in some of these cases only then may it become necessary for the surgeon to employ skin or muscle grafts, or some plastic form of operation on the meatus.

Conclusion

I have attempted in this paper to give a résumé of our present knowledge of the treatment of chronic middle-ear suppuration. It will be seen that we cannot claim to have made any great progress during the past decade, particularly as regards conservative measures. What, then, of the future? Upon what lines can we profitably advance, either in our medical or surgical methods? In prophylaxis there must always remain considerable scope for improvement, and much work is being done in this respect in the early observation and treatment of school children. The employment of more effective antiseptic and bactericidal solutions and powders demands further considerable

research into their properties in respect to their effect upon bacteria as well as on the living tissue cell. The same applies to ionization and the various other electrical methods.

As regards surgery we can surely devise a better technique in dealing with the problem. If, as I firmly believe, the maintenance of useful hearing depends in a large number of these cases upon the continued mobility of the oval and round windows, I can foresee our efforts in the future being directed towards this end, and, if fixation has occurred, even to the repair of this mechanical fault, originating surgical methods that will restore the function of the liquid within the labyrinth, thus allowing it to transmit once again the necessary correct pressure variations. Perhaps our ideal will be reached when we can graft on a new tympanic membrane, and fashion a new mobile window to replace the one that has become fixed by some adhesive or sclerotic process.

THE HYPERTENSIVE CEREBRAL ATTACK

BY

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Until recently cerebral symptoms, such as convulsions and coma, occurring in a patient who showed raised blood pressure and albuminuria were attributed to uraemia. Volhard,⁷ however, pointed out that such cerebral symptoms could occur in the absence of renal insufficiency, and he used the term "pseudo-uraemia" to distinguish this condition from true uraemia. He divided pseudo-uraemia into acute and chronic forms. In the acute form there is raised intracranial pressure, and, clinically, headache, vomiting, and slowing of the pulse. These symptoms are produced by cerebral oedema, the result of an acute disturbance of the circulation in the brain. In chronic pseudo-uraemia the principal factor in the production of the cerebral symptoms is angiospasm. He believes that transient focal symptoms—such as weakness in a limb, aphasia, disturbance of vision—are caused by spasm of the corresponding cerebral vessels. In both forms a raised blood pressure is the determining factor. Oppenheimer and Fishberg⁴ emphasized the fact that cerebral symptoms in hypertension are generally preceded by an additional rise in blood pressure above the previous level. They give the name "hypertensive encephalopathy" to the cerebral symptoms occurring in hypertensive states. They believed that these symptoms are caused by vasoconstriction of the cerebral vessels.

It was previously held by the majority of physiologists that there was no nervous control of the cerebral blood vessels. However, the brilliant work of Forbes and Wolff¹ and their collaborators has definitely established the existence of a nervous control of the pial vessels in the cat. A summary of their experiments must suffice.

Stimulation of the cervical sympathetic in the neck by a faradic current was followed by constriction of the pial arteries. Stimulation of the vagus resulted in a dilatation of the pial arteries. They also found that the calibre of the pial vessels could be altered by changes in the chemical composition of the blood. Vaso-constriction resulted from: (a) intravenous injections of hypertonic salines; (b) a decrease in the carbon dioxide content, or an increase in the oxygen content of the blood; and (c) intravenous injections of pituitrin. Dilatation of these vessels followed: (1) an increase in the carbon dioxide content of the blood, or a state of anorexia; (2) a condition of acidosis; and (3) inhalation of amyl nitrite (Wolff and Forbes,¹⁰ Wolff,⁹ Wolff and Lennox⁸).

Histologists such as Stöhr,⁶ Hassin,² and Penfield⁵ have also provided further evidence of a true nervous control of the pial vessels. They have demonstrated the existence of nerve endings in all vessels of the pia and choroid plexus. Nerve fibres have also been found in the walls of vessels entering the brain itself. These experiments and histological researches prove beyond doubt the existence of a nervous control of the superficial blood vessels of the brain, and, in all probability, of those entering the brain itself. From this it may be assumed that the blood supply of the brain is dependent on the state of tonus in the cerebral arteries and arterioles, which in its turn is under the influence of the sympathetic and autonomic nervous system. The cerebral vessels will respond to any disturbance of the normal balance existing between these two systems, whether this be temporary or permanent, in the same manner as the blood vessels in other parts of the body, and also to changes in the chemical composition of the blood. These facts would readily explain such conditions as migraine, epilepsy, and certain of the cerebral symptoms of arterial hypertension.

A Case of Pure Uraemia

Before describing the cerebral symptoms as met with in cases of hypertension a case of pure uraemia will be described, as it affords interesting points of contrast.

A. P., a male aged 36, was admitted to the neurological ward, Middlesex Hospital, on July 26th, 1934. For a few months previously he had suffered from headaches. However, he continued at work. On July 24th, 1934, he suddenly lost consciousness for a few minutes; the tongue was bitten, and he was incontinent. The past history was unimportant, except that in 1907 he was run over by a hansom cab and his "kidneys were damaged."

The patient had two generalized epileptiform attacks on the day of admission, one at 3 p.m. and one at 10 p.m. Between these attacks he was conscious but drowsy. There was marked pallor of the face; speech was dysarthric. Respirations twelve per minute; definite air-hunger. Fundi normal. No abnormal signs in the central nervous system. No muscular twitchings observed. Chvostek's sign negative. Heart: apex beat not felt, but no obvious enlargement; no added sounds. Pulse regular. Blood pressure 120/80. Tongue markedly furred. Liver and spleen not enlarged. Bilateral irregular renal tumours felt. Urine: slight trace of blood; albumin, 0.07 gram per 100 c.cm.; urea, 0.57 gram per 100 c.cm. Frequent blood-pressure readings on the day of admission and the following few days showed figures ranging between 140/75 and 130/80. During the first three days the twenty-four-hourly output of urine varied between 49 and 57 ounces with an intake of between 62 and 87 ounces. The following blood analyses were made (figures represent milligrams per 100 c.cm.):

	Urea	Non-protein Nitrogen	Alkali Reserve c.cm. CO ₂	Calcium
26/7/34	321	176	18	—
27/7/34	339	192	19	6
31/7/34	379	171	29	—
28/8/34	215	122	40	—
19/9/34	200	115	36	7.7
2/10/34	160	97	33	7.9

In the first forty-eight hours after admission there was no improvement. Five hundred c.cm. of 2 per cent. solution of sodium bicarbonate in saline was then given intravenously. Within twenty-four hours the patient seemed much better. Air-hunger was less marked, and he read a newspaper with interest. He continued to make slow progress, and gained one stone in weight whilst in hospital. He was seen at intervals during the next four months. He remained free from symptoms, and was most anxious to return to work. He died of uraemia in another hospital in February, 1935.

This example of polycystic disease of the kidneys presented, on admission, a picture of true uraemia. At no time was the blood pressure raised. The convulsions can be explained by an altered blood chemistry. The absence of any clinical evidence of tetany was surprising in view of the persistently low blood calcium figures. Temporary recovery in this case was scarcely expected in view of the marked degree of nitrogen retention, and it was undoubtedly helped by the use of intravenous sodium bicarbonate.

Before leaving this case emphasis must be laid on the fact that a number of those with true uraemia die without having had a fit.

The Hypertensive Cerebral Attack

When we come to consider those cases of hypertension which present cerebral symptoms—usually of sudden onset—and in which there is no evidence of nitrogen retention, we are confronted with a series of events ranging from severe headache to epileptiform convulsions and coma. An attempt has been made by the present writer to understand the pathogenesis of these symptoms. After studying a number of cases they were found to fall into one of two groups, depending on the presence or absence of cerebral oedema. Occasionally a case was met with which showed features pertaining to both groups. The term "hypertensive cerebral attack" is a simple one, and therefore has been adopted to describe these symptoms.³

With Signs of Cerebral Oedema

The patient is usually under the age of 40, and may be a child suffering from acute nephritis, or an adult with raised blood pressure, either primary in type or secondary to an old nephritis. Previous health has usually been good. Severe headache is the characteristic feature of these attacks; it may be referred to the frontal or occipital regions, and is often accompanied by vomiting, cerebral in type. The headache may continue for several days or weeks. Drowsiness is usual, and may develop into a mild degree of coma. Epilepsy; generalized or Jacksonian, may occur at the onset, but is more rarely met with, when compared with the type of hypertensive cerebral attack later to be described. Ophthalmoscopic examination almost invariably shows evidence of a hypertensive retinitis. The earliest change is a constriction of the retinal arterioles, which varies from time to time. Within a few hours of the onset haemorrhage, exudate, and papilloedema may make their appearance. In rare cases, of which I have seen several examples, papilloedema may be the only obvious change on ophthalmoscopic examination, and this finding in conjunction with severe headache and vomiting may suggest a diagnosis of cerebral tumour.

The outstanding feature of these attacks is the rise in blood pressure. In young persons the diastolic pressure may reach a figure of 160 or higher. The cerebro-spinal fluid pressure is raised. A marked degree of papilloedema is always accompanied by a high cerebro-spinal fluid pressure reading. In acute nephritis the changes in the urine will be characteristic of that condition. In the remaining cases albuminuria is a constant finding, as well as casts and red cells, which indicate an ischaemic condition of the kidney. Blood nitrogen figures are within normal limits, unless uraemia is a complicating factor.

Without Signs of Cerebral Oedema

The patient is usually between the ages of 40 and 65, contrasting with the previous type, which occurs particularly in younger persons. Hypertension has been present for a number of years. Cerebral symptoms, such as headache, temporary attacks of aphasia, weakness in a limb, or even a hemiplegia, may have occurred previously. In other cases there is no warning of the attack; in still others it is preceded by a severe headache or some minor angiospastic symptom such as aphasia. Two symptoms characterize this form—one is coma and the other convulsions; in many cases these two symptoms are combined. The state of coma is usually preceded by severe headache; the patient becomes drowsy, and then lapses into a state of unconsciousness, from which it is difficult to rouse him. This state may last for several days, during which the blood pressure is found to be exceedingly high. The plantar responses are, not uncommonly, bilaterally extensor. There may be retention of urine. In the first of these attacks recovery usually follows a fall in blood pressure.

In other cases there is no warning of the attack, but there is often severe headache. The patient suddenly becomes unconscious, and the various stages of an attack of epilepsy are passed through. In some cases consciousness returns in a few minutes; in others, however, coma persists for one or more days, and during this time there may be further epileptiform attacks, but not infrequently the duration of the coma seems to be out of proportion to the number of fits. Instead of being generalized the epileptiform attack may take a local or Jacksonian form, in which case consciousness may be retained. Bilateral extensor responses are temporarily present immediately following this form of attack. On ophthalmoscopic examination papilloedema is absent, but the retinal arteries are constricted. On lumbar puncture the cerebro-spinal fluid is found to be under normal pressure. The most significant finding is a high blood pressure reading, which is not due to the actual epileptiform attack, as a reading during the actual state of coma will show. The urine may contain albumin in varying quantities. The blood urea and non-protein nitrogen figures are normal, provided uraemia is not a complicating factor. Consciousness is regained with a fall in blood pressure. As a rule no organic signs in the central nervous system remain. However, in other cases, hemiplegic signs of varying degree may persist.

These attacks of epilepsy may occur at intervals, and are sometimes regarded as cases of idiopathic or senile epilepsy. It is noteworthy that they seldom occur at night. Their appearance late in life, with a negative Wassermann reaction and the existence of a raised blood pressure between the attacks, proclaims their relationship to arterial hypertension.

T. E. R. B., a male aged 48, had suffered from attacks of migraine. He always had a nervous and quick temperament. His blood pressure was known to have been raised for nine years. Early in August, 1934, he was seized with a very intense headache and became drowsy. Systolic blood pressure was 240. A large quantity of albumin was found in his urine, of which the output was diminished. He remained in

a stuporose condition for about four days, and thereafter recovered partially. It was noticed, however, that his memory was now poor. On September 23rd he complained of a severe headache. This recurred on the following evening with increased severity, and was accompanied by repeated vomiting. At the same time he became drowsy.

When examined on September 26th he lay in a drowsy, stuporous condition; he could be roused with considerable difficulty, but relapsed quickly again into a semi-comatose condition. He did not reply to questions, and his attention was poor. The skin of the face was markedly pale and breathing was slightly increased in rate and shallow. Pulse rate was 100 per minute. The pupils were normal. Ophthalmoscopic examination showed no papilloedema and no retinitis, but there was a marked degree of retinal arteriosclerosis, with spasm of the arterioles. Power was very fair in all four limbs. Deep reflexes were much exaggerated, and both plantar responses were extensor. The apex beat was in the nipple line, and was diffuse and thrusting; there was a short systolic murmur at the apex, with a tendency to tick-tack rhythm; blood pressure 290/148. Skin was moist. No muscular twitchings were observed. Urine showed a trace of albumin. Blood urea was 31 mg. per 100 c.cm. Non-protein nitrogen was 29; alkali reserve 56 c.cm. CO₂ per cent. Lumbar puncture showed a normal pressure of cerebro-spinal fluid. Venesection was performed, and 1/2 grain erythrol tetranitrate was given at eight-hourly intervals. The patient's condition did not improve, and the systolic blood pressure fell, whilst the diastolic rose. Death took place on September 29th.

The cerebral attack at the beginning of August, and the final one which proved fatal, were both associated with very high blood-pressure readings. There was a marked degree of spasm of the retinal arteries, which pointed to a similar condition being present in the cerebral vessels. There was no evidence that the bilateral extensor responses were due to a gross lesion. Their presence at a time when the patient was able to be roused suggests that they were due to the effects of a widespread ischaemia. Death took place as the result of cardiac failure, doubtless brought on by ischaemia of the heart muscle, associated with a rising diastolic blood pressure, which finally reached the figure of 160.

An Illustrative Case

The following case is an example of hypertensive epilepsy.

G. W., a female aged 45, was admitted to the neurological ward of the Middlesex Hospital on December 20th, 1932, as she had lost consciousness in a bus on that date. She gave a history of severe periodic headaches commencing at the age of 30. These were referred to the frontal region and were throbbing in character. They were never preceded by any visual phenomena or accompanied by vomiting. In 1927, at the age of 39, she had her first attack of epilepsy, and since then she has had seven further attacks. The attack is always preceded by severe headache, frontal and occipital, which comes on suddenly. If she is able to lie down quietly in the dark no fit usually occurs, otherwise after the headache has lasted for about five minutes she suddenly loses consciousness. She has been told that she goes stiff, and then struggles in the attack. Occasionally she bites her tongue, and is incontinent during the attack. The period of unconsciousness has lasted as long as two hours. After the attack severe headache persists for a day. Occasionally she has difficulty in expressing herself for a short time after the attack, but has never noticed any weakness in the limbs. No history of nycturia was obtained. Her general health between the attacks is good. In 1928 she was admitted to the Middlesex Hospital, under the care of Dr. C. E. Lakin, following her second attack of epilepsy. The blood pressure during three weeks in hospital varied between 180/138 on admission and 150/95. Blood urea was 40 mg. per 100 c.cm.

On readmission on December 20th, 1932, she was conscious, but complained of headache. The fundi were normal, apart from a moderate degree of retinal arteriosclerosis. There were no abnormal signs in the central nervous system. There were signs of moderate hypertrophy of the left ventricle. Blood pressure was 230/140. The Wassermann reaction was negative in blood and cerebro-spinal fluid; the pressure of the latter was 120 mm. of water. The urine contained a trace of albumin, but no casts. Blood urea was 38, non-protein

nitrogen 36 mg. per 100 c.cm. Within twenty-four hours her blood pressure fell to 200/120, and remained in that vicinity during her fortnight's stay in hospital. Since that date the patient has gained 3 stone in weight. She has had three further attacks, and has had to give her work up (she was a midwife) as the result of breathlessness.

This patient had her first fit at the age of 39. The occurrence of severe headache, recalling that of migraine, before each attack is of interest. Her blood pressure on each of the two occasions on which she was admitted to hospital following an attack was considerably raised, and fell rather more rapidly than could be accounted for by a day or two in bed. This suggests that the attacks of epilepsy were due to a sudden additional rise in blood pressure. There was no evidence of nitrogen retention on either occasion. A number of cases of epilepsy occurring for the first time in hypertensive patients over the age of 40 have been observed within a few hours of their admission to hospital. The blood pressure in such cases has always been much raised, and has subsequently fallen more rapidly than could be accounted for by a few hours' rest in bed. It is believed that in such patients a temporary further rise in blood pressure has precipitated the attack. The adjective "hypertensive" might be used in describing this form of epilepsy.

It is well recognized that epileptic attacks may occur in arteriosclerotic patients in whom the blood pressure is normal. In some of these cases the attacks follow a cerebral thrombosis. I have recently had under my care several of such cases in which the blood Wassermann and cerebro-spinal fluid were normal. Confusion may arise in this type of case, as headache may be present, and the differentiation from a cerebral tumour may be difficult, the question only being settled after some months of observation or by the occurrence of a sudden hemiplegia. Such attacks are almost certainly due to a localized disturbance of cerebral circulation, but independently of a rise in blood pressure.

Minor Angiospastic Cerebral Symptoms

Apart from these severe attacks certain further cerebral symptoms may occur in a patient suffering from arterial hypertension, and they are of interest, as they point clearly to the existence of spasm of cerebral vessels. These consist of attacks in which temporary paresis, aphasia, numbness, and disturbances of vision may be present. A feeling of numbness and tingling in the hand and around the mouth on the same side may be experienced, sometimes accompanied by a motor type of aphasia, if the vessel affected is in the left side of the brain. Such an attack may last for varying periods, from a few minutes up to forty-eight hours. This may be followed by a permanent paresis; in such cases vaso-constriction, presumably, is severe, and the resulting ischaemia produces permanent damage. Accompanying these cerebral attacks there may be evidence of peripheral vaso-constriction of the limbs, either as a dead or tingling feeling in the hands or feet, or of pain in one lower limb, of the type seen in intermittent claudication. Vision may be affected, independently of a retinitis, by spasm of a branch of the posterior cerebral artery; the loss of vision is usually temporary.

Aetiology

The factor common to both varieties of cerebral attacks is a raised blood pressure. The actual precipitating cause seems to be a further rise in blood pressure. This rise may be only temporary. Volhard has suggested that in young persons the cerebral capillaries seem unable to adjust themselves to the rapid rise in pressure, and cerebral oedema results. As soon as the cerebral circulation has adjusted itself to the new conditions prevailing, either spontaneously or as a result of treatment, the

symptoms may subside. I have seen more than one case in which, although the cerebro-spinal fluid pressure was much raised (for example, 280 mm. of water or above) the patient at the time of the lumbar puncture was free from headache. If the intracranial pressure is very high a "pressure cone" may be formed through the forcing down of the tonsils of the cerebellum into the foramen magnum. In such a case attempts to relieve the pressure by lumbar puncture may be followed by death.

In the type of hypertensive cerebral attack without evidence of cerebral oedema the sudden rise in blood pressure induces spasm of cerebral vessels, with resulting ischaemia. The symptoms which follow probably depend not only on the degree of spasm, but on the situation and number of vessels affected. If the process is a local one, confined, for example, to a branch of the middle cerebral artery, Jacksonian convulsions may be produced. If, on the other hand, the angiospasm is widespread, then, in addition to generalized convulsions, coma results. That an additional rise in blood pressure is not the sole cause of the cerebral symptoms in this type of attack is made clear by the fact that variations in blood pressure are probably not uncommon in hypertensive patients, yet convulsions are comparatively rare.

The second factor which seems to predispose a hypertensive patient to convulsions is the previous occurrence of a cerebral thrombosis, by virtue of the local circulatory disturbance consequent upon such a lesion. The experimental work of Forbes, Wolff, and their collaborators has opened up a large field for speculation as to the effects of changes in the chemistry of the blood on the cerebral circulation, and it is certain that here lies a third factor.

It might be objected that an arteriosclerotic vessel is unlikely to undergo spasm as a result of changes in its walls. The type of case which is now under consideration is not the atherosclerotic with athéromatous plaques on the circle of Willis, and in which raised blood pressure is quite often absent, but the victim of high blood pressure and diffuse hyperplastic sclerosis in whom the small arteries and arterioles in the body are widely affected. Such changes, except in their later stages, should not materially interfere with the contractility of a vessel. Furthermore, since the lumen of the smaller vessels is narrowed through thickening of the intima it would require a smaller degree of spasm to produce ischaemic phenomena.

Prognosis

Attacks of the type described in this paper are naturally serious in view of the nature of the underlying process. In the form accompanied by cerebral oedema the immediate outlook is favourable, provided steps are taken to combat the increased intracranial pressure. The ultimate prognosis will depend in some degree on the type of case. In a young adult the state of the kidney is an important factor. During the acute phase of generalized vaso-constriction it may be impossible to decide whether the hypertension is primary or secondary to an old kidney lesion. The effect on the kidney will be much the same in either case—namely, a sudden reduction in its functioning power as a result of the disturbance of its normal circulation. As long as the nitrogenous contents of the blood remain within normal limits then the immediate prognosis is good. Subsequently it may be possible to determine the damage to the kidney which the sudden wave of raised blood pressure has caused. In favourable cases the albumin disappears from the urine, and tests for renal function may show good figures. Such cases may go on for a number of years. The blood pressure usually remains, however, at a high level, and it is only a question of a very few years at the most before death results from renal failure or a cerebral haemorrhage. In

older persons recovery from the first cerebral attack is usual, but others are liable to follow, and a fatal cerebral haemorrhage or a cardiac failure may supervene. Again, an attack may be followed by actual thromboses, resulting in a permanent hemiplegia.

Treatment

As the essential factor in both forms of cerebral attack is believed to be a sudden additional rise in blood pressure accompanied by widespread vaso-constriction, treatment should be directed towards a reduction in pressure by the speediest methods. Good results have been obtained by a liberal venesection; the use of French's apparatus is advocated. In the form of hypertensive cerebral attack with signs of cerebral oedema venesection will help towards a reduction in intracranial pressure. Further steps should, however, be taken. Intravenous injection of hypertonic salines will diminish headache. Lumbar puncture should also be carried out, first, with the purpose of determining the extent to which intracranial pressure has risen, and, secondly, as a therapeutic measure. Care should, however, be taken in cases where the pressure is high, as by the withdrawal of large amounts of cerebro-spinal fluid a "pressure cone" may be formed, which may lead to a fatal termination. If the cerebro-spinal fluid is under moderately increased pressure (approximately 200 to 250 mm. water) it is safe to remove 10 to 20 c.cm. of fluid. If increased headache follows, then no further lumbar puncture should be done.

In the form of attack without signs of cerebral oedema lumbar puncture and intravenous hypertonic salines are valueless. Venesection is the most important measure. An inhalation of amyl nitrite may bring to an end a series of convulsions; if this fails a subcutaneous injection of 3 grains of luminal is usually successful. Erythrol tetranitrate, 1/2 grain three-hourly, may be given until the blood pressure begins to fall. In both forms an enema should be given at the first possible moment. The likelihood of further attacks may be lessened by the continuous use of vaso-dilators with a hypnotic such as luminal.

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H. Nevinny (*Zentralbl. f. Gynäk.*, September 14th, 1935, p. 2162) strongly recommends—and in his own experience has been greatly pleased with—a trial of injection into the presenting part in pelvic presentations (usually the buttock, less frequently the foot) of cardiazol, to diminish foetal mortality. Such injections, done in 142 primary pelvic presentations in 1934, gave in Nevinny's clinic among 130 live-born vaginally delivered babies weighing at least 1.5 kilograms a mortality of 5.4 per cent., among those weighing at least 2 kilograms 4.1 per cent., and those weighing at least 2.5 kilograms 3.7 per cent. Comparable figures in the previous experience of the same clinic and in other clinics showed about 10 per cent. total foetal mortality, in spite of the use of a general anaesthetic and frequent performance of prophylactic lateral incisions of the perineum. The injection of cardiazol, 0.3 to 0.5 c.cm. of 10 per cent. solution, is given as soon as the presenting part appears in the outlet: it was first proposed by Poeck in 1926.