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A Clinical Lecture
ON
**SUDDEN CEREBRAL LESIONS: THEIR
DIAGNOSIS AND IMMEDIATE
TREATMENT.**

DELIVERED AT THE POLYCLINIC.

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GENTLEMEN,—Sudden cerebral lesions are among the uncommon places of medical practice. They are sure to be met with, and yet not frequently enough to furnish practical experience adequate to deal with them, at any rate with the promptness and confidence that their urgency requires. They are among the most perplexing problems the practitioner encounters, more so indeed than he always recognizes. The nature of the lesion must be discerned at once, as clearly as the symptoms permit, in order that treatment may be wise and prompt. Yet the measures needed may be diametrically opposite in cases which present almost the same symptoms. Confidence in diagnosis is essential for decision in treatment. The urgency arises from the fact that with the practitioner who first sees the case rests most of the opportunity for treatment. After the first few hours the chief part of the opportunity is over. Hence the necessity for a sure grasp of the elements of the diagnosis on the part of those who first see the patient. These elements are not so extensive as to preclude the hope that I may be able to give you an outline of them in the brief time at my disposal, but no account, however exhaustive, no knowledge, however full, will prevent occasional perplexity. Then you must be clear in your uncertainty, and adopt measures which, whatever the nature of the case, can do no harm and may do some good. Experience shows that, even at the present day, a better knowledge of the elements of the diagnosis is really needed.

It is unnecessary to tell you that the most common evidence of a sudden lesion of the brain is hemiplegia, very rarely diplegia, affecting both sides. Sometimes there is only loss of speech or impairment of articulation. Rarely there is loss of sensation without affection of motor power, and not uncommonly there is loss of sight to one side—lateral hemianopia. This is a symptom always to be looked for, if there is reason to think an organic lesion is possible; it is quite as significant as hemiplegia, and generally overlooked. Often there are convulsions.

An organic lesion of the brain of sudden onset, coming on in a few minutes or a few hours, is a vascular lesion. Rarely the onset occupies one or two days. Moreover, in the vast majority of cases it is an arterial lesion; it is due to the rupture of an artery causing haemorrhage, or the closure of an artery causing necrotic softening. This also you know. But it is important to realize that the primary morbid state, although in the brain, is not of the brain itself. Its effects are manifested by disturbance of cerebral function, but the disease itself is of the vascular

system and circulation. The fact is of extreme importance, as we shall see presently.

The several arterial states which cause these lesions differ in their frequency in the first and second periods of life. In later life they are commonly due to degenerative changes in the walls of the vessels; in the former, to other disease. The causes that are active in the first half may, however, extend on into the second, but it is convenient to begin with the later forms. Most sudden lesions—most cases of hemiplegia, for instance—occur in later life, and therefore the problem they present is the most important because the most common.

DEGENERATIVE LESIONS.

Hence the first question in a case of sudden hemiplegia is the age of the patient. Has he reached the degenerative period of life? This may be said to begin at 50 in fairly normal individuals but earlier under two conditions. First, there is occasionally an inherited tendency to early arterial degeneration. We meet with families in which many members have suffered from hemiplegia during the later forties, and, in such cases, the probability of premature arterial degeneration must be recognized. Secondly, Bright's disease may induce arterial degeneration at a still earlier age. Bright's disease may lead to cerebral haemorrhage even in the early thirties. Certain constitutional maladies, such as leucocythæmia and pernicious anaemia, may have the same effect at any age. In the absence of these or other obtrusive influences, one of two lesions is probable, as you know—haemorrhage from the rupture of a minute artery which has dilated to a "miliary aneurysm," or necrotic softening from thrombosis in a larger artery the seat of atheroma, or in one or more branches arising at the atheromatous part. The distinction between the two is of immediate importance because the immediate treatment is opposite in some of its features.

The diagnosis rests on several indications. If all point in the same direction, a conclusion can be drawn with confidence. If they differ, the balance must be ascertained. For this it is not enough to count the points, the relative weight of each must be taken into consideration. The first point to be noted is not within the nervous system. It is the condition of the pulse and heart, the state of the circulation. The importance of this arises from the fact I just mentioned: these diseases are in the brain, but not of it; they are primarily vascular.

The first point, therefore, to consider is whether the state of the pulse and heart are such as to show a condition of the circulation likely, on the one hand, to dilate and burst an artery, or, on the other, to permit a clot to form within it. The two are opposite in nature, and so are their indications. High tension of the pulse and a strongly acting hypertrophied heart are the conditions that point to haemorrhage, and a soft feeble pulse with a weak dilated heart are the indications of thrombosis. If this element is decided in its character, it has great weight. A man was admitted to hospital with severe hemiplegia, which I think had come on with loss of consciousness. But from the state of his heart and circulation I expressed the opinion that it was thrombosis from atheroma of the middle cerebral. A few days later he suddenly became comatose, with hemiplegia on the second side. His condition so closely resembled that produced by ventricular haemorrhage that I changed my

opinion; I thought I had been wrong, and that it must be haemorrhage which had burst into the ventricles. He never recovered consciousness, and we found the first diagnosis was right; each middle cerebral was atheromatous and closed by clot. The fact illustrates the weight that may be placed on the state of the circulation, if its indication is clear. Distinct degeneration of the arteries of the arm is perhaps a little in favour of atheroma, but they may be soft when the cerebral vessels are affected. Tortuosity of the temporal arteries is sometimes regarded as significant, but it only means the mechanical influence of hard hats, and is no index to the state of the arteries within the skull. It must also be remembered that atheroma is present in most cases of cerebral haemorrhage, and softening may have resulted, so that the state of the heart and circulation is of more importance than the indications of degeneration of the arteries elsewhere.

If a heart, whether weak or strong, presents also a valvular murmur, the diagnosis from embolism arises, but the question can be better considered in connexion with this lesion. The presence of albuminuria, such as indicates organic renal disease, is in favour of haemorrhage before the degenerative period, but subsequently has little influence on the diagnosis. It conduces to atheroma as well as to miliary dilatation. Even a few haemorrhages on the retina leave the problem unchanged, but a high degree of albuminuric retinitis, especially if acute in course and haemorrhagic, is in favour of haemorrhage. The condition of the retinal arteries is of little differential significance.

Preceding cerebral attacks, if slight, and especially if repeated, are strongly in favour of thrombosis and softening. But a single attack of definite severity has much less significance. It may occur in haemorrhage as well as in softening. Moreover, this indication is always subordinate to any other strong evidence as to the nature of the present seizure. An attack of haemorrhage may have been preceded by others clearly thrombotic. It has been mentioned that atheroma is often found in cases of haemorrhage due to miliary aneurysms. In a recently published case, fatal haemorrhage into the pons was preceded by an attack having the features of thrombotic softening in the region of the middle cerebral, and found to be such and due to atheroma of that artery.

Other important indications are furnished by the conditions under which the attack comes on and by its features. A valuable series of facts, from fatal cases, has lately been published by Dr. Ernest Jones.* Important and instructive as they are, the limitation to fatal cases, essential for certainty, narrows much the application of the inferences to the needs of practical diagnosis.

Warning symptoms during the few days before the attack are an important indication of thrombosis. They consist of tingling in the parts afterwards paralysed, transient weakness, brief loss or impairment of speech, giddiness, and the like. They are probably due to the impairment of the circulation in the narrowed vessel. In haemorrhage they are absent, although slighter symptoms may immediately precede the onset in which they culminate. Probably this is because the sudden onset of the definite attack often does not correspond to the actual commencement of the escape of blood, especially when this is into the white substance and attains some size, a point I will refer to presently.

The opposite character of the two processes naturally leads us to expect a corresponding contrast between the causal conditions that precede the onset or in which it occurs—that haemorrhage would occur during exertion or mental excitement, thrombosis during exhaustion. The former is seldom to be traced—a fact made very clear by E. Jones. It may be that the small size of the arteries on which the aneurysms occur makes the effect of exertion on these slight; it may be that it is partly due to the fact that the onset of symptoms often succeeds, at an interval, the actual rupture. This may be the explanation of the cases often met with in which the onset is during the

exertion of dressing, which may cause a previous slight extravasation to become larger and produce laceration.

On the other hand, in thrombosis, a condition of fatigue, exhaustion, or depression is often to be traced just before the attack. It is important to recognize the three conditions on which this lesion depends: (1) There is the chronic disease of the artery; (2) often a blood state, which promotes coagulation, as in the gouty condition; (3) an enfeeblement of the circulation, which lessens the blood-flow, especially just above the narrowed part. At the sides of a branch, the orifice of which is almost closed, and where the inner wall is changed, a clot readily begins if the circulation is enfeebled. Physical fatigue is a frequent antecedent of thrombosis—a tiring journey, a walk unwisely long, a day's shopping, influences often combined with insufficient food. Diarrhoea may be the excitant, a potent cause of faintness. In other cases the attack follows mental distress, a sudden shock, or depressing grief, which for long may enfeeble the action of the heart. Such preceding influences are of definite significance as an indication that the lesion is thrombosis. In passing I may mention that if there have been no preceding slight attacks, such an excitant distinctly improves the prognosis, as regards a recurrence. It makes it probable that the state of the circulation had a larger share in the causation than the degree of atheroma. I have met with many illustrations of this.

The onset of the symptoms in thrombosis is much more frequently during sleep than in the case of haemorrhage; more than twice as frequently in Jones's series, in which, however, the cases of softening were few. When the attack is slow in course, as often in softening, it sometimes begins, in slight degree, in the evening, and becomes severe during the night.

The features of the attack vary in each lesion to a greater degree than they differ in the two lesions. Its course is partly determined by the rapidity with which the process develops. The onset of symptoms in cerebral haemorrhage is usually supposed to coincide with the commencement of the haemorrhage. It is doubtful whether this is always correct. The relation between the symptoms and lesion probably depends on the structure into which the blood first escapes. If this is the white substance, it probably first merely separates the fibres and causes few or no symptoms until it begins to lacerate them, or reaches grey substance which is readily torn. When the extravasation is first into the central ganglia, laceration occurs at once and the symptoms occur much earlier. The early separation of the fibres was shown, in an exaggerated degree, by a girl who, having had a blow on the left parietal bone some time before, had slight and equivocal symptoms for two days, and then suddenly became unconscious, with bilateral palsy, and died in a few hours. Beneath the site of the blow was found thickening of the meninges, which had prevented a traumatic aneurysm of a large branch of the middle cerebral bursting externally. The blood had escaped into the substance of the hemisphere and had extended as a thin sinuous riband, $\frac{1}{2}$ in. wide, from the aneurysm to the lateral ventricle. The blood had suddenly poured into the ventricles, but had apparently taken two days to make its way through the white substance. That which happened here doubtless sometimes occurs on a smaller scale from the rupture of a miliary aneurysm. These are always numerous and others adjacent may give way when liquid blood replaces external support, and they rapidly increase the extravasation and its effects.

Loss of consciousness occurs in either lesion, but in haemorrhage more readily than in softening, especially if the severity of the paralysis is considered. It is probably first due to irritative inhibition of the cortex, which is maintained by increased intracranial pressure. The laceration causes powerful irritation, and when the haemorrhage is into structures that have a wide relation to the cortex, as in the pons, a small extravasation, which can have little effect on intracranial pressure, causes early loss of consciousness, which endures. We can understand from what has been said that in some cases the loss is not the first symptom; slighter symptoms precede it, which are part of the onset, and, as it were, lead up to it.

In thrombotic softening loss of consciousness depends chiefly on the size and importance of the artery that is closed and on the suddenness of the closure. The size of the vessel involves the extent of cerebral tissue that is

* Dr. Ernest Jones, The Onset of Hemiplegia in Vascular Lesions. *Brain*, 1905. He has ascertained the facts, as far as recorded, in all the fatal cases contained in the old case-books of University College Hospital, and, in publishing the results, he has compared them with the facts ascertained by others. Only verified cases can be sure evidence, but the records of a general hospital unfortunately contain a disproportionate number of lesions quickly fatal.

suddenly deprived of blood. Closure of the middle cerebral, for instance, commonly causes it; while that of one of its branches to the cerebral ganglia usually does not. The clot more often forms in a branch than in the main artery, and so this symptom is comparatively infrequent. For that reason there is a history of loss of consciousness in a larger proportion of the cases that die in hospitals, because thrombosis in a main artery is more often fatal. When loss occurs it is usually an initial symptom, because it coincides with the actual arrest of the blood supply to an important region of the brain.

But old hospital records are liable to fallacious interpretation, because a patient is often said to be unconscious when he is only in a condition of what we should call partial stupor. Indeed every degree of delirium, even slight, is popularly designated as "loss of consciousness." It is surprising how different are the first statements of friends from those ascertained by careful questioning, and this is one of many points on which records are of value only if taken with the special point in view. It is, therefore, essential to ascertain, regarding a case seen at a later stage, whether what is called loss of consciousness is a definite comatose state. If it is initial and brief, and attends a lesion that is evidently extensive, this is rather more likely to be thrombosis than haemorrhage, while the latter is more probable if it develops soon after the commencement of the symptoms, and is prolonged. But, unless it is profound and of long duration, it must only be regarded as one of the series of symptoms to be considered and compared, as evidence of the nature of the lesion.

Convulsions at the onset are more frequent in softening than in haemorrhage, at any rate if they are unilateral; and their significance is greater if they are partial and repeated, and attend imperfect hemiplegia. Their occurrence in such cases two or three days after the actual onset has still greater significance of the same character. The reason is because they are generally caused by partial disease of the motor cortex, and a small cortical lesion is much more frequently due to thrombosis in a terminal branch of the middle cerebral than to haemorrhage. On this point also the evidence of hospital cases is misleading. True aphasia has the same significance for the same region. If it occurs alone, with little evidence of hemiplegia, it is almost always due to arterial closure. If it accompanies the onset of complete right hemiplegia no inference can at first be drawn from it, because transient aphasia may attend a lesion even in the central ganglia which interrupts the motor path. But enduring aphasia means destruction of the cortex, which is seldom due to haemorrhage. A week or more must elapse before the duration has this significance, but it may be suspected from the first, if complete aphasia exists with only slight hemiplegic weakness, or if accompanied only by weakness of the tongue and face, the indication of softening is still more definite.

Locality has some further significance. Hemianopia, alone or with hemianaesthesia, suggests softening, because this is, as a fact, more frequent in the region of the posterior cerebral artery than is haemorrhage. The same is true of lesions of the pons. Consciousness is readily lost, in haemorrhage early, and may be attended with rapid and fatal hyperpyrexia. Coma also attends thrombosis of the basilar, but usually comes on gradually as deepening stupor, always of the gravest augury in such an association.

Lastly, early signs of considerable irritation are in favour of haemorrhage. The laceration of the nerve elements has this effect in a marked degree. It causes loss of consciousness by upward irritative inhibition, and sometimes, though not often, it causes convulsions. It also, acting downwards, may cause early and great rigidity, but sometimes initial flaccidity of the paralysed limbs, even with loss of the knee-jerk during the first twelve or twenty-four hours, while on the second day it becomes excessive, with foot clonus. Early rigidity with clonus may be met with during the first few days in softening, but the irritative inhibitory relaxation is probably confined to cases of haemorrhage. Considerable depression of temperature during the first day—two, three, or four degrees below the normal—is a sign of haemorrhage. A rise of temperature to about 100° about the third day is more common in softening.

The rare cases in which death occurs in a few minutes

do not afford time for a diagnosis; there is usually haemorrhage into the lower part of the pons or the medulla, or into the third ventricle. Cases of primary haemorrhage into the membranes, not due to the rupture of an aneurysm, are too uncommon to make it worth while to confuse you with their complicated facts.

"Ingravescent," the steady increase of coma from a slight to a profound degree, when it takes place in a few hours, signifies haemorrhage. When it occupies one or several weeks it is usually the result of spreading thrombosis from extensive atheroma. As just stated, a peculiar state of deepening stupor, from which at first the patient can be roused, often attends thrombosis of the basilar, other evidence of which can usually be found. A rapid rise of temperature to a "hyperpyrexial" degree sometimes attends ventricular haemorrhage, but is especially suggestive of a lesion in the upper part of the pons, which may be of any character. It gives great significance to anomalous ocular symptoms; usually it means death, but I have seen a patient who survived a temperature of 110.8° , apparently due to embolism of the extremity of the basilar.*

To sum up. The patient is in the degenerative period of life, or has advanced kidney disease. The indications of haemorrhage are few; hypertrophy of the heart and a tense pulse; an onset is attended with loss of consciousness, either profound and of long duration, or disproportionate to the degree of paralysis, and there is often a considerable initial depression of temperature, and sometimes complete flaccidity of the paralysed limbs. To these symptoms the absence of the indications of thrombosis may be added.

These indications are more numerous: A feeble, soft pulse, and weak, often dilated heart, sometimes irregular; previous attacks, especially of slight degree; transient premonitory symptoms, often headache; the onset often deliberate, with loss of consciousness if the paralysis is severe. Convulsion at the onset is not of much significance, but, later, is in favour of softening; and so with aphasia, which, if lasting, indicates softening. So does much impairment of articulation or swallowing, suggestive of a pontine lesion; and also hemianopia, showing that the mischief is in the region of the posterior central artery.

In the immediate treatment, some measures may be adopted in each lesion. A gouty state is common, and generally presumable, because age-imperfection of the organs generally leads to an approximate condition, and justifies some lithia or potash, combined with a diuretic, nitrous ether and spirits of juniper, to which some digitalis may be added—more if thrombosis is suspected than if haemorrhage is probable. The opposite rule should obtain with an aperient. If a clot has formed, only gentle action of the bowels should be secured; if a vessel has ruptured, an active purge is wise, such as croton oil, to lower the blood pressure. Salines, it should be remembered, seldom alone act well in the horizontal posture; they need the aid of gravitation. Blood should be drawn to the extremities.

If there is reason to regard the lesion as thrombosis, little more can be wisely done. We have no practical agent to reduce the coagulability of the blood. Citric acid is said to have this effect, and may be given as lemon juice in plenty of water. When haemorrhage may be confidently diagnosed, the treatment should be such as to increase the tendency of the blood to clot. Salts of calcium have this effect, and may be given in whatever form can be most promptly obtained. If the lactate or chloride is not at hand, common chalk may be converted to a soluble salt by any suitable acid that is available. Arsenic, by hypodermic injection, has a similar effect, and $\frac{1}{2}$ grain of the arsenate of sodium may be given in a few drops of water. There is no reason to believe that small doses of iodide of potassium, which are constantly given at the early or later stage, exert the slightest influence.

But one measure is imperative in every case of either kind—physical tranquillity. It should surely be superfluous even to mention this, but experience shows, too often, that it still needs to be insisted on. Even in the slightest

* The patient had mitral constriction and the attack was just after rheumatic fever. The clot probably broke up and passed on into the terminal branches (perhaps supplying the red nucleus or part of the cerebellum), leaving universal and enduring inco-ordination of movement. The temperature was observed and carefully verified by Mr. Townsend Barker, of Fulham.

case, if the patient must be moved, it should be as little as possible, and he should be absolutely passive. Some months ago I saw a man who had an attack, probably of hæmorrhage, at his office in a provincial city. By medical consent, instead of being moved passively to another room, or the very nearest place available, he was put into a cab, taken to and over a long ferry, and then driven three miles over the roughest, most jolty, road in the United Kingdom. I can testify, for I traversed it afterwards in a similar vehicle. When he reached his home he was unconscious, and his state when I saw him later, showed that the damage to the brain was the most severe and extensive that could possibly be survived. Let me mention also a striking contrast to this. A lady was seized with hemiplegia, probably also due to hæmorrhage, while consulting the late Sir Russell Reynolds. She was laid on the sofa of his consulting room, and from that room he would not allow her to be moved. There she lay until her death some weeks later. He would not permit any degree of inconvenience to him to weigh against the slightest risk of increasing the danger to her.

PREDEGENERATIVE LESIONS.

During the first part of adult life, up to 45 or 50, most sudden cerebral lesions are due, as you well know, to arterial closure. They are due to embolism, or to thrombosis the result of syphilitic disease of the wall of a cerebral artery. These are not, indeed, limited to this period. Each occurs later, during the period of arterial degeneration, and it is then that the chief diagnostic difficulties are encountered. Each may occur in earlier life, but arterial disease from inherited syphilis is very rare.

EMBOLISM.

In considering the problem presented by cerebral embolism, it is better to leave aside the cases that are due to malignant endocarditis. The presence of this is sufficiently evident by its special symptoms; its embolic effects are often multiple and severe; they may be attended by extravasation of blood, and in the membranes this may be the chief manifestation of multiple minute emboli. These cases furnish an undue proportion of the fatal cases met with in general hospitals or in their records. But the simpler form chiefly concerns us in the general diagnosis of sudden cerebral lesions.

In the predegenerative period the diagnosis of embolism, as a rule, presents a simple problem. It depends on the discovery of a source from which the embolic plug has come. With rare exceptions, this source is a diseased valve on the left side of the heart, and therefore the presence of an organic cardiac murmur, aortic or mitral, in the absence of any other causal condition, justifies the diagnosis. Not only does it justify, it is essential for the diagnosis. An embolus has sometimes come from a clot in a dilated left auricle, but mitral disease usually coexists with this. It has sometimes come from a roughened area in the aorta, but a murmur may accompany this, and the source is not frequent enough to be important. I have never met with it. Some mysterious cases suggest that minute plugs may reach the cerebral arteries through the lungs, but the diagnosis in these cases is scarcely justified. Local thrombosis, perhaps set up by particles brought from the general circulation, is a more reasonable explanation.

Yet difficulties may arise even at the time of life when the diagnosis is simplest. No features of the onset have such a special relation to embolism as to afford much help. It is sometimes useful to remember that the mitral orifice is a far more frequent source of a plug than the aortic, and that mitral constriction is specially prone to give rise to detachable vegetations. Perhaps this is because the blood flows through it slowly during the diastole, when vegetations easily increase in size, and swiftly during the auricular systole, favouring their detachment. But no murmur is overlooked so easily or so often as the simple presystolic. I have more than once known this fact to cause a clear case of cerebral embolism to be regarded as altogether mysterious. Moreover, some months after the onset the absence of a cardiac murmur does not exclude embolism, because acute endocarditis may have passed away and left the heart sounds clear. A known cause of endocarditis just before the onset may, in that case, be adequate evidence. A girl, during chorea, attended with endocarditis and a loud systolic mitral

murmur, had sudden severe left hemiplegia. A year later no murmur could be heard, but subsequently, without any fresh inflammation, a presystolic murmur developed, the contraction of the once inflamed orifice having produced constriction.

A very important fact is that in embolism there are no related preceding symptoms. There may have been a slighter preceding attack of the same nature, but this is rare. The state of the vessels is normal until the embolus is brought by the blood current. Suspicion should therefore be felt as to the nature of any case, apparently embolic, in which premonitory symptoms were distinct, especially unfamiliar headache. A clergyman of 35, with a double aortic murmur, had an attack of hemiplegia, naturally ascribed to embolism. But he had much headache for some weeks before the attack. A few months after the hemiplegia a node developed on the forehead, and a history of syphilis in his younger days was obtained. Indirectly the case reminds us that, as I have said, the aortic orifice is not a common source of embolism.

Embolism may occur also in the degenerative period of life, at any age, and the diagnosis is then much more difficult; indeed it is often impossible. Embolism less commonly results from degenerative changes in the valves than from actual endocarditis, but it does occur from them. Such changes often exist in cases of thrombosis, and sometimes in hæmorrhage. In proportion as the indications of either of these lesions are pronounced, the existence of valve disease has less significance, and it has also less the older the patient. The problem of distinction from hæmorrhage seldom arises, unless the cardiac affection is aortic regurgitation with much hypertrophy, and then the signs of hæmorrhage have undiminished significance. Usually the problem presents itself in cases of mitral disease with a feeble heart, in which a clot may have been formed *in situ* or carried from the heart. In some cases the occurrence of premonitory symptoms, or previous slight attacks, added to the evidence of arterial degeneration, decide the diagnosis in favour of thrombosis. No features of the attack are of special value in the diagnosis. There is a slight excess of lesions on the left side of the brain in embolism, and also considerable aphasia is more frequent, while pyrexia during the first week is more pronounced, but the preponderance of these is not enough to have diagnostic weight.

One point is sometimes of significance; embolism of the basilar is much rarer than thrombosis, and is rendered especially improbable by indications of closure of the branches of this artery to the pons. The region of the cerebrum supplied by the terminal arteries in which the basilar ends is also seldom the seat of embolic softening; hence this is rendered unlikely by the presence of hemianopia.

It is probable, indeed, that embolism sometimes occurs in late life when its diagnosis is not justified by the symptoms. As I have said, it is uncertain how far it is associated with thrombosis in cases of septicaemia, in which particles that can pass through the lungs seem to be arrested and to excite a secondary clot in the brain, but such cases are beyond my subject.* The distinction between embolism and thrombosis is not of great practical importance in regard to treatment, which differs little in the two conditions. It has some significance in prognosis. In embolism there is far less danger of a recurrence than in atheromatous thrombosis.

It must be remembered that valvular disease is one of the chief causes of intracranial aneurysm, by embolic transplantation of inflammation from the heart. Hence, at any age, even in the young, heart disease may accompany the most fatal form of cerebral hæmorrhage. In embolism loss of consciousness is not rare, but is seldom of long duration. Symptoms that strongly suggest hæmorrhage are not made less significant by the presence of heart disease, however young the patient. In these cases there is not the tension of the pulse or unexplained hypertrophy of the heart met with in the ordinary form.

Of the treatment of embolism there is little to say. It is chiefly that of the cardiac and general state. Whatever

* So also are the cases recorded by Newton Pitt in his third Goustonian lecture (1890) in which no source for a plug could be discovered. I have necessarily passed unnoticed many points that are outside the limitation of practical diagnosis.

tends to excite the heart must be carefully avoided and its action steadied and strengthened, with the object of hindering the extension of the clot and of promoting the establishment of any collateral circulation that may be possible. It is believed that sometimes a soft plug breaks up into particles small enough to pass away, but this can only be possible during the first few hours, and no means of promoting it are known, nor would any attempt be wise. Occlusion, once established by distal clot, must endure, and so must its effects, unless the blood supply can be renewed by collateral communication.

SYPHILITIC ARTERIAL DISEASE.

The other cause of sudden hemiplegia in the predegenerative period of life is thrombosis in an artery narrowed by syphilitic disease of its walls. Although the primary condition is a slow, gradual process, the actual closure is sudden or rapid, because the flow of blood through the narrowed passage becomes less and less, until at last clot suddenly occludes it. The thrombus probably begins to form just beyond the narrowing, where the flow is reduced to the middle of the lumen, and at the sides the blood must be almost stationary. It must be so, whether the cavity of the main artery is thus reduced, or the orifice of a branch at the spot diseased is progressively narrowed. It is important to realize this fact, that the vessel is closed by a process independent of the syphilitic disease which causes it, because the clot cannot be influenced by the treatment that may lessen the disease in the arterial wall, and an artery once closed is closed for ever.

This cause may be assumed, in any case during the predegenerative period without heart disease, with confidence when previous syphilis is known, and with probability whenever it is possible. It is rendered possible by any risk of infection. The fact should never be forgotten that, among the subjects of certain specific lesions, one out of every five, on an average, is unaware of the primary disease. In the case of married women, in whom the problem often arises, premature births adequately confirm the suspicion, or some other conclusive indication may be found. Without such indications, less harm is done in women by treatment that may be needless than by questions to the husband that may cause enduring distress.

Always remember that the absence of positive evidence has little negative weight. Yet how often we hear, "It cannot be syphilitic, for the children are quite healthy." Such a fact does not neutralize the slightest ground for suspicion. Always look carefully over the skin, especially of the legs, for any suspicious scars. Although vascular disease is usually early, it sometimes coexists with symptoms generally late. I have found in several cases loss of the light reflex of the iris, always strong evidence of preceding syphilis, and also the even more conclusive leukoplakia on the tongue.

In most cases the absence of any other causal condition, and the fact of certain or presumable syphilis, suffice for the diagnosis and render the special features of the attack of subordinate importance. But this is not always the case. Heart diseases may coexist, and the possibility of embolism then arises, or the patient may have reached the degenerative period of life. Then the conditions and characters of the attack may be important.

If a cardiac murmur is found, remember that aortic disease is not a common cause of embolism, and is sometimes caused by syphilis. The most important evidence is afforded by premonitory symptoms, especially headache, during a week or more before the onset. Sometimes there is a history of mental dullness, giddiness, or of numbness, tingling, or passing weakness in the limbs afterwards paralysed. Some symptom of this character is to be heard of in more than half the cases; showing as it does a morbid state before the attack, it is an almost conclusive distinction from embolism. But such premonitory symptoms are of less weight in the degenerative period of life, when they may be due to atheroma, but even then severe unaccustomed headache seldom precedes an attack due to any other cause.

No features of the onset present a special character or frequency sufficient to be of strong weight in the diagnosis. It occurs during sleep in a third of the cases (14 in 44). Loss of consciousness is rare. Transient loss can, of course, only be recognized when the onset is in the

waking state, but of these it was described only in 12 per cent. Perhaps this is because branches of a large artery are more often closed than the trunk itself. Neither convulsion nor considerable aphasia is common, but either may occur. Initial convulsions are prone to occur afterwards. Compared with other morbid processes, syphilitic disease has a disproportionate tendency to affect the basilar artery sufficient to be of some diagnostic significance in a doubtful case.

When the past history of a patient is known, the length of time that has elapsed since the primary disease is an element that may properly be allowed some weight. After what interval is arterial disease intrinsically improbable? I have found that one half of the cases occur during the first five years after infection, and a quarter during the second five years; during the first fifteen years, seven-eighths of the whole occur, and after twenty years examples are doubtful. Hence the probability is low after fifteen years and negligible after twenty. But we may not draw any similar inference from the age of the patient. The lesion is, indeed, rare in late life; it is absolutely rare, and still more in proportion to other sudden processes. But syphilis may be acquired at any age, and in later life the tendency to secrecy is far greater than in earlier years, and is often, indirectly, the cause of death. A man of 65, for long alcoholic, presented symptoms of a lesion of the pons, sudden right hemiplegia with a great degree of impairment of articulation, presumably due to thrombosis of branches of the basilar, consequent on atheroma. But it was preceded for two or three months by an unusual amount of occipital pain. A few days after the onset of the right hemiplegia, his left side suddenly also became paralysed, and he died. The day before his death his doctor was informed by a friend that the man had a severe attack of syphilis at 55. Characteristic syphilitic disease was found in the lower part of the basilar, a recent clot throughout it and in the posterior cerebrals, and an area of softening in the left side of the pons.

I remember another case of basilar thrombosis due to the same cause in a woman of 60, who had a presystolic murmur; there was also atheroma of the middle cerebrals, and advanced mitral constriction was found. Without knowledge of the syphilis, the recognition of the exact lesion in such cases is impossible. I mention these cases also because they illustrate the tendency of the basilar to suffer.

It is needless to tell you that iodide should be promptly given in such a case. Twelve or fifteen grains may be given every three hours for the first two days, then every four hours, and reduced to three times a day at the end of a week. Larger doses are unwise, because they may increase the coagulability of the blood. You may afterwards employ a course of mercury, but lesions of the type of that in the arterial walls seem to yield very promptly to iodide, and the system can be saturated with iodide as it cannot with mercury. Of course, nothing can influence accomplished occlusion. A thrombosed artery can never again become permeable. Improvement generally follows, just as it does in embolism, because the initial symptoms are more severe and extensive than those that are permanent. The reason for energetic treatment is that the disease generally exists in other arteries also; its amount cannot be known, and some other vessel may be on the verge of closure. Equally important is the prompt treatment of premonitory symptoms. I remember one case in which I believe a practitioner thus averted thrombosis in branches of the basilar, perhaps in its trunk, and probably thus saved the patient's life. Of course, it is desirable also to adopt the measures recommended for atheromatous thrombosis.

MISCELLANEOUS LESIONS.

These processes do not exhaust the causes of sudden lesions of the brain. Puerperal hemiplegia is probably due, in most cases, to thrombosis in a normal artery in consequence of the blood state. A tendency to coagulate with readiness seems normally evolved to facilitate the closure of the uterine vessels when the placenta separates, and sometimes to become excessive and to lead to the formation of a clot where it is undesirable. This influence may co-operate with another cause. If there is heart disease, the blood state may assist vegetations to form on the valves, or increase their size, and so may lead to embolism. This is clearly the cause of post-puerperal hemiplegia in some cases. Again, the same influence may apparently

facilitate the formation of a clot in an artery previously the seat of syphilitic disease. The recognition of the latter is difficult and depends on preceding symptoms, such as headache, etc., combined with evidence of the constitutional malady. Its importance arises from the fact that iodide, then necessary, is to be avoided in other cases, lest it increase, even in the slightest degree, the clotting tendency of the blood.

The hemiplegia that occurs during or after acute disease is generally thrombosis from exhaustion but sometimes embolism. Endocarditis may result from almost any acute specific disease, and least rarely from scarlet fever. A grave sudden lesion in tuberculous patients is generally venous thrombosis on the convexity of the brain. In these cases the causal association is sufficient guide to the diagnosis.

A sudden lesion, sometimes severe, leaving lasting hemiplegia, occasionally occurs in young adults, chiefly girls, who are in feeble health. Beyond this, no indication of its nature can be discerned. The heart is healthy, and no suspicion of arterial disease can arise. The general health, though not good, is not attended by such prostration as would reasonably account for spontaneous thrombosis. Yet no other cause is conceivable. The cases are very rare, and I think it is possible that the accident is determined by some congenital anomaly in the arterial distribution. We sometimes meet with such, unsuspected, and they are occasionally found to have altered the symptoms of disease. It is conceivable that such an anomaly may enfeeble the circulation in some vessel, so as to determine the formation of a clot in it, when the blood state is inadequate alone to induce the thrombosis. The treatment for such a case should be that suited to thrombotic softening.

The sudden cerebral lesions that occur in infancy constitute a subject too large and too special to be dealt with now. I have endeavoured to help you to perceive more clearly the nature of the cases that occur in adult life and to adopt measures that can do no harm and may do good, sometimes much good. What you achieve you cannot know. That which we prevent is always unperceived, but we may feel sure that it is definite, if not distinct, and often it is greater than we conceive.

A Lecture

ON

LAËNNEC AND THE EVOLUTION OF THE STETHOSCOPE.

DELIVERED BEFORE THE MEDICAL DEPARTMENT OF THE UNIVERSITY OF OXFORD.

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LAËNNEC was a very remarkable man, a Breton by birth, a man of very enthusiastic disposition, distinguished by his love for his country and by his great talent for original research. At his birthplace, Quimper, the French Government erected a statue to his memory in front of the fine cathedral, and at a later date they published a cheap edition of his great work at their own expense.

His father was a lawyer, who died early, but our Laënnec was educated by an uncle, a professor of medicine at Nantes, whose influence gave a distinct bias to his studies, and he soon showed a great predilection for medicine and morbid anatomy. Shortly after he was qualified he was sent as an assistant surgeon to the Army of the West to quell the troubles in the Morbihan, and this was his only military service. On his return he went to Paris and joined the clinic of Corvisart at the Charité Hospital, at once commencing work of singular merit, chiefly in morbid anatomy.

He fortunately became acquainted with Bayle, who was also himself interested in tubercle, and the two became great friends and fellow workers. He remained three years in Paris, working indefatigably, and collected no fewer than 400 clinical cases, of which he took notes and drew up detailed descriptions, and these formed the basis

of his future researches. His first published work was *Histoire d'Inflammation du Péritoine*, consisting of a series of cases detailed in a very clear and satisfactory manner, illustrated by much learned annotation, and terminated by general conclusions, specifying the anatomical characters and signs of peritonitis in a more accurate manner than had been previously done. His next publication was a *Lettre sur les tuniques qui enveloppent certains viscères*, addressed to Dupuytren, then principal anatomist in the School of Medicine. He also gave a course of lectures on pathological anatomy, which, it seems, was his favourite study previous to his being given charge of wards.

In 1816 he was appointed chief physician to the Necker Hospital, and then it was that he took up clinical medicine, and was in the same year rewarded for his labours by the discovery of Mediate Auscultation. In June, 1818, less than two years after the introduction of the stethoscope, he published his great work entitled, *De l'Auscultation Médiate, ou Traité du Diagnostic des Maladies des Poumons et du Cœur, fondé principalement sur ce nouveau moyen d'exploration*. The book has gone through five editions, two of which were translated into English by Sir John Forbes and Dr. Theophilus Herbert. The production of this momentous work was too much for the delicate frame of Laënnec; it injured his health and obliged him to resort to his native air a month after its publication. He recovered after a long stay in his beloved Brittany, and returned to Paris to receive the public appreciation which his great discovery warranted. He was made Professor of Medicine and of Clinical Medicine of the College of France. His lectures were attended by zealous students from every country in Europe, among whom was the late C. J. B. Williams. He showed the utmost courtesy and consideration to all who were attracted to his clinic by his great name and the wish to perfect themselves in the use of the stethoscope, and he was a most successful teacher. Nevertheless, with all his engagements, he did not neglect his great work, which he enlarged and corrected and brought to a second edition, which again exhausted his physical powers and compelled him once more to retire to his native province.

He had been attacked with pulmonary consumption, the very disease which he had done so much to elucidate, and he died on August 13th, 1826, at the age of 45, presenting all the features of advanced consumption.

In order to estimate the debt of medicine to Laënnec we must glance at what thoracic pathology was before the stethoscope was introduced.

The only means for diagnosing lung disease or diseases of the heart were, first, the well known *percussion* method, invented by Avenbrugger, which Laënnec did much to advance, and, secondly, that of *palpation*, of which a great exponent was Dr. Matthew Baillie, who was blamed by Sir Henry Hallford for thus treating a patient with too much familiarity. Thirdly, there was what has been called non-mediate auscultation, that is, simply by putting the ear to the chest wall with linen intervening. This is sometimes impossible with females, and it is not possible to auscultate the supraclavicular or suprascapular regions in this way. There was, therefore, a great want of some means of diagnosis, and Laënnec's discovery met it. His discovery was made in this way, to quote his own account: "In 1816 I was consulted by a young woman labouring under general symptoms of diseased heart, and in whose case percussion and the application of the hand were of little avail on account of the great degree of fatness. The other method just mentioned being rendered inadmissible by the age and sex of the patient, I happened to recollect a simple and well-known fact in acoustics, and fancied, at the same time, that it might be turned to some use on the present occasion. The fact I allude to is the augmented impression of sound when conveyed through certain solid bodies—as when we hear the scratch of a pin at one end of a beam of wood, on applying our ear to the other. Immediately, on this suggestion, I rolled a quire of paper into a kind of cylinder, and applied one end of it to the region of the heart and the other to my ear, and was not a little surprised and pleased to find that I could thereby perceive the action of the heart in a manner much more clear and distinct than I had ever been able to do by the immediate application of the ear. From this moment I imagined that the circumstance might furnish