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EXPERIMENTAL OBSERVATIONS ON HEADACHE*

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There are few symptoms more common or more obscure than headache. In most instances we do not know how, or from what structure, the pain arises. Partly perhaps as a consequence the diagnostic value of headache is less than in many other varieties of pain and its therapeutics is disappointing. The earlier work described the occurrence of headache in certain maladies and speculated as to its origin, but in elucidating mechanism it was relatively unproductive. In the last few years attempts have been made to approach the problem experimentally, and it is this work which I propose to discuss here.

The term "headache" is used to indicate pain felt in the region of the cranial vault. The pain may arise in several ways. Each form of headache must be considered separately, and in this account I shall not be able to discuss all. I shall do no more than refer to one of the commonest—namely, headache arising from affections of the muscles and other structures of the neck. Many of the observations described here are fragmentary, and are put forward chiefly in the hope of encouraging further work on a subject which I now find little time to pursue.

Sensibility of the Cranial Cavity

We might expect the disturbances responsible for headache to arise in the scalp, the skull, or within the cranial cavity, and it would at least give our inquiry some direction if we knew what structures in these parts were capable of giving rise to pain when suitably stimulated. The sensibility of the structures comprising the scalp and the skull would seem to be that of similar structures elsewhere in the body, and needs no special description: skin, muscle, fascia, artery, and periosteum are all painful when injured. The sensibility of the cranial content has been investigated by several neurosurgeons operating, as Lennander (1906) did, without the use of anaesthesia, or more usually with local anaesthetization of the scalp. There is good general agreement as to the findings (Fay, 1931), which have been well described recently by Penfield (1935). The brain with its enveloping pia arachnoid is completely insensitive to mechanical injury, except in the Sylvian fissure, where pain is elicited from a stimulus near the vessels. Over most of its extent the dura is also insensitive, but pain is elicited by a variety of stimuli applied along the course of the meningeal arteries and

their branches and in the neighbourhood of the large venous sinuses. Pain elicited by injuring the dura near an artery is fairly well localized to the point of stimulation, whereas that from the dural sinuses tends to be felt at a distance and to be more diffuse. Traction on the falx cerebri or on the tentorium cerebelli is also painful, presumably by distortion of the venous sinuses where these expansions are attached to the parietal dura mater. In addition the dura mater at the base of the brain and the adjacent circle of Willis with its main branches are said to be sensitive, but information concerning this region is rather meagre.

To suppose that headache of intracranial origin arises chiefly from disturbances near the dural arteries or venous sinuses would be consistent with these observations on the location of pain nerves. But we cannot on this information exclude its origin elsewhere. The heart, exposed under local anaesthetization of the chest wall, is insensitive to mechanical injury, yet there is little doubt that the pain of angina pectoris and of coronary thrombosis arises within this organ. It is clear that the mechanism of each form of headache must be judged from the circumstances of its origin and its behaviour to interference.

Histamine Headache

When, after conversation with Mr. Wilfred Trotter, I first became interested in headache, the chief difficulty was to find an approach to the problem: in most pathological headaches the precise nature of the circumstances precipitating the attacks of pain is unascertained, and the skull provides an effective screen to the events taking place within.

At about that time, however, intravenous injections of histamine were being used in the laboratory to measure the circulation time, and I noticed that the injections were followed by severe headache in myself and other subjects. This substance seemed to provide an unusual opportunity of working out the mechanism of one variety of headache, since with it headache could be produced at will and under any set of circumstances of the observer's choice. In these observations (Pickering, 1933-4) I was joined later by Dr. Werner Hess. We were able to show that the histamine headache does not arise from the scalp, since it can still be produced by intravenous injection of histamine when the blood supply to the scalp has been arrested by an Esmarch bandage. That the headache arises from the meninges is suggested by its increase when the head is quickly shaken from side to side; for in such movement the brain tends to lag behind the calvarium and thus the meninges are temporarily strained.

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Further, the pain seems to arise from a structure supplied by the fifth nerve, since the histamine headache is usually confined to the normally innervated side in patients with unilateral trigeminal section. Current anatomical knowledge suggests that the dura mater is the chief meningeal structure innervated by the fifth nerve.

Histamine acts chiefly on smooth muscle which, in the cranial cavity, is confined to the blood vessels. It thus seemed probable at the outset that the headache might be due in some way to a vascular change. In response to intravenous injection of 0.1 mg. histamine acid phosphate several changes, which may be ascribed to vasodilatation, begin simultaneously in about fifteen seconds; they are flushing of the face, a fall in arterial pressure, and a rise in cerebrospinal fluid pressure due to cerebral vasodilatation. Headache begins later at about the first minute; it is maximal at about the second, and disappears about the eighth minute (Fig. 1). The head-

FIG. 1.—Showing the cerebrospinal fluid pressure, the systolic blood pressure, the facial flush, and the headache before and after injecting intravenously 0.1 mg. histamine acid phosphate at 0 min.

ache begins after the flush is maximal and increases while the flush is fading; it begins after the arterial pressure has recovered from its fall; it begins when the cerebrospinal fluid pressure is falling from its highest point, and it is maximal when this pressure has returned to its original level. This relation between the vascular events and headache is not fortuitous, for if histamine is infused intravenously for several minutes at a constant rate of 0.1 to 0.17 mg. per minute, then the time at which headache elects to appear is not during the infusion but a minute or more after this is stopped. Here again the onset of headache coincides with the recovery of the arterial pressure from its fall. The delay in the onset of headache effected by continuing the administration of histamine shows that the headache is not due to chemical stimulation of pain nerve endings, by histamine or some product of its breakdown, and suggests that something occurs during the stage of intense vasodilatation preventing the appearance of headache. This idea is confirmed in the following way. If, when the histamine headache is at its height, a second injection of histamine is given, then the headache is abolished at the time when the face flushes and the arterial pressure falls in response to the second injection; when the arterial pressure rises again the headache returns. This effect of a second injection is due to vasodilatation, because the same transitory relief is achieved by another substance having rather similar dilator properties—namely, amyl nitrite.

Two changes occur constantly during the first phase of the action of histamine which, by altering conditions in the cranial cavity, might conceivably inhibit the development of headache; they are a rise in cerebrospinal fluid pressure and a fall in blood pressure. The effect of these changes was investigated separately. The cerebrospinal fluid pressure in the cranial cavity may be raised by injecting normal saline into the subarachnoid space at lumbar puncture; or by raising cerebral venous pressure, as in compression of the jugular veins in the neck; or by lowering the head, which raises cerebral arterial and venous pressures also. All these

measures raising intracranial pressure relieve the histamine headache, but the effect is most pronounced with subarachnoid saline injection and least noticeable with lowering of the head. That the intensity of the pain also varies with intra-arterial pressure is shown by its waxing and waning with each pulse beat, and by the pain being temporarily relieved by Müller's experiment, in which forced inspiration with closed glottis results in a transient fall in arterial pressure. The most striking demonstration of the relation between intra-arterial pressure and headache is provided by an experiment which I have made on several occasions since our original observations were published (Pickering, 1933-4). If, when the headache is maximal, one carotid artery is obliterated by digital pressure low in the neck, then the headache promptly disappears on that side of the head only; when the compression is removed the pain returns with a temporarily increased intensity.

The relief of headache by a fall in pressure inside the intracranial arteries or by a rise of pressure outside them admits of one interpretation—namely, that the pain is due to stretching of some sensitive structure in the walls of the intracranial arteries or closely adjacent to them. We supposed that such stretching might result from relaxation of the arterial wall or from accumulation of perivascular fluid, but we had no evidence on which to differentiate between these possibilities. Evidence was provided later by Clark, Hough, and Wolff (1935), who confirmed our observations, and who showed that the waxing and waning of headache ran parallel to the increase and decrease in the pulsations of the cerebrospinal fluid recorded optically from the lumbar sac. They regard these pulsations as reflecting the diameter of the intracranial arteries, and the results suggest that the headache is due to dilatation of these vessels.

In the light of such experiments the course of events following histamine injection would seem to be as follows. The first phase is one in which dilatation of the small vessels predominates and in which the intracranial arteries, though probably relaxed, are prevented from dilating by the fall of arterial pressure inside and by the rise of intracranial pressure outside. During the second phase the arterial and intracranial pressures return to normal as dilatation of the small vessels subsides, and coincidentally the larger intracranial arteries, whose walls are still relaxed, expand; it is this expansion which seems to give rise to headache.* Finally relaxation of the intracranial arteries passes off and headache subsides.

Observations such as these are of interest in demonstrating with some degree of probability the mechanism of one variety of headache and in showing that it is possible to attack the problem of headache by experimental means. We may now consider two forms of headache which resemble the histamine headache in certain particulars.

Lumbar Puncture Headache

A very characteristic headache occasionally occurs a few hours to a few days after lumbar puncture, and particularly, I think, when the patient has been allowed up and when several attempts have been made to enter the spinal theca. The headache is usually throbbing, and is frequently felt diffusely over the cranial vault; it may be accompanied by vomiting and by neck rigidity and other signs of meningeal irritation. The most characteristic feature of the headache is its very conspicuous increase when the patient sits up. Jacobaeus and

* In our previous article Hess and I concluded that the vessels concerned were probably the arteries of the dura mater, because of this being the only meningeal structure known to be innervated by the fifth nerve. Recently Northfield (1938) has produced experimental evidence for the view that the histamine headache arises from branches of the internal carotid artery and not from the arteries of the dura, for he has found that histamine gives rise to headache when injected into the internal carotid artery, but gives none when injected into the external carotid artery, of which the middle meningeal is a branch.

Frumerie (1923) showed that the cerebrospinal fluid pressure recorded in the horizontal position is extremely low during post-puncture headache, and attributed this to leakage of cerebrospinal fluid into the epidural space from the hole made in the spinal membrane by the first lumbar puncture. This leakage had already been suggested by McRobert (1918), and was demonstrated by Ingvar (1923) in a patient with tuberculous meningitis; it was subsequently demonstrated by Heldt (1929). Jacobaeus and Frumerie also showed that the low cerebrospinal fluid pressure was the primary disturbance in post-puncture headache, because injection of normal saline into the subarachnoid space restored the pressure to normal and abolished the headache. I have had the opportunity of confirming Jacobaeus and Frumerie's observations in the following case.

Case 1.—A young man was unconscious for three to four days following a motor accident in 1927. In 1933 he began to complain of frequent attacks of right fronto-parietal headache. He presented no signs of central nervous disease. On April 2, 1934, lumbar puncture was performed, the spinal pressure being 105 mm. H_2O . About 5 c.cm. was removed. The same evening he began to experience a severe continuous bilateral retro-orbital headache quite different from that previously experienced; and he later vomited twice. On April 4, when I saw him, the headache continued and he had slight neck rigidity. The headache was greatly increased by shaking the head; it was increased by digital compression of both jugular veins in the neck; sitting up greatly increased the intensity, the effect beginning in about five seconds and not increasing after thirty seconds. Lying down decreased the pain, the effect beginning in twenty seconds and being fully developed in forty-five seconds. Lumbar puncture was performed with the patient horizontal. Fluid was only obtained by attaching an aspirating syringe to the lumbar puncture needle, and the fluid failed to rise in the manometer tube. Injecting 12 c.cm. of warm normal saline into the spinal theca raised the pressure to 30 mm., and the headache became easier; a further 13 c.cm. raised the pressure to 45 mm. H_2O , and the patient stated that the headache had gone. He now sat up and the pain remained absent.

I have made less extensive observations on post-puncture headache in four other cases. I observed that the headache throbbed with the pulse (three instances), was increased by jugular compression (three instances), and was increased by shaking the head (two instances). In one case carotid artery compression did not affect the intensity of headache.

From the available facts it is clear that the pain is due to a mechanical disturbance occasioned by a diminution in the cerebrospinal fluid pressure in the cranial subarachnoid space, since headache is associated with low cerebrospinal fluid pressure and is abolished when the pressure is restored to normal, and since the headache is so conspicuously increased by sitting up. It is conceivable that the pain arises from widening of the intracranial arteries owing to the fall in the pressure outside these vessels. It is also possible that the pain may arise from distortion of the dura in the neighbourhood of the large venous sinuses, as by a backward and downward displacement of the brain consequent on the emptying of the subarachnoid space. If the first hypothesis were correct we should expect the lumbar puncture headache to behave to experimental interference in the same way as the histamine headache. The two resemble each other in their throbbing character, in their relief by subarachnoid injection of saline, and in their exacerbation by shaking the head. They differ in the lumbar puncture headache being more conspicuously affected by posture and being increased by jugular compression, and perhaps also in the effects of carotid compression. However, Hess and I observed that if 15 c.cm. or more cerebrospinal fluid is removed by

lumbar puncture before the injection of histamine, the subsequent headache is increased by jugular compression. I am uncertain as to the explanation of this peculiar finding, but it suggests that the differences in behaviour of the two headaches may reflect a difference in the amount of cerebrospinal fluid in the subarachnoid space rather than a difference in the actual source of the pain. The behaviour of the lumbar puncture headache is, however, also quite consistent with the view that it arises from stretching of the dural venous sinuses by a hyperaemic and displaced brain, and we must leave a final decision until a more exhaustive study, including the effect of carotid artery compression, has been made.

Pyrexial Headache

Headache occurs commonly in association with pyrexia in many acute infections. I have occasionally observed the behaviour of such headaches, and have found them to behave very similarly to the histamine headache, though I have not tried carotid compression. One case of pneumonia may be quoted briefly.

Case 2.—A woman aged 53 became hot and feverish on August 28, 1932. On the next day she had a severe throbbing generalized headache which interfered with sleep. She was admitted to hospital on September 1, and on September 4 the following observations were made. Her temperature was 103° , her pulse 110; physical examination showed signs of consolidation over the upper lobe of the right lung. Headache was still present, and was relieved by jugular compression and increased by shaking the head. The pain was so severe that lumbar puncture was done. The cerebrospinal fluid pressure was 230 mm. H_2O , the fluid being normal. Injection of 20 c.cm. of warm normal saline into the subarachnoid space raised the pressure above 400 mm. and relieved the headache. Withdrawal of fluid reduced the pressure to 110 mm. and increased the pain. On September 5 the patient's temperature fell to normal by crisis. On September 20 the temperature rose again, and a dull ache without throb was experienced, and was said by the patient to be quite different from that previously felt. She was tender over the right frontal sinus, which was opaque to transillumination, as was the right maxillary antrum a few days later. On October 2 injection of histamine produced a headache which the patient declared identical with that associated with her first pyrexia.

Their similarity in behaviour to the histamine headache suggests that these pyrexial headaches are likewise due to stretching of the sensitive structures around the intracranial arteries, presumably arising from relaxation of these vessels; as yet there is no indication as to how such relaxation might arise.

Migraine

In the classical migraine attack there are three events—a visual or other aura being succeeded by headache and vomiting; but sometimes one or more of these components are absent. The headache is typically unilateral and throbbing, and is increased by movement, particularly of the head. In the two latter features it resembles the histamine headache, as it does in its relief by compression of the common carotid artery. This simple and significant observation was first made nearly 150 years ago by Parry (1792) and has since been repeatedly confirmed, but it has not received the attention it deserves, and I shall therefore quote from some of the writings describing it.

In 1867 Möllendorf wrote: "If the common carotid artery be compressed on the painful side at the level of the thyroid cartilage during the hemicranial paroxysm, so that the pulse in the temporal artery begins to fail, the headache vanishes as if by magic. . . . Unfortunately, however, with the intermission of the compression, with the first full pulse wave, the

pain begins afresh, and indeed the first pulsation will be felt to be much more painful. . . . Conversely, compression of the carotid artery of the other side enhances the pain." Wilks (1878) wrote: "Thus a pressure on the carotid in the neck will produce a suspension of the throbbing and the pain, but the effect is only for a time, as the blood soon finds its way to the head by other channels." Lauder Brunton (1908), describing observations on attacks in himself, stated: "That the pain in migraine is to a great extent due to the stretching action exerted on the contracted temporal artery by the blood which tries to pour into it from the dilated carotid is shown by the fact that if pressure be exerted upon the carotid so as to stop the pulse in the temporal artery the headache will frequently disappear instantly . . . the moment the finger is removed the pain comes back with a rush of peculiar intensity." Similar observations were described in 1905 by Hare, who found in addition that compressing the occipital, temporal, or angular arteries would relieve headache over the distribution of these vessels.

These observations, and particularly those of Hare, would seem to suggest that the headache in migraine arises from stretching the coats of the arteries of the scalp or of immediately adjacent tissues; but of the workers quoted only Lauder Brunton actually drew this inference. The idea that a vascular disturbance is the basis of headache and possibly of the other phenomena of migraine, though adopted by all these workers, has not won acceptance because of the variability in the colour and temperature of the face and in the state of the arteries of the scalp as determined by the eye and by the finger. Thus Du Bois-Reymond (1860) considered that in his own case the temporal artery on the side of the pain was contracted and the face pale, while Wilks says: "The carotid on one side with its branches throbs inordinately, is apparently very full. This I knew when quite a boy, for when leaning my head on my hand I distinctly felt the increased size of the throbbing temporal artery on the side of the pain, which would sometimes be on the right and sometimes on the left side."

In recent years the important fact has been added that the headache of migraine is abolished by intravenous, or less certainly intramuscular, injection of ergotamine tartrate. Graham and Wolff (1938) have made use of this to study the relation between the intensity of the headache and the amplitude of pulsations of the temporal artery and of the cerebrospinal fluid. They have found that after the intravenous injection of 0.37 to 0.5 mg. of ergotamine tartrate the decline of headache runs parallel to a decrease in the temporal pulse, but that the cerebrospinal fluid pulse is not consistently altered. They have also found that digital compression of the common carotid, temporal, or occipital arteries relieves the pain in the distribution of these vessels. Finally they have shown that pain arises in the distribution of the vessel when the temporal artery is exposed under local anaesthesia and distended with saline injected into the proximal end, the distal end being obliterated with the finger. They conclude that migraine headache is due chiefly to tension set up around the dilated arteries of the scalp, but they believe that dilatation of intracranial arteries may play a part, because in certain instances greater relief is afforded by compression of the common carotid than by simultaneous compression of the temporal and occipital arteries on the same side. In this connexion it would be interesting to know the effects on the pain of deliberate alteration of cerebrospinal fluid pressure by injecting saline into the subarachnoid space. Lennox (1934) and I have both found that injection of ergotamine tartrate does not diminish the intensity of headache from subsequent injection of histamine.

Although these observations suggest that the diameter of the temporal and other arteries of the scalp is an important factor in determining the migraine headache, we do not know whether the pain is due to simple arterial dilatation or whether there is another factor such as sensitization of the pain nerve endings by a chemical agent. The older writings have some doubt as to whether the temporal arteries are always dilated in migraine, and it has not yet been shown whether, in those suffering from the malady, vasodilatation of the scalp—produced, for example, by a hot bath—is necessarily accompanied by pain. We may ask, too, whether the arteries of the scalp are peculiar among the superficial arteries of the body in giving rise to pain when dilated, for we do not yet know of any comparable condition elsewhere; and we may ask what is the agent which dilates the extracranial arteries, and what is its relation to the preceding and presumably intracranial events responsible for the aura.

Raised Intracranial Pressure

The most widely held hypothesis of the mechanism of intracranial headache ascribes the pain to stretching of the parietal dura by raised intracranial pressure. The appeal in this idea lies in its accounting in a simple manner for headache being the commonest and often the earliest symptom of a number of diseases in which the intracranial pressure becomes raised. I have never been clear as to how the parietal dura can be supposed to become stretched, for it is well supported by the rigid cranium. The hypothesis does not account for the histamine headache, lumbar puncture headache, and pyrexial headache, since in these conditions, as we have seen, raising the intracranial pressure by subarachnoid injection of saline actually relieves or abolishes the pain. Nevertheless, the hypothesis must be judged on its merits.

CEREBRAL TUMOUR

Cerebral tumour is the classical example of a disease raising intracranial pressure, and, in support of the theory now being considered, provides two facts which have been widely quoted—namely, that headache is frequently abolished by decompression, and that it can often be relieved by intravenous or rectal injection of hypertonic solutions. Such few facts as I have had the opportunity of eliciting in cerebral tumour are not, however, in accord with the idea that the pain is the direct result of raised intracranial pressure. Thus I have observed two cases of temporo-sphenoidal tumour and one of cerebellar tumour, all verified at operation or necropsy, in which headache was produced or an existing headache accentuated by the removal of cerebrospinal fluid by lumbar puncture. Similar observations have been described by Ingvar. These cases are briefly as follows:

Case 3.—A steeplejack aged 30 was seen in June, 1932, complaining for twelve weeks of attacks of tingling, beginning in the right hand and involving the right half of the body; more recently his right arm and leg became weak and his speech defective. For two weeks he had experienced frequent attacks of diffuse headache lasting a few minutes to an hour and occasionally accompanied by vomiting. The headache was sometimes throbbing and sometimes not, and came and went without his knowing why. His mind was clear, but speech was halting, and he presented a right hemi-anaesthesia and right hemiparesis. There was no papilloedema. On June 7 lumbar puncture was done while he was complaining of a throbbing headache over the calvarium and worst over the forehead. The cerebrospinal fluid pressure lay between 70 and 110 mm. H₂O. Jugular compression raised the pressure to 300 mm. H₂O and relieved the headache, which returned to its original severity on release of the compression. Six c.cm. of normal cerebrospinal fluid was removed, the

pressure falling to 10 mm. H_2O ; during the removal of the fluid the headache increased in severity. On June 30 a left temporal decompression by Mr. Taylor revealed an inoperable tumour deeply situated in the temporal lobe. The patient subsequently developed a hernia cerebri, and died on April 2, 1933.

Case 4.—A schoolmaster aged 28 had in January, 1934, suffered for three and a half years from headache and convulsive fits heralded by aphasia, and for four months from increasing weakness of his right arm and leg. Headache was bilateral and frontal, and occurred in paroxysms lasting about fifteen minutes. Nothing was known to provoke the pain. The patient presented bilateral papilloedema, halting speech, and a right hemiparesis. On January 31 lumbar puncture showed a spinal pressure of 320 mm. H_2O . Removal of 15 c.cm. of normal fluid immediately produced a headache of the usual kind, lasting three hours. On February 5 he was examined while complaining of headache. Compression of the jugulars relieved the pain. Lumbar puncture showed a fluid under considerable tension (lumbar pressure after removing 5 c.cm. was 250 mm. H_2O). Removal of cerebrospinal fluid increased the headache. On February 6 he became comatose, and died the next day during an operation for decompression. At necropsy a glioma about the size of an orange was found in the left temporal lobe just anterior to the basal ganglia.

Case 5.—This case was that of a boy aged 10, suffering from a glioma of the right lobe of his cerebellum, who presented gross papilloedema, and who showed throughout much drowsiness and other signs of cerebral compression. Removal of cerebrospinal fluid by lumbar puncture once precipitated and once increased his headache. Between the two lumbar punctures cerebrospinal fluid was thrice removed by ventricular puncture without any headache resulting.

In the first two of these cases the relief of headache when intracranial pressure was raised by jugular compression, and the onset or increase of headache when cerebrospinal fluid was removed and its pressure lessened, clearly show that the pain cannot have been due to simple rise in intracranial tension. The observations would be consistent with the headache of cerebral tumour being of similar origin to that following histamine, but the points of similarity demonstrated are too few to establish identity. Another mechanism which we might envisage as being brought into play particularly in intracranial tumour would be some change in the spatial relation of brain and dura mater, with consequent tension on the venous channels uniting them. Clearly we must await further evidence.*

MENINGITIS

Meningitis is another condition in which headache and raised intracranial pressure are associated. Here the headache is much more stable than in cerebral tumour, and it is a simple matter to investigate the relation between cerebrospinal pressure and headache.

A typical observation made in a woman of 48 on the ninth day of acute meningococcal meningitis is summarized in Fig. 2. At the beginning of lumbar puncture the patient complained of moderately severe headache, mainly occipital.

* Since this lecture was delivered Northfield (1938) has published a valuable account of the behaviour of headache in intracranial tumour to experimental interference, and reference should be made to his paper for a full discussion of the subject. He has demonstrated that the occurrence of headache in tumour is not related to the height of the cerebrospinal fluid pressure, and that headache may often be provoked by suddenly raising or by suddenly lowering intracranial pressure. In many cases the headache produced by histamine accurately reproduces headaches experienced spontaneously in the course of the disease, and he concludes that the two are identical in their source. As already mentioned, he has provided evidence that histamine headache arises from arteries derived from the internal carotid rather than those of the dura, and supposes that headache in intracranial tumour arises from changes in tension in the walls of the cerebral arteries.

Injection of 18 c.cm. of normal saline into the subarachnoid space raised the cerebrospinal fluid pressure to 700 mm. H_2O and greatly increased the severity of the pain; removing the injected quantity of fluid restored the headache to its original severity. Removal of a further 27 c.cm. of fluid reduced the pressure to 15 mm. H_2O and again greatly increased the headache. Injection of 20 c.cm. of anti-meningococcal serum raised the pressure to 65 mm. H_2O and restored the headache to its original severity. Observations similar to this were made repeatedly on this patient and in two other cases of meningococcal meningitis and one case of tuberculous meningitis. It will be noted that headache is increased when cerebrospinal fluid is raised or lowered sufficiently, but that it still continues at intermediate pressures. In cases where the headache is less severe it may be present only at the lower and upper pressures.

These observations offer a simple explanation for headache being usually relieved but sometimes enhanced by lumbar puncture in meningitis. The facts can scarcely be reconciled with the view that headache is due simply to raised intracranial pressure. The afferent impulses responsible for the pain evidently may arise at all levels

FIG. 2.—Cerebrospinal fluid pressure and headache in a case of meningococcal meningitis. (Explanation of procedure in text.)

of cerebrospinal fluid pressure, but are increased when this is very high or very low. In this condition stimulation of the pain nerve endings may be due to a chemical substance resulting from the inflammatory process. On the analogy of the experiments of Lewis and Hess (1933-4) on the skin we might imagine that such a disturbance might also sensitize the pain nerve endings in the meninges to a mechanical disturbance otherwise inadequate. The throbbing character of the pain, its susceptibility to variations in intracranial pressure, and its increase by shaking the head and jugular compression, which I have found usual in such patients, would all be compatible with some such explanation.

Traumatic Headache

This form of headache has also been supposed, without evidence, to be due to raised intracranial pressure, but Penfield (1927; Penfield and Norcross, 1936) has explained it more satisfactorily in another way. The complaints of headache and giddiness usually begin several weeks after a blow on the head. The pain is maximal near the site

of the injury and spreads to a varying extent; it is commonly persistent, but may be increased by acts such as lifting heavy weights and blowing the nose. In such patients Penfield found that when he injected air into the cranial subarachnoid space by lumbar puncture the old headache usually failed to return when the pain due to air insufflation had subsided. In some patients the symptoms were unrelieved by this treatment, and in these he observed abnormalities in the cranial subdural space, whither the air passes from the subarachnoid space in the course of a few days. In some of these patients the dura was exposed by trephining near the site of the injury, the scalp alone being anaesthetized. A blunt probe passed into the subdural space encountered adhesions and, when these were near a meningeal artery, elicited pain precisely similar to that of which the patient complained. Penfield therefore supposes that the headache following head injury is due to tension on adhesions which develop between the dura and arachnoid near the site of the injury. The development of tension in the adhesions is explained as follows. When the brain is injured it swells either from haemorrhage or oedema near the site of injury and moves a little in relation to the skull; in this position the adhesions form. The brain eventually returns to its original position as the effects of injury subside and the adhesions come under strain.

Conclusions

This brief survey of the mechanism of headache will have served its purpose if it has indicated that the problem is one which will repay fuller investigation by experimental methods. Of measures which influence headache, arterial compression and the addition or subtraction of fluid to or from the subarachnoid space may be stressed because of their simplicity in execution and interpretation. Except in the case of the histamine and post-traumatic headache, the facts available are too few and too poorly established to enable us to draw any conclusions. But from the little knowledge we have it would seem probable that most headaches of intracranial origin are due to tension; and that the regions where tension may produce pain are around the intracranial arteries, as may occur when these vessels dilate—possibly also around the venous sinuses, as might occur from displacement of the brain or parts of it. Whether these are the only sources of headache of intracranial origin may be left to the future for decision.

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PELVIC DISPROPORTION

TREATMENT*

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It is common knowledge that in proportion as diagnosis becomes more exact does choice of treatment become more obvious and clear-cut: a striking example of this fact is furnished by pelvic disproportion. Obstetrics, as we have seen, emerged from the darkness of the Middle Ages with the advent of Ambroise Paré and his advocacy of podalic version in the sixteenth century. There then followed the introduction of forceps in the seventeenth century; induction of labour and symphysiotomy in the eighteenth century; and Caesarean section (modern operation) late in the nineteenth century. Let me review briefly these operations and the place they have occupied and occupy to-day in the treatment of contracted pelvis.

Version

The introduction of podalic version was a tremendous step forward: with the child's legs brought down and traction made upon them, the trunk and after-coming head could be dragged through the deformed pelvis, except in cases of extreme disproportion. And so version held its place, and the premier place, far into the nineteenth century. We find, for example, the great Simpson (Sir J. Y.) advocating version in preference to forceps in 1847. He claimed that version was the better procedure because (a) the head came through the contracted brim more easily base first than vertex first, as the former was the narrower part of the wedge; (b) there was a greater overlap of the parietal bones with an after-coming than a fore-coming head. Version, however, gradually came to be abandoned because of the high foetal mortality associated with the operation, even in cases in which there existed only a minor degree of disproportion.

Forceps

You recollect the three phases in the evolution of the obstetric forceps. First, the straight forceps with the cephalic curve invented by Chamberlen; secondly, the long and double-curved (cephalic and pelvic) forceps invented by Levret; thirdly, the axis-traction forceps invented by Tarnier in the seventies of last century and modified later by A. R. Simpson, Milne Murray, Cullingworth, and Bonney. With each of these phases in its development the forceps became more powerful as a tractor for pulling the head through the pelvic brim.

Fortunately, in most schools, both at home and abroad, the dangers of the operation were early appreciated and the era of extreme force with axis-traction forceps, and the patient in the Walcher position, was short-lived. I witnessed the birth and demise of this most dangerous operation. While dragging the foetus through the pelvic brim by axis-traction forceps is tabooed to-day, I am still of the opinion that, in special circumstances, the operation may be justified provided the operator has had considerable experience in the practice of operative obstetrics. Suppose, for example, a patient were admitted to my department advanced in labour with the child's head arrested and fixed at the pelvic brim, several exam-

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