CLINICAL LECTURE

ON

THE DIAGNOSIS OF CERTAIN FORMS OF APOPLEXY.

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GENTLEMEN,—You have before you, in contiguous beds, two patients whose cases present certain common features, and also some marked points of difference. They both illustrate varieties in the history of the so-called apoplectic attack; our object is to determine the nature of the disease from which each is suffering.

Our mental process at the bedside, in attaining to a diagnosis, is to apply to particular cases definitions based on generalisations drawn from larger numbers. In some diseases these definitions are more accurate than they are in others, and in proportion to this accuracy the greater is the certainty of our art.

In diseases of the nervous system we have, however, mainly to do with symptoms which present themselves as disturbances in the form either of excess, deficiency, or perversion of the phenomena of innervation. In these, therefore, as in all cases where we found our diagnosis on symptoms, we have to look rather to the mode of their sequence and their combination with each other, than to any specific or pathognomonic signs, to aid us in our discrimination of individual diseases.

The diagnosis of any particular case, therefore, founded on this method, is the result, in a great measure, of a balance of probabilities, our final decision being formed according to the greater or less resemblance which any individual case may bear to a known group or sequence of symptoms rather than to any other; and it is proportionately facilitated by the definiteness of the resemblance to the distinctive characters of the group or sequence to which we endeavour to assimilate it.

Now this method of the arrangement and grouping of symptoms in cases of cerebral and nervous disorders has been most carefully and elaborately systematised, and in particular in the work on *Diagnosis of the Brain and Spinal Cord* by our Professor of Medicine.* It is the application of his data to the cases before us to which I now desire to direct your attention and at the same time to furnish you with some pathological explanations which may assist your memory in applying those generalisations from symptoms to special morbid conditions.

The cases for our consideration are both males, both past middle life, both otherwise healthy, though rather worn-looking, both of comparatively sound health previously to their present illnesses, and both (curiously enough) following the same occupation as shoemakers.

Let us recapitulate the main features observed in each. Robert Hines, aged 55, long subject to headache, was ten days before admission attacked by sudden vertigo, and in half-an-hour lost consciousness and fell off his seat. He recovered consciousness in twenty minutes, and complained greatly of frontal headache. *He was, however, able to work the next day, though his left arm and leg felt weak. The paralysis of the left arm and leg increased during the whole week, without any further attack of loss of consciousness.* There was no return of pain in the head. It is now seen that the left side of the face is flattened; there is deviation of the tongue and of the soft palate to the left, and the buccinator is partially paralysed. There is no ptosis. The left pupil is more dilated than the right, and contracts less under the influence of light. There is paralysis of the external rectus of the left eye. (This, slight on admission, increased in intensity subsequently.) Sensibility is found to be greatly diminished on the left side, but not absolutely destroyed. He knows when he is sharply pinched on the hand, or slightly pricked on the foot, but cannot localise the position. Voluntary movement of the left arm and leg is almost completely abolished. There is very slight voluntary movement of the left arm. *There is, however, tonic contraction, with considerable rigidity, of the biceps, great pectoral, and latissimus dorsi muscles on the left side. The flexors of this hand also are rigid, and the fingers are flexed upon the palm.* There is no rigidity of the leg. The electric irritability is diminished in the muscles both of the arm and leg on the affected side, and reflex movements are easily excited in the left leg. His mental state is peculiar. He has shown, since admission, intervals of composure, during which his speech has been good and his memory fair, though somewhat impaired. These, however, alternate with periods of delirium and excitement, in which he is

noisy and violent, and requires restraint to confine him to bed. He passes most of his urine and faeces in bed. The urine is acid, and free from albumen. The heart's impulse and sounds are normal. There are visibility and rigidity of the brachial and radial arteries. A minor degree of arcus senilis is also present. The lungs are somewhat super-resonant, but not to an extreme degree. The respiratory sounds are healthy. There is no pyrexia.

The other patient, Richard Antill, aged 49, has also enjoyed perfectly good health previously to his present illness. He was attacked suddenly, without prodromata, with severe pain in the head, affecting principally, if not entirely, the left side. After this had lasted a few hours vertigo ensued, followed shortly by a complete and sudden loss of consciousness. He recovered consciousness after about four hours, but his power of speech was destroyed for some hours longer; it was, however, regained the same night. The pain in the head lasted three or four days. On recovering, he found that his right arm and leg were partially paralysed, but that sensibility was not diminished. The motor paralysis was greater in the arm than in the leg. The paralysis slightly increased in intensity for a day or two after the attack. When admitted, three weeks after his seizure, his manner was composed and natural. He expressed himself clearly. His memory and apprehension appeared good. He had no affection of any of the special senses. He could walk unsupported except by a stick, though he turned in and dragged the foot; but his voluntary power in the right arm was very much diminished, and notably more so than in the leg. There was stiffness of the biceps tendon, and this gave him pain when extended. The flexors also of the fingers of the right hand were contracted. This contraction had increased of late. Sensibility was very tolerably preserved both in the arm and leg, there being little difference in this respect in the two sides. The electric irritability was notably diminished in the muscles of the paralysed side. No distortion of his face is observed when he is at rest, but paralysis of the same side as the limbs is observed when he smiles. There is no deviation of the tongue or soft palate. The heart is healthy; but the brachial artery is rigid and tortuous, and there is marked arcus senilis. There is no pyrexia, and the urine, passed voluntarily, is free from albumen. Since his admission he has had galvanisation regularly applied to the muscles of the right arm, with the effect of greatly increasing their electric irritability and of restoring the voluntary power. The contraction of the biceps and of the flexors of the fingers has also greatly diminished under this treatment.

Now in these two cases we have examples of the so-called apoplectic form attack. I use this word as one of clinical significance, having the meaning of sudden loss of consciousness, with impaired motor power and diminished sensibility, but without failure of the cardiac action. Such a definition includes, however, a wide group of diseases, from the majority of which the cases before us are clinically separated with tolerable ease. Before discussing them, allow me, however, to point out to you a few leading features which attend affections of the nervous centres, and which may serve as preliminary guides to point your inquiries in determinate directions. To these, it is true, exceptions occasionally occur; and it is to this comparative frequency of exceptional cases that the difficulty of the diagnosis of cerebral affections is mainly due. Still, however, the following observations will be found to be relatively true in the majority of instances.

In the first place, affections of the periphery of the brain and of the meninges are rarely attended with paralysis unless when they cause pressure on deeper parts. They are, on the other hand, commonly accompanied by mental excitement or perversions of the intellect, and by muscular cramp, spasm, or convulsion. Secondly, lesions of the central parts, on the contrary, have usually paralysis, motorial, sensory, or both, more or less complete, as one of their symptoms—the position of the paralysis, and its unilateral or bilateral character, depending on the position of the lesion. Thirdly, local hyperæmia of an active kind usually causes excitement. Local anæmia is usually associated with diminished power. Local anæmia in one part of the brain, associated with local hyperæmia in another part, usually presents combinations of both paralysis and excitement. General anæmia and general hyperæmia produce signs either of perverted action or of paralysis, in proportion to the degree in which they occur.

In speaking, however, of general anæmia as a cause of perversion of cerebral function rather than of paralysis, I believe that it must be limited in this application either to defective qualities of the blood or to a sudden interruption of its supply. In these cases, either mental excitement or loss of consciousness attended with convulsions may ensue. Anæmia gradually produced, either general or local, is more commonly associated with loss of power without definite excitement, unless it be accompanied by hyperæmia occurring in other parts of the brain.

In discussing these questions of disturbed circulation, I wish, however, to say a few words as to the chief causes of the disturbance.

* *Diagnosis of Diseases of the Brain, Spinal Cord, Nerves, and their Appendages.* By J. Russell Reynolds, M.D., etc. 1855.

Firstly, anæmia, using the word in the sense of a positive deficiency of the total amount of blood supplied to the brain may arise from several causes. *a.* It may result from weakened action of the heart; which, like the effect of large bleedings, or the sudden closure of the carotid arteries, may sometimes be attended with general convulsions. *b.* It may arise (and this is one of the most common causes) from an atheromatous condition of the main arterial trunks of the circle of Willis and of the basilar artery, diminishing the calibre and also the expansibility of their tubes, or locally from complete obstruction of an arterial trunk. *c.* It may *probably* be caused by contraction of the smaller arteries under nervous conditions, an effect analogous to the pallor, seen under emotion, in external parts. *d.* It may either proceed locally from local atheroma, or be caused by local pressure on the brain-substance.

Hyperæmia of the brain, on the other hand, may be active or passive. Active hyperæmia may be caused by increased force of the heart with diminished resistance of the arteries, or by dilatation of these under nervous excitement in a manner analogous to the action of blushing in external parts; or it may arise from undue stress thrown upon the collateral circulation by the interruption of a main arterial branch; and it may also probably be caused by the action of certain medicinal substances. Passive or venous hyperæmia may be caused within the brain by diminution of the propulsive force, through obstruction of the arteries, by pressure on the venous trunks, or by coagula forming in these, and by causes impeding respiration or the passage of the blood into or through the right auricle of the heart.

The two cases before us present the primary combination of loss of consciousness attended with unilateral paralysis. Of the whole group of apoplectic diseases, a certain number are attended with loss of consciousness and with *general* paralysis. We may, therefore, exclude from our diagnosis simple general cerebral hyperæmia, and anæmia, and the coma of Bright's disease; and also the so-called serous and nervous apoplexies, which are probably included under the two last-named conditions. We may also exclude ventricular hæmorrhage, which is usually attended with profound coma and with general paralysis or convulsions, and other forms of hæmorrhage at the base of the brain, or into the pons Varolii or medulla oblongata, which are generally marked by symptoms very similar to the ventricular form. It is beyond my purpose to dwell specifically on the diagnosis from one another of these different affections, and I would therefore direct your attention to the other diseases of this group which are marked by an initial attack of sudden loss of consciousness, followed by unilateral paralysis. These are arachnoid or other unilateral forms of meningeal hæmorrhage, some cases of tumour, softening of the brain, and hæmorrhage into the deeper parts of the brain-substance. Now we may, I think, exclude the idea of tumour in both the cases before us, because it is only very rarely indeed that its invasion is marked primarily by apoplectic symptoms; the most usual symptoms being cerebral pain, disturbances of vision, and signs of local pressure on special tracts of the nervous centres.

Meningeal hæmorrhages also usually come within the definition before given of the effects of diseases of the meninges. Their invasion is generally associated with excitement, with spasm or convulsion, and with *incomplete* paralysis; and, though one of the cases before us (Hines) has signs of mental excitement and of spasm of the muscles of one arm, his paralysis is, nevertheless, nearly complete.

There yet remain to be considered the two most common causes of apoplectic attacks ending in hemiplegia: viz., softening of the brain, and hæmorrhage into the substance of the brain. The diagnosis between these two affections is among the most difficult that we are often called upon to make; indeed, it is so difficult that some eminent men have pronounced it to be impossible. This arises from their symptoms being in many respects very similar. Their resemblance depends, in part, on the fact that they commonly affect similar regions; being usually found either in the corpus striatum, or optic thalamus, or adjacent parts; and also on the effect produced equally by both, of an interruption or destruction of the central influence of these parts on peripheral innervation.

The diseased conditions in which these disorders originate, and the manner in which their effects are produced, deserve, however, some consideration; as it is through these that we are, I believe, mainly enabled to understand such divergence in their symptoms as may frequently enable us to discriminate on which of these conditions such cases of paralysis as these before us depend. I must, however, tell you that softening of the cerebral substance may depend on several very distinct causes, and that the manner of its production not unfrequently materially influences the symptoms by which it is revealed. Setting aside for a moment the oedematous softening which occurs in tubercular meningitis, we find that local softening of the brain may arise from

1. Acute inflammatory processes.—These, however, are rare, unless when extending inwards from the periphery. They may in some cases apparently occur spontaneously; but more commonly they are the result

of extension of inflammation from other parts, such as the temporal bone or the sinuses. They may be latent in their course; but, when revealed by symptoms, these are usually of the nature of excitement, and consist of severe pain in the head, delirium or mania, various forms of paresthesia and imperfect paralysis, or spasm or unilateral convulsive conditions.

2. The more common forms of softening are all characterised by anatomical conditions of degeneration, signifying arrest or material defects of nutrition. They may both anatomically and clinically be distinguished under four chief forms: *a.* Softening from imperfect supply of arterial blood without complete arterial obstruction: *b.* Softening from complete but gradual arterial obstruction: *c.* Softening from complete and sudden closure of an arterial branch: *d.* Softening from obstruction of a venous sinus—usually the superior longitudinal. This last form is exceedingly rare. Its characters anatomically and clinically present a combination of superficial softening of the convolutions and of meningeal hæmorrhage. Both the first two forms are commonly senile. They result from changes in the arteries, either of the nature of atheroma or of calcification, to the effects of which on the circulation I have already alluded. The primary effect of these is, then, the production of a local anæmia, which, when the affection of the arterial system is extensive, may be unattended by any compensatory hyperæmia in the adjacent vessels. Their secondary effect is to give rise to malnutrition of the brain-tissue. This variety of softening is therefore usually attended with so-called "prodromata" resulting from diminished functional powers due to the defective circulation. These appear in the forms of defective memory, loss of single words, or slight defects of articulation, or the wrong substitution of a single word—or of a letter of a word—for another; slight numbness, tingling, or other forms of defective sensation occur in the extremities. The motor power may also be partially enfeebled. Attacks of vertigo occasionally occur, which may lead to momentary or transient losses of consciousness; and all these are most common when the patient's health is in any way enfeebled, as after an attack of indigestion or diarrhoea, or when exhausted by undue exertion. Cases of this nature may advance with slow progress, through gradually increasing hebetude and impaired powers, owing to the diminished functional activity of the brain; usually, however, there is at some period of their course a more sudden seizure. This is determined by a more acute impairment of nutrition, which may be simply an exaggeration of that previously present, or may be caused by a comparatively sudden obstruction of the artery through a coagulum formed by spontaneous thrombosis.

In the first case, the condition is nearly akin to forms of white gangrene in the extremities. There is little inflammatory action—little compensatory hyperæmia, and the pathological state is that of simple white softening, the histological characters of which consist in a fatty degeneration of the brain-cells and fibres. The failure of nutrition commonly follows some exertion, mental or bodily, which has exhausted the tissue beyond the reparative powers of the circulation; and a simple softening is commonly the result, the symptoms of which are usually only an exaggeration of those previously existing. The loss of consciousness may be only very transient, and the coma is seldom profound. The face is often pale. There is sometimes vomiting, but no cramp, spasm, or convulsions at the outset. On recovery, one side is found with a variable degree of diminution of sensibility and of motor power, which, however, are usually gradually restored in proportion as the injured part recovers some part of its nutrition by rest. When the disturbance has been more severe, and the paralysis is not recovered from, some rigidity may later appear in the limbs, owing to atrophy of the muscular tissue.

In the second class, resulting from coagulation of the blood in the vessel, the change is more sudden, and there is consequently greater disturbance of the circulation and of the nutrition of adjacent parts.

The coagulation is generally determined by the combined effect of the narrowing of the calibre of the vessel through atheroma and through consequent retardation of the blood-stream (which in all cases predisposes to this event), and also frequently by roughness on the wall. Such events may affect single arteries, when the adjacent branches may shew no traces of similar disease. The effect is a more or less suddenly produced local anæmia, with a hyperæmia in adjacent parts proportioned to the dilatation by pressure of the blood in the collateral arterial branches. This varies in degree; but, in proportion as these are capable of allowing an increased stream of blood to pass, is the amount of active congestion in the vessels surrounding the affected part. With this is usually associated some inflammatory reaction of the margins of the softened portion. Capillary rupture takes place from increased pressure. Oedema also ensues; the hæmatine of the blood effused is absorbed by the tissue, and a red softening is produced, due in great part to this imbibition. It is, however, attended with hyperæmia of the meninges, owing to the stress thrown on the collateral circulation, and

also partly to the delay of the blood in the veins due to the diminished *vis à tergo* from the obstructed artery. The redness, thus, as you see, in part arises from imbibition, and in part from increased adjacent vascularity from collateral pressure; and it is no measure of the *inflammatory* nature of the process, which is, comparatively speaking, slight in proportion to the appearances present. The obstruction which causes the softening may in some cases lead to more extensive extravasations, when the vessels around are brittle and unable to bear the increased stream thus thrown on them; and hence softening has been rightly considered to be one of the *modes* by which hæmorrhage may be produced.

Very closely akin to the preceding conditions are those observed in the form of softening resulting from a sudden obstruction of an artery by a plug or embolus carried from other parts of the body. The most common sources of these are fibrinous coagula formed on the valves or the auricular appendage of the left side of the heart, or those derived from the pulmonary veins. Their usual seat is in the middle cerebral artery of the *left* side, since they almost invariably are carried up the left carotid artery in preference to the more tortuous course through the innominate. In one respect the phenomena produced are sudden, since such obstructions often occur in arteries which previously were perfectly healthy; and hence the attack of apoplexy which they produce is almost invariably sudden, unless under exceptional cases, when a larger mass has been preceded by *minuter granules*, which have only arrested the circulation in single capillaries—a phenomenon which has been shown by MM. Prevost and Cotard to be capable of producing prodromata similar to that occurring in the other forms of senile softening. These latter cases are, however, very rare, though they may be produced artificially. The more common event is, that loss of consciousness, with hemiplegia, takes place suddenly. It arises from suddenly induced local cerebral anæmia, attended like the previous form with collateral hyperæmia, which is usually the most intense in this form, because the arteries, being commonly unaffected by previous disease, are more distensible, and a greater strain is thrown upon the capillary circulation; hence the adjacent redness is greater, and capillary extravasation is more marked, and the œdema of the surrounding brain is considerable. The tendency, however, to larger extravasations is, comparatively speaking, less, because the elasticity of the vessels more easily permits their distension without rupture. There is also a greater “reactive” tendency of the tissue immediately surrounding the parts deprived of its blood, and thus the “irritative” characters of the process are more marked.

Now, if we look at the effects produced by these processes, we shall see that the symptoms, by which the last two forms of softening are disclosed to us, correspond to the effects resulting from a combination of local anæmia and local hyperæmia with “irritative” action occurring in different parts of the brain. We have loss of consciousness primarily produced; seldom, however, very intense, or of long duration. In the form where the attack is caused by disease of the arteries, it is usually preceded by prodromata, and, as the occlusion even from coagulation may be gradual, so also may be the period of invasion; and it is often preceded by delirium, due, I believe, to the increased vascularity of the meninges and peripheral parts taking place gradually; while in the second variety the attack is usually unpreceded by any cerebral symptoms. Both are, however, generally subsequently attended by signs of perverted but excessive cerebral function; by mental excitement, delirium, convulsion, or more commonly by cramp or tonic rigidity of the muscles, occurring at an early period of the attack; by pain in the limbs; or by some other forms of *paræsthesia*. In some cases slight appearances of pyrexia are observed; the skin becomes hot and dry, and the tongue dry and furred; but these symptoms are seldom marked, and are often entirely absent. Their presence is generally the measure of the degree of inflammation surrounding the softened part; and when they occur they are usually associated with more marked forms of delirium, and also of tonic rigidity or cramp of the muscles.

Let us contrast with these characters of softening those which are observed anterior to and during an attack of cerebral hæmorrhage. This necessarily results from rupture of an artery; and implies, as a necessary antecedent condition, either increased vascular pressure or diminished resistance of the arterial wall. Frequently a combination of both concurs in its production.

The conditions of increased vascular pressure are threefold. They are those which I have already mentioned to you as causes of general hyperæmia. Hypertrophy of the heart has had a great part assigned to it in the production of cerebral hæmorrhage; but many cases occur without this being present. I believe that quite as frequent a cause is that state of “determination” of blood which arises from dilatation of the vessels, owing to relaxation of their coats from the arrested action of the sympathetic nerve.*

Hindrances to the return of the venous blood are, however, also a very common cause; and their efficiency in producing rupture is seen in the frequency with which the extravasation follows muscular efforts during which respiration is impeded.

Of the causes capable of diminishing the vital elasticity of the arterial coats, and thus rendering them more liable to rupture, atheroma, calcification, and fatty degeneration, are among the most common. There are probably, however, other conditions of degeneration as yet unknown to us, which may have a similar influence, and which explain the cases where no changes are recognisable in the vessels, and especially such as take place during the earlier periods of life. One point, however, deserves notice; viz., that the rupture seldom takes place in the larger arteries, but frequently affects those only one degree removed from the capillaries in size; and that these are much less frequently the seat of true atheroma than of simple fatty degeneration. This distinction is not unimportant; as it may explain the comparative absence of prodromata in some cases of hæmorrhage, since fatty degeneration of the smaller vessels may be found unattended by such a degree of atheroma as is sufficient to diminish the supply of blood, by narrowing the calibre of the larger trunks. Such prodromata in cases of hæmorrhage are, I believe, generally due either to this cause, or to previous attacks of “determination” of blood arising from nervous excitement.

The effect of the extravasation of the blood into the brain-substance differs, however, markedly from the softening arising from the obstruction of an artery. We have here an element introduced which is entirely absent in softening; viz., pressure. The brain-tissue is pressed aside; and the effect of the pressure is the production of anæmia in the surrounding tissue. Hence, though the interruption of the circulation in the vessel which has given way would otherwise lead to collateral hyperæmia similar to that which occurs from the obstruction which precedes softening, this is prevented, in cases of hæmorrhage, by the pressure exercised by the blood-clot. We find, as a consequence, that the parts surrounding the clot are pale, that the convolutions above it are flattened, and that the pia mater over these is comparatively anæmic. I believe that it is this condition of anæmia from pressure on the surrounding brain-substance caused by the clot, as contrasted with the collateral hyperæmia in cases of softening, which mainly explains the differences observable in the phenomena of the two affections. Both may arise from very similar conditions of the arterial system; but softening, acutely induced, is usually attended by signs of excitement, mental, motor, and sensory, while the phenomena of hæmorrhage are more commonly those of simple paralysis.

The diagnosis of hæmorrhage, preceded by or attended with softening, is a question of greater difficulty, and may in many cases be impossible, though the mixed class of the phenomena observed may in some cases suffice as a guide. The distinction of hæmorrhage from the first form of senile softening is chiefly made by the more gradual invasion, and by the comparative incompleteness both of the coma and of the paralysis, in the latter, and also by the signs of “determination” commonly present in hæmorrhage.

If we compare these data with the phenomena observed in our two patients, we may, I think, conclude that Hines has had an attack of softening from obstruction by atheroma of the vessels, and that Antill's attack has been one of cerebral hæmorrhage. We find, however, that in neither was the attack preceded by any very distinct prodromata; but possibly the headache to which Hines was subject may be viewed in this light.

The attack, however, presented very distinct differences. In Hines it was slowly developed; and the paralysis was only fully produced after forty-eight hours, or even after a longer period. In Antill, although there was some slight increase of the paralysis subsequently to the attack, yet this was not nearly so marked as in the former case; and his loss of consciousness and of speech was much more distinct. I do not regard the headache of which both complained on their recovery as very significant. It is a symptom common to both hæmorrhage and softening, though not constant in either. In both the recovery of consciousness was, comparatively speaking, speedy and complete, though less rapid in Antill than in Hines. The subsequent progress of the two cases has been, however, very diverse. Antill has presented us with a gradual return of voluntary power, unmarked by any condition of spasm, and his intellectual faculties are unimpaired. Hines, on the other hand, has frequent fits of excitement, attended with a gradual failure of intellectual power; and his paralysis, instead of diminishing, has remained stationary.

I must here call your attention to the conditions of the muscles which, though presenting at first sight an apparent similarity, really are essentially different from one another; and this difference is one of great clinical importance. There is rigidity in both; but in Antill it is limited to the biceps and the muscles of the forearm. It appeared late; it was constant in amount; but it has diminished gradually after the affected muscles have been galvanised. In Hines, on the other hand, it affects

* In support of this, see Axmann's experiments.—Virchow's “Archiv”, xv, 431.

some of the muscles of the trunk as well as of the arm; it was comparatively early in its appearance; it varied in intensity at different times; and it has been, if anything, increased by the galvanic stimulus, when this has been applied. This difference is due, in Antill's case, to the commencing atrophy of the muscles which so constantly occurs in paralysed parts, and through which the muscular tissue wastes, leaving only the fascia and intervening fibrous tissue; and this, being devoid of elasticity, undergoes a steadily progressive contraction. The improvement under the galvanic action has been a further proof of this, shewing the return of the nutrition of the muscular tissue; and, as this has coincided with an improvement in his cerebral state, he has, in consequence, gradually regained his voluntary control over the motor action. The means by which this latter is accomplished is a question of very interesting features, but one upon which I cannot now enter. I believe, however, that, in the majority of cases in which late rigidity occurs after hæmorrhage, it is due to this cause. It is, however, I must tell you, believed to be occasionally due to inflammatory action arising around the clot. In such cases it approximates to the tonic contraction seen in Hines, which arises from inflammatory action affecting the meninges and the brain-substance surrounding the softened portion. It frequently affects also the lower limb, though this is not seen in the present instance.

Both these cases present certain points of resemblance in respect of the state of the vascular system, and this always deserves attention in cases of hemiplegic apoplexy. I have already told you that the states of the vessels giving rise to softening and to hæmorrhage are in many cases very similar. The state of the heart may, however, be sometimes a guide in cases where the symptoms are of a doubtful nature. The existence of cardiac hypertrophy or of disease of the tricuspid valve may afford a presumption that the nature of the cerebral lesion is hæmorrhagic. Disease of the aortic or mitral valve, especially if originating in rheumatism, would raise a well founded suspicion that hemiplegic apoplexy originated in the obstruction of the middle cerebral artery by a fibrinous coagulum carried from the left ventricle. The manner of the attack may also aid you, though in both these cases we could obtain no information on these points. Hæmorrhage is often preceded or accompanied by fulness of the vessels of the head, by flushing of the face, and heat of head. In softening, these signs are usually absent; the face may be pale, and the circulation is often depressed. Later, however, signs of "determination" may supervene, even in the latter disorder, though the mechanism of their production is not very evident. They are not improbably determined by a reflex effect, causing distension of the vessels in the attempt to establish the collateral circulation.

There are several minor points connected with the pathology of both these disorders which, though of considerable interest, I am compelled to pass by for the present. I wish, however, to direct your attention to some points of importance to which the pathology of these affections, considered in regard to diagnosis, leads us in relation to their treatment, though I can only now discuss them briefly and somewhat summarily.

In the first form of senile softening, our main indications are rest, the preservation of the normal tone of the circulation, and the avoidance of causes of cerebral hyperæmia, which may lead to the rupture of vessels in the softened parts. Bleeding and counterirritation are equally out of place and useless. Nutritious food and stimulants, administered with great caution, are the measures chiefly to be relied on. With these must be combined the maintenance of the nutrition of the paralysed limbs by friction, by passive motion, and by galvanism, until the nutrition of the brain is restored or the action of the injured portions is supplemented by that of adjacent parts. Lastly, the action of the bowels must be gently regulated, since a costive condition of these is one frequent cause of cerebral congestion. Cases of hæmorrhage, when coming before you after the active part of the attack has subsided, are to be treated in a very similar manner. You cannot remove or diminish the pressure of the clot by depletion, counterirritation, or antiphlogistic measures; and by lowering the patient you only weaken his reparative powers. When seen early, and there are signs of a determination of blood to the head, you may in some cases possibly diminish the amount of extravasation by general or local bleeding and revulsives to the extremities. If the case be seen after this has passed, though comparative inaction on your part is harder to practise in the presence of a threatening disease than what may appear to the friends more vigorous measures, this is in the majority of cases the more philosophical and the safer treatment. You have seen the benefit of rest and galvanism in the case of Antill; this is fortunately an example of which you will meet with many instances; though in others of greater severity the paralysis of the arm may be more permanent than it has been in his case.

In cases of softening from obstruction of the artery, your treatment may also be guided in part by the state of the circulation. You cannot reopen the obstructed vessel; and the compensating hyperæmia in the adjacent parts is in a measure only in accordance with a natural and

well known physiological process. It is, however, within your province to see that this does not overstep salutary limits. I need hardly tell you that venesection in the initial stages of the attack is likely to prove positively injurious. If, however, you observe signs of subsequent excessive determination, you may control this by cold, or occasionally by topical bleeding and counterirritation, by which means you may endeavour to limit the excessive dilatation of the vessels by acting on the peripheral extremities of the spinal nerves. Moderate purgation is also admissible in such cases. I believe that no good is effected by the use of mercurials; but the other indications for rest and nutritious food are called for in this as in the other affections of which I have spoken.

THE CLOSING OF BLEEDING BLOOD-VESSELS BY ARTERIAL FORCEPS.

By WILLIAM TAYLOR, M.D., Edinburgh.

MY attention has been directed to an article in the BRITISH MEDICAL JOURNAL, entitled "A New Method of closing Bleeding Blood-vessels by Moveable Forceps. By Thomas Nunneley," etc.

The experiments therein recorded bear a close resemblance to a series in which I was engaged, while a pupil of Sir James Simpson, two years ago; and the results of which I had then the honour of submitting to him and to Professor Spence. These, although not published then, were embodied in a thesis written shortly afterwards, and presented to the Medical Faculty of the University of Edinburgh in March 1866.

From a rough draft of that paper which I have by me, I make the following extract; and I think the "acupressor arteriæ" therein described will be found to supply the desideratum which Mr. Nunneley requires.

Extract from Thesis.—This may be regarded as emphatically the iron age in surgery. The knife has, in a great measure, supplanted the caustic-holder, the metallic suture, the silken seam; and now acupressure threatens the extinction of the ligature. We cannot have too many changes, provided that each be an improvement on that which went before; and, as acupressure is manifestly a step in the right direction, let us lend our aid in bringing it as near to perfection as possible. A healthy cicatrix is a far prettier sight than a suppurating sore, and a week's superintendence far preferable to a month's dressing; and if such benefit to our patient and such pleasure to ourselves can by any means be obtained, even once out of a hundred times, I hold it to be our duty to give him the full benefit of that chance. But the advocates of acupressure expect—and I think reasonably—far higher triumphs than these; they expect to see at least fifty per cent. of their patients benefited by its use; and they have the pleasing reflection that those wounds which fail to heal by first intention under its application, belong to a class the healing of which is not by any means favoured by the ligature.

Although convinced of the supracellence of acupressure, I confess that, like many others, I have failed to see the possibility of its universal application, more especially to the deeper seated parts; and, although firmly believing that the ligature is most certainly doomed to disappear from our vision within half a dozen years, still I think it probable that something besides the straight needle, but made to act on the same principle, will be required to aid in supplanting it.

Acting under these convictions, I set myself to devise a method whereby all the advantages of acupressure might be applied not only in regions where direct pressure from a straight needle would be unattainable, but also in parts which are fairly accessible. The result has been the construction of a self-acting wire-compress, which I have dignified with the name of the "acupressor arteriæ". This minute piece of mechanism is so constructed as to retain a firm hold of the artery, and, although hid from view by being enclosed within the divided tissues, it can be removed at pleasure any time after the operation. It is, therefore, tantamount to a metallic ligature, for the separation of which we have not to wait for two, three, or four weeks, but which can be removed at any moment without the slightest trouble. It consists of a needle, or rather a piece of steel wire, bent upon itself in the middle and turned into a single spiral, which constitutes a spring; the extremities are so adjusted that, by the aid of the spring, they lay hold of the artery to be compressed. The compress is then left in the wound, but connected with the spiral at the end is a small wire; this is allowed to hang out in the same manner as the ligature is at present. When the time has come for its removal, a slender silver tube is passed inwards over the wire until it reaches the acupressor arteriæ; the wire is then held firmly by the left hand, and the tube passed steadily inwards: with the right. On reaching the converging arms of the instrument, they be-