Hemiplegia

arising from chronic softening of the brain.

by

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to

original case

Method of curing: the point of injection of the brain.
Hemiplegia is a form of paralysis, well deserving the careful study of the physician, from the circumstances of its frequent occurrence, the formidable nature of the disease producing it, and the importance of a knowledge of correct principles for its treatment. It is an effect, whose cause is a lesion in the central nervous system. It may vary in degree and also as regards the functions affected; thus, there may be simply a paralysis of motion on one side, the function of sensation and also that of appreciating heat and cold remaining unimpaired. In other cases the paralysis of motion may be incomplete or there may simply be a paralysis of sensation. These differences depend upon the locality and extent of the primary lesion, which may be, in order to act either by producing pressure on the brain, by solution of continuity of its substance, by destroying the integrity of its tissue, or by the presence of a poison interfering with the chemical vital changes produced.

The chief varieties of central lesions arise from hemorrhages, haemorrhage, inflammation, abscess, tumour, rupture of nervefibres in white matter, or, as some think, from congestion and round effusion. There are one or two other conditions occasionally causing hemiplegia.
The diagnosis of several of these affections is sometimes exceedingly difficult, and no uncertainty is still greater, if we endeavour to define the precise part of the brain which is the seat of the disease. Some progress, however, has been made of late years in this department of medical inquiry, and we may justly hope that as our knowledge of the functions of the several parts of the normal encephalon increases, so will we be able more correctly to appreciate its morbid manifestations.

A great impetus has been given to the study of the pathology of brain disease by the use of the microscope, through the medium of which, we have arrived at a more correct knowledge of the nature of those frequently occurring cases, where the paralysis is either the immediate or the indirect result of a degeneration of the substance of the brain itself, whereby it is rendered incapable of generating or conducting nervous force, or wherein though the disease has not advanced so far as this, the organ, when subjected to any unusual strain, gives way in some situations.
The proposition is not unanimous in opinion as regards the nature of the pathological conditions inducing softening, and extensive microscopical experimental observations are yet required to elucidate the very difficult questions arising in the inquiry.

In this paper, I propose to confine myself to the consideration of hemiplegia in its relations to atrophic disease of the brain, as I fear the prescribed limits of an inaugural thesis forbid the determination of the subject in all its etiological bearings.

Under the influence of certain favouring conditions, it is found that there is no tissue or organ in the body which is exempt from the liability to fatty degeneration. We see examples of it in the 'arcus senilis' in the corners of some old people, and if we were to make a microscopical examination of a muscle long denied from paralysis, we should probably find evidences of this change. Of its exact chemical nature, we are not fully acquainted, nor is the proposition unanimous in opinion regarding the nature of the transforming process even some of its most familiar forms, as atheroma of vessels, lime, viewing it as a mere precipitation from the flowing
blood, others, as an excitation, to others again, as a
cellular metabolism of the coats.
It is not within my province to discuss these
different theories, and in reference to them, I would
simply remark, that neither seems inconsistent with fact, nor are they incompatible with
one another.
Fatty degeneration of the coats of arteries is more
apt to occur to people in the decline of life, and
particularly to those who have long indulged
in a highly carboacids diet such as alcohol
in its various forms, and have different exercise.
Chronic softening of the brain may result from
embolism, or the plugging up of a cerebral vessel by
a piece of fibrin which had originally be deposited
upon some of the fibrinous structures of the heart, an
occurrence not at all uncommon in acute rheu-
matism, & which having become detached had been
carried by the circulation into a vessel too small
to admit of its further passage.
When this accident takes place, the nerve substance
proper of the brain undergoes a process of true fatty
degeneration, the nerves cells become enlarged,
their walls dissolve, and the nerve tubes separate
and break up.
But a plug may form in an artery in another way & precisely the same results will follow.
Arteries affected with atheroma have thin inner walls thickened in many situations. Upon one of these aneurysms in a vessel of small calibre, the fibrine of the blood may become deposited and the circulation at this point in the course of time be totally arrested, or the deposit, before it has acquired sufficient bulk to plug up the whole calibre of the vessel at this spot, may become detached, swept away and ultimately lodged in a smaller artery. The portion of brain supplied by the ramifications of this vessel is thus deprived of its nourishment, and the process of fatty transformation then takes place. In these cases it is of course understood that the collateral circulation has not compensated for the cutting off of the normal supply.

The inflammatory form of chronic softening is the variety concerning which there has been so much discussion. Its essential feature is an atheromatous condition similar to that affecting the larger vessels, may affect the central capillaries, and the integrity of the surrounding tissue become in consequence destroyed. Dr. Hughes Bennett maintains that this condition of the vessels is the result of an arteritis, and that
the evuludation undergoes the process of fatty degener-

ation; in the case of the capillaries of the brain

becoming detached from the inner aspect of the wall

upon which he considers it to be deposited, and

becoming interpenetrated between, or among the nerve

fibres & cells, to which the degenerating process is

communicated & which in places become separated

& even broken up.

When this evuludation takes place, with great rapidity,
it constitutes "acute softening" of systematic arteries.
In the former form, it is "chronic cerebritis;"
the evuludation undergoing the fatty degeneration
instead of being absorbed; in the latter it is
"acute cerebritis." & I do not think the terms "red"
or "acute softening" can properly applied to them.
The fatty transformation requires time.

When the evuludation is poured out so rapidly as to con-
stitute "acute cerebritis," softening is not the result
but rather "hardening," if I may so use the term; the
subsequent softening arises from the conversion of the
evuludation into fat.

The cases so frequently described as proceeding
from "red softening" over their colour, I think, to
consequent proceeding death & to the circumstance that
in those situations, the disease had not progressed
So far as to obliterate this particularity, though it had to prevent it from contracting. The so-called red softening "therefore, I think may result from either the acute or chronic forms of the disease.

As before remarked, palsy disease may affect any tissue or organ in the body, and frequently attacks the kidney, a most serious complication, as by its interference with the secretory function of the gland, it causes the retention of urea and other effete or morbid products, which induce con
cavitations, a frequent proximate cause of apoplexy.

In consequence of the diseased state of the walls of the vessels, the circulation is deprived of one of its forces, and to compensate for the loss or impairment of vascular contractility, the left ventricle of the heart becomes hypertrophied, having more work to do, or to overcome a greater resistance. Hence, in proportion as the strength of the arterial walls diminishes, the disruptive force exerted upon them from within, increases; thus haemorrhage within the substance of the brain is a very frequent termination of the disease.

I shall now endeavour to describe how hemiplegia may result from simple softening of the brain.
The late Dr. Todd was of opinion that softening may progress so far as not to entirely prevent the performance of the functions of the part, but yet sufficiently so to impair the generating and conducting power of the nerve substance & to render it unequal to bear great effort or strain.

When subjected to this, the nerve fibres, being unequal to the high tension, give way and paralytic results. This explanation my revered teacher was accustomed to give, but though ingenious, I think at present it must simply be regarded as a hypothesis, as we do not know the nature of the molecular change the nerve substance undergoes during the performance of its function. There can be no doubt, however, that paralysis may result from simple solution of continuity of the fibres of the brain without languishment from morbid restlessness of the extremities & impairment of volitional power.

It often occurs gradually, producing at first numbness of the extremities & impairment of volitional power. It seems to me intelligible also, that when the nerve tubes have become soft & fragile, they are less able to bear disturbance & that slight conditions of congestion, which in healthier states of the system would have no noticeable effect, would now be adequate to produce their rupture & consequent paralysis.
It is thought a varicose condition of some of the cerebral veins may promote softening by the imbibition of the effused serum. If this be so, we should expect to find some lesions obstacle to the return of the venous blood from the head, such as would be offered by emphysema & other thoracic diseases. The imbibition of serum in hydrocephalus is also said to be a cause of softening. I do not think we have sufficient evidence of the power of this imbibition to produce softening, and it is probable the appearances which led morbid anatomists to recognize it as such, were simply the result of post mortem change.
Having described the pathology of softening, I now
propose to consider how we can diagnose the
condition and the particular part of the brain affected,
in cases producing hemiplegia not through haemorrhage,
but from inability of the brain, in certain parts, to
perform its functions.

In studying this and the other causes of hemiplegia,
I see instance the necessity for more accurate notions
of the functions of the various parts of the nervous
system. In a case of hemiplegia, if the patient were past
the meridian of life, had been addicted to moderate use of
liquors, had an aurae senni, signs of fatty degeneration
of the larger vessels, and hypertrophy with dilatation
of the left ventricle, we might justly infer that it
was due to softening of the brain.

On further inquiry into the case, we might perhaps find
that the patient had for some time exhibited diminished
motoric power, had complained of weakness of the
muscles on the side, almost dragging one leg in walking,
and of being unable to grasp with one hand as firmly
as formerly. If the paralysis had come on gradually,
or had assumed an intermittent course, lasting
for a few minutes or hours & then disappearing, the
probability of softening being the cause would be
still greater.
When the softening has affected a large extent of the brain, so as to more or less involve the convolutions, a diminution of intelligence is exhibited. At first, apprehension & attention may be enfeebled. The individual becomes less capable of receiving new ideas. The memory is impaired & more or less confusion of thoughts & incoherence. There is a tendency to dwell upon a single idea or group of ideas. With regard to emotion, there is remarked -- dullness or melancholy if not uncommonly, the expression of feeling seems little under control, as shown by the patient crying or laughing without assignable cause. Pain, sometimes localised, sometimes diffuse and of variable intensity, is also said by some writers to be a sign of white softening. I think, however, the absence of pain in the head is as pregnant as its presence. An occasional sense of weight and of feeling "as if the mind were going" is often complained of.
Painful sensations in the limbs are often present and are sometimes referred to the surface, sometimes to the muscles; in other cases, to the joints. There may be numbness, or coldness, or a feeling of formation. Hyperesthesia is common, anaesthesia—very rare.

If the hemiplegia has set in slowly & by degrees, the diagnosis is comparatively easy; it has then however to be differentiated from other conditions which may be followed by a similar result, particularly from tumour & chronic meningitis. If the paralysis set in suddenly, it is occasionally very difficult to distinguish it from the effect of haemorrhage into the substance of the brain, and in fact, when the latter is extensive & occurs in the center of a large area of softened brain tissue, it is almost impossible.

Sudden hemiplegia, as has before been mentioned, may occur from division of degenerated nerve fibres, acted on by by some unusual force, such as would take place during a violent voluntary effort. One important diagnostic symptom of this cause of hemiplegia, is to be found in the condition of the muscles, which are relaxed and remain so for a long period.
In sudden hemiplegia from a not very large clot, early rigidity of the muscles occurs. On the accession of paralyses of the first kind, loss of consciousness is rare, while in the second, it is frequent and probably invariable when the haemorrhage is rapid or extensive.

Electricity may be advantageously used as a means of diagnosis. If it be found that the muscles of the affected limbs do not contract so readily on the application of this stimulus, as those of the opposite side, we may infer that the hemiplegia is not the result of an irritating lesion, but simply of chronic softening of the brain. For its further differentiation, we must duly estimate the signs of the other causes of hemiplegia.
Having concluded that the hemiplegia is the result of degeneration of some part of the central nervous system, we endeavour to discover the extent of the disease & the precise seat of the lesion, which is the immediate cause of the palsy. Though the inquiry must in the nature of things meet with but limited success, it will bring its reward in enabling us to form a prognosis, a matter of importance, particularly to the patient's friends.

Acknowledging that the intellectual functions of the encephalon, sensation & the special senses are essentially associated with particular parts of the encephalon, these are to intimately connected by centripetal & centrifugal fibres, that the complete manifestation of some depends more or less upon the mutual cooperation of all. Hence a lesion affecting one centre will impair the function of another or of several.

In illustration of this, I may refer to those very numerous cases where one of the optic thalami had been affected with softening, followed by rupture of fibres, as the result of laceration or otherwise. These segments are considered to be the chief focus of sensibility, hence a lesion of one of them does not destroy sensation, at least permanently.
Though occasionally the shock does cause a temporary impairment, but invariably a motorial palsy takes place from the division of the efferent fibres of another segment more particularly connected with voluntary motion, (the corpus striatum) which pass through the thalamus.

As a rule, it may be said, that the functional disturbance in hemiplegia is proportioned to the extent of the central lesion.

The lesion is generally on that side of the encephalon opposite to the limbs paralyzed.

If there be simple paralysis of motion of the extremities of one side, sensibility not being heightened, the reflex movements — lively, the respirator and sphincters — unaffected as also the muscles of the tongue and cheek, the special senses — sound and the intelligence not much altered, we may infer that the lesion is probably confined to the corpus striatum alone, or to that centre and the optic thalamus, through which the motor tract runs.

In a case thus limited, the prognosis would be favourable, as under concurring circumstances and suitable treatment, the divided efferent fibres may reunite, and a softened condition of the brain
be arrested or even cured.

Hyperaesthesia is much more frequently observed in the less severe cases of hemiplegia than anaesthesia, and I believe it is diagnostic of the lesion being limited to the motor tract, as we find in experiments upon animals, that when the part of the cord concerned in voluntary motions is divided, the function of the un-injured part isesa and there is great hyperaesthesia. I have witnessed an experiment of this kind upon a rabbit.

The seat of sensibility is of much extent, and anaesthesia only accompanies the worst cases of hemiplegia, where besides this symptom, there is generally paralysis of the bladder, sphincter, and an emaciated state of the respiration from palsy of the diaphragm and the intercostals of the same side, diminished excitation, deviation of the tongue towards the sound side of the body, paralysis of some of the special senses and the buccinator and pharyngeal muscles. In these severe cases, the lesion is extensive, seriously involving the corpus striatum, the optic thalamus of the base of the brain. (Nucleus lentiformis and the Medulla Oblongata). They generally terminate fatally. The symptoms may develop themselves gradually; loss of consciousness, complete Cerebellum.
occurring in the very worst cases.

Andral has related several cases of hemiplegia caused by disease of the cerebellum.

There are a few cases on record of disease of the cerebellum being accompanied by a tottering gait, like that of a drunken man, these being stemmingly a defective power of co-ordination, but to intimate its connection with any contiguous parts of the encephalon, that we are not acquainted with any distinctive signs sufficiently distinctive.
Treatment.

It differs at the present day greatly from that notion prevailed formerly, in consequence of our improved knowledge of the pathology of the disease.

In some persons, there exists a great tendency to the assimilation of hydro-carbonates in normal situations; in others, tissues have a tendency to assume a degeneration of the fatty form. Both peculiarities are often hereditary; the latter, particularly so.

Two classes of people seem especially prone to paralysis from chronic softening of the brain. Those who have long indulged in rich carnoseous food, such as the various forms of alcohol, and have taken insufficient exercise, and those who, from their occupations having been of a sedentary nature or other circumstance, have had little muscular exertion, have not indulged in alcoholic or rich food, but rather on the contrary, have had insufficient nourishment. It has been frequently or perpetually the subject of trying cases, anxiety, and prolonged mental labour.
In the most cases of hemiplegia, there is little hope of recovery & our duties will be confined to rendering the patient as comfortable as possible.

If the case should be protracted, the patient ought to be placed upon a water-bed, the body be kept clean, the bladder regularly emptied by catheterism, the bowels cleaned by occasional enemas, & the food should be finely divided & of a nutritive kind.

Some practitioners, with the view of preventing bed sores, have the body frequently washed with a solution of tannin in water, or one of corrosive sublimate in spirits of wine.

In less severe cases, we would recommend the abandonment of beverages very rich in alcohol, the substitution of others of more tonic properties, a generous nitrogenous diet, as much exposure to the air as possible, and the adoption of any measures calculated to improve the digestion & strengthen the system. Mental labour, anxiety & all causes of emotional excitement should be avoided, and more particularly so, if there be disease of the heart, a frequent precursor of hemiplegia, to which I have before alluded.

The state of the kidneys should be investigated, and if there be albuminuria or other evidence of
disease, suitable remedial measures are to be adopted. Friction, frequently in the centripetal direction, may be applied to the paralyzed limbs and electricity is often employed with equal benefit, to promote the nutrition of the limbs & the tone of the muscles. To effect this, it should be localized in the muscles affected, so that they alone may be stimulated and the brain not irritated; hence, the electrodes (moistened sponges lodged in metallic holders, furnished with non-conducting handles) should be applied close to one another on the skin, in order to limit the current to the muscles beneath beneath & between them. The intermittences of the current should be rather slow.

The use of the electricity is simply to improve the tone and nutrition of the muscles & should not be applied too long after the stroke, when in fact, one and done there is no irritation in the brain so that the paralysis is due as much to debility of the parts as dependent upon central lesion.
Case illustrative of Hemiplegia caused by chronic softening.

J. D., a native of London, aged 55, a charwoman by profession. Has long been addicted to habits of intemperance. The family history is then traced, pasty complexion, says she has for some months had a feeling of drowsiness & severe pain & numbness in the left hand & arm, and that her feet have often felt cold. One morning, while she was rising from bed and pulling on her stockings, she suddenly fell, without at the time experiencing loss of consciousness, or suffering pain. It was soon discovered that the left extremities were paralyzed. At first, her speech was not affected. The muscles were flaccid & flabby. The sensibility not affected, it having been tried with the compasses. Her intelligence, which had been becoming duller during the preceding year, was now more so. She could appreciate least cold as usual. When galvanism was applied to the paralyzed muscles, it was found that a very strong current produced scarcely any contraction, while a moderate one produced violent movements of the muscles of the opposite side. The pupils were rather dilated & sluggish. The special senses were unaffected. Her urine yielded a deposit of albumen with the ordinary test.
The sound of the heart (which beat feebly) was accompanied by a murmur.
Her appetite and digestion remained much as usual, though the former at length became voracious. The bowels were rather sluggish.
She continued in this condition nearly three months, when she experienced another attack, suffering this time loss of consciousness, which persisted till death. The right side of the face was now pallid, the tongue turned to the left and the two buccinator muscles flapped synchronously with the respira-
tory movements, which were purely diaphragmatic. The sphincters were pallid. She died within thirty-six hours after the attack.

An examination of the body was made on the following day. The brain was found to have undergone very extensive softening, the cerebral convolutions here there were soft readily broke up when a thin stream of water was poured upon them. In some situations too, the colour seemed to be more of a dirty white than the usual greyish hue. The right corpus callosum and optic thalami were pultaceous, and the softening had extended to even the mesencephalon of the upper part of the medulla oblongata.
On a microscopic examination of specimens taken from various parts, oil granules, fragments of nerve tubes, capillary vessels, encrusted with granular matter

A case of hemiplegia conjectured to be caused by thickening arising from arterial obstruction.

J. S., aged 53. Has been subject to attacks of gout, his fingers exhibiting deposits of urate of soda about the joints. Towards the apex of the heart, a distinct but feeble mitral systolic bulbo-v sound is heard at the apex of the heart.

One day while at work, he felt suddenly ill, in a few minutes, he discovered he had lost the use of his right side. His consciousness was not affected. The tongue deviates to the right side. Sensibility in the arm is somewhat impaired. Reflex action, as shown on tickling the sole of the foot, is somewhat evaded. The muscles of the paralyzed limbs are perfectly relaxed. The radial artery feels hard & unyielding. There is no indication of disease of the kidneys.
The treatment of this case was simple & consisted in improving the general health by tonics & nutrition, food & the administration of small doses of alkalis & calomel.

The patient progressed favourably & at length made a complete recovery.

The diagnosis formed of this case originally, was that the paralysis was due to simple, uncomplicated white softening from mere delinquency of the nerve fibres, induced by the accidental plugging up of a small cerebral vessel by possibly a detached piece of the deposit on the mitral valve or its chordae tendinae.

The gradual recovery explained on the supposition that but a limited portion of the brain was impaired in its nutrition, so that the collateral circulation becoming gradually improved, the nutrition of the part was re-established.
Probably the most fruitful proximate cause of haemiplegia is "sanguineous apoplexy". It most often takes place during temporary congestion of the brain, the vessels not being strong enough to resist the increased internal pressure, in consequence of the diseased condition of their coats, which sometimes is so far advanced that they may give way from the ordinary pressure of their fluid contents. Hemiplegia from haemorrhage like the variety just described occurs more frequently after middle life. Cerebral haemorrhage may occur at all ages, and in persons in whom there is no sign of fatty degeneration. It is however rare in young persons. It probably still more so in people whose brains have not undergone any atrophic change. A severe blow or concussion can produce it.

Congestion is a proximate cause and is induced by an obstacle to the return of the blood from the brain, as in pulmonary Emphysema, asthma and other thoracic diseases and from straining during great exertion.

I suspect that haemorrhage from this source more frequently takes place on the surface, either between or upon the meninges or within the ventricles, than in the substance of the brain, and that general rather than hemiplegic paralysis follows.
The subject of sanguineous congestion has excited considerable discussion; some maintaining that the unyielding skull renders it impossible that it should contain more blood at one time than another.

Since the publication of the explanatory remarks of Dr. Thomas Bennett upon this question, the opinion of the profession has been less divided in reference to it. The opinion that generally attains now, is that though probably the entire bulk of the whole of the contents of the cranium may undergo no change, there may be a relative alteration of the quantity of the fluid contents. A variable condition of the veins of the choroid plexus is a source of haemorrhage into the ventricles.

The frequent occurrence of haemorrhage into the corpora striata or optic thalami is attributable to the great vascularity of these segments.
Diagnosis.

It has been remarked that hemiplegia from simple softening very generally attacks people who are not of a plethora habit of body.

In my own experience, I have seen exceptions to this rule as frequently as examples.

Hemiplegia from sanguineous apoplexy often attacks people of a florid complex and habit of body.

As before mentioned, if the lesion be very extensive, the differential diagnosis between hemorhage and simple softening is very difficult.

If the case be not very severe, the diagnosis is easier.

Besides observing the general appearance of the patient, we should endeavour to discover the extent of the softening from criteria already described. When sanguineous apoplexy of a mild type takes place from softening, this latter condition is often more limited than it is, when it alone causes hemiplegia. Hematal hemorhage produces its effect suddenly, if there is pain in the head immediately before the attack, if the intellect is less affected before the attack and upon recovery from the immediate shock, also after, than in cases arising from uncomplicated softening.

Here is also more commonly profound coma lasting for a variable time, followed either by delirium or complete return of consciousness.
The shock of the effusion not unprovocably produces for a short time partial anaesthesia; sensibility however, generally soon returns. Hyperaesthesia is rare. The hemiplegia is complete from the first, though they may become more from a subsequent oozing of blood from the divided vessels. The pupil, I have observed, to be generally contracted immediately after the fit, but soon to become dilated. If the effusion be very large, the pupils are dilated soon.

If the haemorrhage had made its way into the ventricles or between the meninges, the coma would probably be persistent and there would be convulsions affecting one side more than the other.

In proportion to the extent of the central lesions, so will be the paralysing effects as explained under softening. When haemorrhage has taken place in the median lobe or affects the base of the brain, there are frequent erections of the penis and seminal discharges.

Early rigidity of the affected muscles would be a sign, being the result of the irritation of a small clot. Late rigidity is caused by the irritation of the cicatrizing process from contraction of the cyst containing the remains of the clot.
Sanguineous apoplectic is often fatal in its termination, and the hemiplegia is not the most urgent symptom which arrests the attention of the physician & friends. The haemorrhage is liable to continue a little after the shock or it may return.

From what I have before written, three classes of cases from haemorrhage into the brain will be recognised. Those originating mainly in the extreme fatty degeneration of the brain structure, independent of disorder in the circulation; those caused chiefly by the latter, acting upon diseased vessels; lastly, where there is no evident disease of the arteries, but where there is great disorder in the circulation arising from temporary or permanent conditions.

In this thesis, I am concerned with the first two varieties: their treatment is very similar to that from simple white softening, but as there is the additional complication of haemorrhage, there is more difference of opinion in reference to it.
In the first class of cases, we shall soon after the attack, find the circulation quickened, and the question will occur to us, whether it would be advisable to bleed. If we were to, the sedative effect would be but temporary, and the nutrition of the whole system suffer to a certain extent. Now, though the effect of the apoplexy at first is to quicken the pulse, the shock has subsequently a most depressing effect, and the circulation becomes affected in the precise manner, medical practitioners declare, it should be after bleeding. I believe therefore on this account it is unnecessary, and that we should confine ourselves to rest, raised posture, cold to the head, and the course recommended in treating of hemiplegia from simple softening, not forgetting a good nitrogenous diet. After much thought upon the subject, the only advantage I think can possibly accrue from bleeding in these cases would be due to the readyTen tendency of the remaining blood to coagulate, if another attack were to occur.

The second order of cases occurs in younger people generally, their immediate cause is some accidental or some permanent disturbance of the circulation, induced by sudden exertion or disease.
We must therefore pay great attention to circumstances of the body which are calculated either to stimulate the heart or to arrest the returning venous stream from the head.

Hence, if the patient be labouring under any pulmonary disorder, suitable treatment should be adopted; stocks should not be worn, nor handkerchiefs tied tightly round the neck.

All sources of excitement should be guarded against.

The bowels are to be kept regularly opened.

The diet should be strictly albuminous & stimulants avoided.

Such measures as these would reduce the chance of a recurrence of the haemorrhage to a minimum.

At the onset of paralysis from this form of disease should one bleed or not? I believe bleed, and if there be evidence of chronic venous congestion from laboured circulation through the right side of the heart or the returning stream of blood from the head be otherwise obstructed. Bleeding may benefit by relieving the distended vessels, for as I have before mentioned, though the actual contents of the cranium is constant, it does not follow that the relative quantity of the respective fluids may not vary. I think they do, and that in fact, they are...
complementary to one another, that is to say, when there is less of one, there is more of the other. For instance, if there be sanguineous congestion, the quantity of serum will diminish, probably becoming in part absorbed and in part removed to the continuation of the sub-arachnoid cavity in the spinal canal, the pia mater of which (the dura mater) is shown by the present observance of being specially adapted to receiving varying quantities of fluid. If the blood rapidly leave the brain, then the vacuity is filled up by an increased quantity of serous fluid from the perimider of the sub-arachnoid space, which there is reason to believe is secreted with a rapidity not less than that observed in the aqueous humour of the eye. In short, it may be said that the contents of the sub-arachnoid and venous spaces, is in the inverse proportion to the bulk of the other contents of the cranium. The fluid from these cavities here passes by interstices into the substance of the brain, and thus partly fills up the void.

Besides relieving the distension of the cerebral vessels, venoectomy increases the disposition of the remaining blood to coagulate, hence a clot would be more likely to form at the mouth of the ruptured vessel. It would of course also act as a depressant to the circulation.
Friction to the limbs may be adopted in this as in other kinds of hemiplegia. Electricity is inadmissible in this as in all other cases of paralysis caused by irritative central lesions, as by its use, the symptoms would be aggravated, and possibly, a return of the haemorrhage-induced. Perhaps, if long after the attack, and there were reason to think that cicatrization had taken place in the injured part of the brain and yet the palsy continue, some muscles being perhaps contracted, it might be applied to the antagonistic muscles with advantage.
The following case I copy from my diary for 1858. The patient was under the care of the late Dr. {T}t. during the time I was that gentleman's clinical clerk. Y. R. married, aged 43. About 14 years ago, he had while awake, a sudden attack of hemiplegia on the right side. His consciousness was not lost. He had severe headache for some months afterwards, but gradually recovered. In thirteen months after the attack, had nearly recovered power completely, when he had another, similar one in some particulars, but this time, he had a short preliminary warning & the left side was affected. He went on improving to the end of May '58 when he could walk, though there was still a slight deficiency of power on the left side. About the beginning of June '58, he had a third attack in which the left arm & leg were again paralyzed with early rigidity. He had since had frequent twitchings in the left arm & about 3 weeks ago after a slight fit, resembling a fainting fit, ptosis of the right eyelid took place, since when there has been a dribbling of the urine. There is constipation of the bowels, but the abdomen
is resistant, seemingly being unaffected.
The appetite & digestion were good throughout.
The has complete hemiplegia of the left side, with
plasia of the right eyelid & paralysis of the internal
rectus of the right eye. There is Considerable edema
of the lower extremities; he says he feels stuffed
at the chest. The urine is at saline. P. G. 1038.
No. 29 The pupil of the right eye, which before was sluggish,
is now almost insensible; the right however remains.
There appears now to be perfect paralysis of the superior,
iinferior & internal recti muscles. The action of the
external rectus continues perfect. Upon tickling the
left side, reflex action is replaced. The sensation of
the left side is not much affected, he cannot
however distinguish the number of fingers with which
he may be touched. The left side of the face is
paralyzed. The orbicularis palpebrarum & the
muscles of the left eye are perfect in their action.
There is a little ascites. The muscles of the
left arm are much more easily excited by galvanism
than those of the right; the difference between the
relative excitability of the muscles of the leg is not
so marked. The reflex action, as shown by galvanizing the toes,
is much greater on the left side. The reverse current produced
a more powerful effect than the direct on depallic limbs.
The question of course arose, what kind of control

The examination of the patient was sufficiently

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the blood, a highly polanized state of the nervous system is produced, thus rendering the body liable to convulsion on very slight exciting causes. Many, if not all, of those cases of so-called "cerebral apoplexy" which were at one time thought to be of such frequent occurrence, doubtless owed their origin to simple white softening. Acturally, those cases where the old morbid anatomists found no adequate sign to account for death were really caused by chronic softening of the medullary variety of the disease. I have described of the very many cases of hemiplegia recorded by Andre and others as having been induced by recent apoplexy, but little more than may be found in those of persons who have died of a different class of complaints, but whose bodies have been lying in similar postures. In chronic softening, there is more fluid within the cranium than in a healthy state of its structure in the ventricular or sub-arachnoid spaces.

Think the normal condition of the ventricles is to be distended with fluid, which passes by imbibition to the substance of the brain and indeed is as essential to the efficient performance of the functions of the latter, as is the fluid in a galvanism
tough to the free elimination of electricity, in fact
I think we are justified in considering that the two
fluids play somewhat analogous parts.

On commencing this thesis, I hoped to have been
able to review all the causes of hemiplegia, and
eit is not without a feeling of regret that I am
compelled by the magnitude of the task to confine
myself to a few varieties.

Since the time of Anandral, I know of no one
who has published a numerous list of these cases.
With an, more correct knowledge of pathology,
extended criteria for diagnosis, I think a
collection of well recorded cases would be
deemed extremely useful by the profession & excite
divest interests.

I append the particulars of a plan I adopt
for taking the specific gravity of parts of the
brain of a certain consistence.

It is a useful method, I think, for a hospital.
Solutions of Sulphate of Magnesia of various strengths are made, their S. G. being ascertained by the densimeter. Solutions of S. G. from 1010 to 1040, will be found sufficient.

These are to be preserved in bottles, and when required for use are to be poured individually into vessels. Small portions of the brain substance about the size of a pear are to be put into the vessel if it is to be observed in which solution they neither sink nor dissolve but remain suspended. This will give the S. G. required.