches are altered by these manoeuvres in a similar manner to the histamine induced headaches. This group includes the headaches of uraemia, all the febrile illnesses, post-seizure and post-concussional (in part). As vasodilatation is common to all these states it is commonly stated that dilatation of the vessels, perhaps with stretch of the fine nerve endings in the wall, is the cause of the headache. But it is an inadequate explanation, for it does not recognise that there may be a process which has in common vasodilatation and pain stimulation. Recent work on the headaches of the migraine syndrome suggests that, in migraine, arterial dilatation is only part of the story. And so it may be revealed with further investigation that the headaches due to intracranial vascular changes have mechanisms similar to that in migraine.

The headache in the migraine syndrome is classically a unilateral throbbing headache that may be peri-orbital, frontal, temporal, or occipital. It may last from under half an hour to several hours. But the headache is only part of a syndrome which may be very variable in presentation. In about 15% of patients there may be prodromal symptoms occurring between 20 and 40 minutes before the onset of the headache. These may include a variety of visual changes, such as scintillations, scotomas, or even hemianopia. There may be paraesthesia, ataxia, vertigo, or changes in consciousness or mood.

But what is known of the underlying mechanisms in this condition? When the vessels of the bulbar conjunctiva are directly examined and photographed, arteriolar constriction is found in the prodromal phase. The finding that E.E.G. changes are consistent with focal cerebral ischaemia, and that the prodromata can be reduced or abolished with breathing 10% CO_2 mixtures suggests that arteriolar constriction with areas of cerebral ischaemia may underlie the prodromal phase.

The origin of the headache of migraine in the extra-cranial arteries is suggested by a number of observations. Pain can be reduced or abolished by direct pressure or procainisation of the extra-cranial arteries. Unlike the histamine induced headaches it is unaffected by manoeuvres which alter intra-cranial pressure. During a headache the superficial vessels become tender, painful and surrounded by oedema fluid. Simple measurements with a tambour show increased amplitude of pulsations during the headache. Three changes must be explained during the headache phase; (1) vasodilatation, (2) oedema formation, (3) pain production. What may be implicated as the perpetrator of these changes.

The release of endogenous histamine from a bound form has been considered an unlikely mechanism as the headache differs in its characteristics from a histamine induced headache. Also, the migraine headache is unaffected by anti-histamines in contrast to the reduction in pain that is produced in the rarer, rather bizarre condition 'histaminic cephalgia' or 'cluster headaches'. Recently, however, Schayer[3] using radioactive tracer techniques, has shown the existence of an 'induced' form of histamine, that is unaffected by anti-histamines, and has a longer time course of action than 'bound' histamine. Its precise role is as yet speculative. But histamine cannot yet be dismissed as having no role in the headaches of migraine.

May 5 H.T. or a kinin be a cause of the vascular changes? To investigate this, small quantities of tissue fluid have been aspirated from the vicinity of the temporal arteries during and immediately after the migraine headaches and also in headache-free intervals. These were then compared with the aspirates from normal subjects by a number of pharmacological assay methods.

Chapman[1] claims to have found in the tissue fluid a polypeptide and also an enzyme that, on incubation with plasma, is capable of producing increased quantities of the polypeptide. This polypeptide is similar but not identical to bradykinin, and clearly differs from 5 H.T. The levels during the headache period were on average about 8 times that of normal subjects, and it was stated that the level correlated very well with clinically estimated severity of the headache. Here, of course, is an illustration of one particular difficulty in work on pain — that of estimating the severity of pain and comparing the severity of pain in one person with that

in another. This polypeptide has been labelled 'neurokinin'. While it does fulfil our three criteria, those of vasodilatation, increased permeability with oedema formation, and pain production, this work is rather tentative. It may be that the improved methods of separation and characterisation of the kinins involved will throw more light on the subject. It may become evident that a multi-factorial mechanism is responsible for the headache. It must also be pointed out that no progress has been made on the questions of what initiates the whole process, why the headache should be unilateral, and why the changes should occur only in the external carotid vessels? The autonomic changes which may occur in association with the headache are varied; there may be lacrimation, bradycardia, sweating, nasal congestion, constipation (or even diarrhoea). It may eventually be shown that the migraine syndrome takes origin in a disturbance of autonomic function.

The headaches of hypertension, on the same evidence as in migraine, appear to originate in changes in the extra-cranial arteries. The headaches do not, however, relate directly to the level of the blood pressure except during acute attacks. The fact that the arterial pressure is elevated throughout the body, but only the extra-cranial vessels become painful suggests that there may be some intrinsic difference in the extra-cranial arteries that renders them liable to develop pain. Little work has been done on the headache in hypertension so that it is only an interesting speculation that the mechanisms may be similar to those of migraine.

Muscle-tension Headache

The other common chronic headache, a cause of distress to thousands of patients, is one which often arises in relation to stress or anxiety and is called a 'tension' or 'muscle-tension' headache. Its characteristics are that it is non-pulsatile, fairly constant in intensity, being a dull ache rather than an acute pain. Patients describe it variously as a 'tight band' or cap or as an oppressive pain on the top of the head. Often it is bilateral in the occipital region, extending into the neck, but it may vary and be either unilateral or bilateral, and may occur in the frontal or parietal areas. It may occur primarily in a neurotic type of reaction or in anxiety producing situations, but it may arise secondary to pain elsewhere, e.g. migraine, or secondary to degenerative changes in the cervical spine.

The term 'muscle-tension' headache has evolved because one feature of the headache is a sustained contraction of the occipito-frontalis, temporalis or the muscles of the neck. E.M.G. recordings from these muscles showed increased activity during the headache periods which was approximately related to the 'clinical anxiety level' (again a very subjective and unreliable estimation). There is, however, another factor besides increased muscular activity, and this is the observation that arteriolar constriction occurs in the bulbar conjunctive during the headaches. As the headaches are intensified by vasoconstrictors and relieved by vasodilator drugs it is reasonable to infer that constriction may be occurring in the vessels of the active muscles and that this contributes to the headache. Presumably, therefore, headache arises from a combination of ischaemia due to vasoconstriction and increased metabolism of sustained muscular contraction.

Conclusion

Headaches are often trivial and transitory and as such tend to be ignored clinically. But they are important in two ways. Firstly, the chronic, recurring headaches cause considerable misery in the community. Secondly, headaches may be of great clinical significance, for example in hypertension and in intra-cranial tumours. The explanation of the causes of these headaches have varied through the ages, and even now ideas change as information increases. Some current ideas have been outlined in this article. As knowledge increases in the future so should the treatment of the underlying causes improve.

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