J. D., aged 40, became an out-patient at St. Mary's Hospital, August 20th, 1866. She had never had acute rheumatism; she had been ailing six or seven years; her face was sallow and tintless; she complained of being soon out of breath, and on exertion or excitement was seized with pain in the præcordial region and epigastrium, extending to the back, and down both arms to the fingers. It came and departed suddenly, and was accompanied by fluttering of the heart. At the same time she became giddy, and immediately afterwards had an evacuation of the bowels. The pulse was regular and frequent. On examination, the heart's impulse was found to be extensive and violent, the apex beat was to the left of the nipple line, and at this point, and to the left of it, a systolic murmur was heard, which diminished gradually in loudness towards the sternum and upwards. This poor woman was living very poorly, and was much distressed mentally by her husband's being long out of work. I was able to supply her with good food for a time from St. Mary's kitchen, and she seemed to improve; the pain and palpitation becoming less frequent and severe; the breath was not so short. On October 20th, however, after hearing nothing of her for nearly three weeks, I was called upon to sign a certificate of her death.

CASE IX.—*Constrictive Mitral Disease: Death*.—S. A. B., aged 27, married, and having two children, came under me as out-patient at St. Mary's Hospital, on October 15th, 1866. She had been ailing for eight years and under treatment for two years, complaining of cough, shortness of breath, and palpitation of the heart on walking. She had had dropsy; she had never suffered from rheumatism. These symptoms were present in an exaggerated degree; there was fluttering of the heart on the least exertion; in the night the legs felt as if going dead; she had no appetite, and was frequently sick. She looked anxious and worn, and her breathlessness was evident; the pulse was scarcely perceptible at the wrist, where only an occasional beat could be detected; the action of the heart was very irregular and intermittent; the apex beat was beyond and below its natural situation, it was readily felt; the chief impulse was at the third and fourth cartilages and below, and a peculiar vibration was felt at times; a systolic murmur was heard on the lower sternum, and along this bone to the level of the third rib, also towards the left, to within an inch and a half of the apex beat, where it was loud and distinct; and the apex, and to the left of this it was not at all audible, nor over the base of the heart or aorta; the veins of the neck were not full. She was examined in the sitting posture. I gave this poor woman food and medicines calculated to give her strength, but she vomited everything. At my urgent request she entered the hospital, and was taken in under the care of Dr. Sibson on October 26th. Nothing could be done for her, and she died on November 6th. The heart was enlarged, especially the right side, and the tricuspid orifice was very large; the mitral valve was thickened and rendered rigid by infiltration into its substance; the orifice was so contracted as only to admit one finger; there were no deposits on its surface, and it had not undergone the usual degenerative change. During life, the seat of the murmur was a subject of question, the conclusion being that it was at the tricuspid orifice. The examination after death leaves it still uncertain. Wherever the murmur had its origin, however, the change in the mitral valve was the cause of the derangement in the circulation, and of the enlargement of the right ventricle. The prognosis in this case was only too plain; the disease in the valve had evidently been progressive, though at the age of the patient it could scarcely be expected that it was atheroma; but this consideration had not much weight under the circumstances. The symptoms of existing derangement in the circulation were so serious as to claim the entire attention to the exclusion of all questions as to the nature of the cause of the derangement. Vomiting is a sign of very bad omen in the late stages of heart-disease, and is not to be met by usual remedies. Its precise cause is not very clear; whether it is from the mechanical disturbance by the labouring heart, which agitates the diaphragm and with it the stomach, or a result of congestion, or whether it is induced through the pneumogastric nerve.

The remarks given with each case will render unnecessary any prolonged remarks on them collectively. The first three show that when the history gives reason to believe the valvular disease to be stationary, and the existing dropsy or pneumonia are traceable to some unfavourable condition, or accidental circumstance, *plus* the heart-affection, recovery may be anticipated. Case V may be instructively compared with Case II. These men were attending the hospital together; and of the two, the symptoms seemed more urgent in Case II; but the history and examination of the heart furnished ground for a very different prognosis. Cases III and IV again may be compared, the actual condition of the heart, and the cause to which the attack was traceable—slight and inadequate in the one, in the other amply sufficient to account for it—being here the indications which pointed to a different result.

CASE OF "RED SOFTENING" OF THE SURFACE OF THE LEFT HEMISPHERE OF THE BRAIN: WITH SUDDEN LOSS OF SPEECH AND HEMIPLEGIA.

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THIS case presents several features of great interest in their bearing upon the physiology and pathology of the brain. The person was seen by myself only in the *post mortem* room; and I have to thank Dr. Alderson for having kindly granted me permission to publish the details of the case. It seems to me desirable to do so, principally because it throws a certain amount of light upon the question as to whether an injury of the convolutional grey matter of one hemisphere of the brain (that is to say, of a portion of the centre whence volition emanates) is adequate to produce a hemiplegia, without coexisting structural disease in or near the central ganglia of the same side. It presents other points of interest, however, as will be seen.

Frances D., aged 62, was admitted into St. Mary's Hospital, under the care of Dr. Alderson, on December 21st, 1866, with signs of renal disease. There was slight anasarca, from which she had been suffering only a short time. The urine contained some albumen; but the specific gravity was nearly normal, and no casts are reported to have been found. She remained in much the same condition for about two months, though she gradually became weaker, and suffered from occasional headaches previously to the commencement of the cerebral symptoms about to be mentioned.

March 7th. At 5.15 P.M., having previously been in her ordinary condition, and having been speaking to a fellow-patient only five minutes before, she was seized suddenly with trembling and great mental agitation. There were no convulsions, but she frequently endeavoured to look behind herself in a terrified manner. She was then found to have lost the use of the right side of the body, though there was no facial distortion or paralysis of any kind. How far sensation was lost on the paralysed side, does not seem certain. At first, she was unable to speak at all; but after two hours she was able to say "Yes," in answer to questions put to her; and this was her invariable reply, whatever the nature of the query. She seemed, however, to understand what was said, and also recognised those around her.

March 9th. When the sister in charge of the ward (who had been absent) saw her in the evening, for the first time since this attack, D. recognised her, smiled, and said "yes." On being asked by the sister what was the matter, after a pause, she said with great effort, and in a stammering way, "I can't tell," just plainly enough to be understood.

March 11th. She said, in reply to the same query, also with great effort, "I can't understand it"; but, on other occasions, almost invariably the only reply, remark, or greeting, was the word "yes," although this was accompanied by actions of a more intelligent nature. From the first she passed feces and urine involuntarily. At no time were there twitches and convulsions; and whilst the right arm and leg were paralysed, the muscles of the face were entirely unaffected.

March 14th. Breathing became very laborious. She became more unconscious—no longer appearing to recognise persons around her—and died the next day without fresh symptoms.

AUTOPSY.—The body was rather thin and emaciated; there was anasarca of the right lower extremity. *Brain*.—The subarachnoidal fluid was more abundant than natural, even considering age; the veins in the pia mater of the right side were very full and turgid, presenting a marked contrast to those of the anterior two-thirds of the surface of the left hemisphere, which were even less distended than usual. Here and there, however, on this side, there were small patches of effused blood in the pia mater, mostly over the sulci. The arteries at the base of the brain were all notably diseased; their coats being thickened, and containing large white calcified patches at intervals. On slicing the cerebral hemispheres, that on the right side was found to present nothing abnormal; whilst the left showed very many patches of *red softening*, which were almost always strictly confined to the grey matter of the convolutions. Those on the upper surface corresponded with the superficial effusions before-mentioned, though there was no trace of other patches till the brain was sliced in various directions. The principal patches of softening were met with in the third or inferior frontal convolution, together with a portion of the adjacent ascending parietal near its commencement; whilst a still larger patch was situated in that part of the superior frontal convolution, where it forms the edge, and descends into the great longitudinal fissure (about opposite the junction of the anterior third with the

posterior two-thirds of the hemisphere). On slicing the hemisphere in various directions, many smaller patches of softening were found over the mid region of the hemisphere, which occupied internal folds of the convolutions, and which therefore did not appear at the surface. Only one small patch of softening was found occupying the posterior lobe, and that was situated on an internal fold of one of the occipital convolutions. The main arterial trunks in the Sylvian fissure, although much diseased, were ascertained to be free from clots or embolic masses. The great mass of the white matter of the hemisphere appeared healthy, and of normal consistence. The central ganglia, pons, and medulla, were unaffected: they were carefully examined in all parts. The heart was large; all its cavities dilated and hypertrophied; weight, eighteen ounces; the aortic valves showed a very early atheromatous change, and a portion of the great tongue of the mitral valve was similarly affected; the walls of the left ventricle were preternaturally pale (as though from fatty degeneration), more particularly the inner layers next the endocardium. All the cavities of the heart were much distended with black semi-coagulated blood. The left lung was healthy. The right lung showed evidences of recent pleurisy about the base, outside a portion of the organ, which presented a black and almost gangrenous aspect. When this portion of the lung was cut into, it presented a very large patch of what is called circumscribed pulmonary apoplexy. Seven or eight other patches—smaller, though still rather large—were found in other parts of the lung, and principally in the lower lobe. The lung tissue intervening between these nodules was much congested and somewhat softened. The right lung weighed twenty-five ounces; and the left weighed sixteen ounces. The kidneys were of medium size, and much congested; showing on section slight wasting of the cortical substance, and on their surface a highly irregular and granular condition, of pretty uniform tint, after the capsules (which were unduly adherent) had been stripped off.

COMMENTARY.—A careful examination of the patches of softening in the recent condition, and also after hardening in spirit and chromic acid, showed them to be almost entirely limited to the grey matter. They were, in fact, hæmorrhagic effusions into the grey matter, produced apparently by the rupture of many minute vessels in each area of disease, followed by degenerative changes. The patches corresponded with what Cruveilhier called "taches scorbutiques", and also in every way with those experimentally produced in dogs by MM. Prevost and Cotard after the injection of fine foreign particles into the carotid arteries, and the subsequent blocking up of some of the minute cerebral vessels. In this case, many of the vessels in the patches of softening were blocked up, although the precise nature of the occluding material was not satisfactorily ascertained. Certainly, in some of the vessels there was something more than mere thrombosis, and apparently an impaction of a granular material. The minute vessels in the surrounding grey matter were very much diseased, the smaller arteries and even capillaries being in an atheromatous condition; whilst outside the vessels, in the lymphatic space, there was a considerable quantity of reddish-brown and orange-coloured blood-pigment, partly free and granular, partly granular and enclosed within large cells, so as to constitute "granulation-corpuses", with pigmentary instead of fatty contents. When examined in the recent condition, portions of the softened tissue showed innumerable blood-corpuses, mixed up with isolated, dark, granular ganglion-cells, and a multitude of dark coloured granulation-corpuses, of all sizes and in different stages of development. Sections of the hardened convolution showed red blood-corpuses infiltrated into the midst of the grey matter, replacing it in some parts, and isolating the nerve-cells in others. In the midst of this tissue, with the disintegrating effusion of blood, there were seen transverse and longitudinal sections of vessels, whose surrounding lymphatic sheaths were enormously distended by blood-corpuses.* Whilst the granulation-corpuses were most abundant in the superficial strata of the convolutions, the effusions of blood were most marked in the middle and lower strata.

With regard to the mechanism of this hæmorrhagic softening, its apparently almost instantaneous production of symptoms, the limitation of the lesions to one hemisphere, and the presence of well-marked and wide-spread arterial degeneration coincident with Bright's disease, point to the probability of embolic fragments having been swept from some portion of the walls of the larger vessels into the left anterior and middle cerebral arteries. These fragments, being composed of debris only, did not occlude the main trunks, but were enabled to pass into some of the smaller vessels which ramify in the substance of the convolutional grey matter. This disturbance of the circulation, and the consequent increased tension in the collateral branches, whose walls were

already so much weakened by disease, caused many of them to give way, and so produced effusions of blood into the lymphatic sheaths, and also out of these into the midst of the grey matter. Then followed the degeneration of nerve-matter, and the production of granulation-corpuses, such as are met with in softening of the brain.

Although no focus of atheroma, from which minute particles may have been swept into the cerebral arteries, was actually found, still this seems to me the most feasible explanation. Of course, with cerebral vessels so diseased, ruptures might have been produced under the influence of exertion, leading to increased vascular tension within the brain. But in this case there was no history of exertion of any kind: the woman was lying quietly in bed. The number of the patches of hæmorrhagic effusion, and their almost complete limitation to the anterior and middle portions of one hemisphere, also make it more probable that they were produced in the manner first suggested. Death was undoubtedly brought about more immediately by the supervention of extensive pulmonary apoplexy in the right lung on the day preceding the fatal termination. Here, also, disease of vessels doubtless had much to do with their rupture and the effusion of blood, since there was no obstruction at the mitral orifice.

In considering the symptoms, the first thing we have to notice was the sudden occurrence of loss of speech in connexion with right hemiplegia. Here the left was the hemisphere involved; and, *post mortem*, a large patch of disease was found in the third frontal convolution—the place indicated by M. Broca as the seat of articulate language. But the evidence is not unquestionable as to the connexion of the loss of speech with this particular lesion, since a very large patch of disease also existed in the upper part of the first frontal convolution, and several other smaller ones were found in the parietal and other convolutions of the middle lobe. These were the situations of the disseminated lesions of the grey matter; and to what extent the loss of speech was due to the particular lesion of the third frontal convolution, rather than to the whole of the morbid changes taken together, we are unable to say from the evidence afforded by this particular case. It is well to note, however, that the loss of the power of speech was not total and accompanied by a profound impairment of the intellectual faculties; neither was it due to the mere loss of the power of articulation. The patient was able to say "yes" distinctly; and on two occasions she contrived, after great mental effort, to utter short sentences. Then it is expressly stated in the report that she knew those around her, and that her manner exhibited a certain amount of intelligence. Here, then, lesions of the convolutional grey matter produced that particular kind of loss of speech which seems more identified with an abolition of verbal memory, than with loss of the power of articulation. Between this kind and that amount of loss of power which has been described as a loss of "the faculty of language", doubtless all intermediate grades may be met with. The one is but an extreme form of the other defect; though why, in the last case, any mention should be made of a hypothetical faculty of language, I am at a loss to understand, since the inability to speak is only one species amongst other kinds of evidence testifying to such an amount of mental impairment that the patient is unable to perform even the most simple and ordinary kind of intellectual operation.

The next notable symptom was the hemiplegia; and, to account for this, the autopsy revealed lesions of the convolutional grey matter of the opposite hemisphere only, but no morbid change whatever in the pons, in the central ganglia, or in the white substance in their immediate neighbourhood. The occurrence of this change in the surface grey matter seems, then, to have led either directly or indirectly to the paralysis of the opposite side of the body, which continued, with little or no alteration in intensity, during the remaining period (one week) of the patient's life. Only three explanations of such an occurrence seem to present themselves: either (1), with Brown-Séquard, the paralysis may be considered as of a reflex character, and therefore only indirectly dependent upon the superficial injury; or (2) we may adopt the more specific, though somewhat similar kind of explanation, which has been advanced by Dr. Hughlings Jackson, to the effect that the irritation in one portion of the vascular territory of the middle cerebral artery, reacting upon its sympathetic fibres, has produced a spasm and contraction of all the branches of this artery, including those which supply the corpus striatum, and that this diminution in the supply of blood is sufficient to bring about such an impairment of function in the anterior central ganglion as to lead to the occurrence of hemiplegia; whilst, lastly (3), there is the suggestion of Dr. Wilks, that hemiplegia may be produced by an extensive lesion of the grey matter of the convolutions of the opposite hemisphere, the mechanism of its production being direct, and immediately dependent upon a destruction or morbid change in the convolutional centre, which formerly presided over the movements of the opposite half of the body. In this case, it is true, the paralysis would belong to a different category from those which are more frequently met

* This condition has been improperly called "dissecting aneurism of the cerebral arteries"; but the blood is not effused between the coats of the vessels; their walls are completely ruptured, and the blood-corpuses are contained between the cerebral artery and its enveloping, though greatly distended, lymphatic sheath.

with; it would neither depend upon degeneration of the muscles, upon disease of the nerves going to the paralysed parts, nor upon disease of the nerve-centres in which the structural arrangements for bringing about the necessary coordinations exist; so that all the anatomical essentials for the production of muscular movements are present, if the volitional stimulus be forthcoming. But, in these cases, this would be the very thing wanting. The centre whence the volitional stimulus emanates, which calls into action the muscles of the opposite half of the body, being extensively diseased and more or less destroyed functionally, no such stimulus is produced; and, Will being absent, the nervous and muscular apparatus pertaining to the voluntary movements of the opposite half of the body are thereby rendered functionally inert and useless, solely because their appropriate stimulus is not forthcoming.

The solution of a question of this kind is a matter of the utmost difficulty, and it seems wise not to be too hasty or exclusive in one's views on the subject until more data have been accumulated. Whilst admitting the value of the reasoning, based upon the most careful clinical observation, advanced by Dr. Hughlings Jackson in support of his theory, it seems to lack that anatomical and physiological warrant which would be desirable. It is perfectly fair to admit, however, that the clinical facts may be truly explained in this way, although anatomy and physiology have not yet advanced so far in this direction. Still, in our present state of knowledge, we may be allowed to ask whether the different arterial regions are presided over by independent sympathetic nerve-centres, with which are connected afferent and efferent fibres having a similar distribution, so that an irritation of these nerves in any part of a vascular area may be conducted centrally to a vascular ganglion, and thence reflected so as to produce spasm and contraction in all the branches of this particular artery. This being true, we have still to consider whether the spasm so produced could be so intense as to cause an almost complete negation of function in the part—not for a few minutes only, but for days and even one week—and yet at the same time that the vascular supply should be adequate to preserve the nutrition of the part, and obviate all tendency to *ramollissement*. All these things must be admitted, if Dr. Jackson's explanation be a valid one. But, whilst alluding to apparent difficulties which stand in the way of our accepting it, I must not omit to call attention to the fact that, in the *post mortem* record, it is distinctly stated that the veins in the pia mater over the right hemisphere were "very full and turgid, presenting a marked contrast to those of the anterior two-thirds of the surface of the left hemisphere, which were even less distended than usual." At the time this observation was made, I did not know of Dr. Jackson's theory, and unfortunately no special observation was made with regard to the relative vascularity of the two corpora striata. This marked difference in the amount of vascularity of the surface in the two sides, though in accordance with Dr. Jackson's theory, does not seem to be otherwise easily explicable.

The position assumed by Dr. Wilks seems to be theoretically unsailable; and, moreover, in his admirable paper in the *Guy's Hospital Reports* for 1866, he records a case which seems to bear out his views. Here almost the whole of the surface grey matter of the right hemisphere was most extensively diseased; there was a considerable diminution of intelligence, and also the gradual supervention of paralysis on the left side of the body. In this case it is said "the most remarkable symptom was the fact that she never seemed aware that she had lost the power of the left side of the body. On being asked about her arm, she always declared she could move it, although it lay helpless by her side." But in the case before us the question seems to be, whether the amount of convolitional grey matter involved by the disease in the right hemisphere, was sufficient to account for such a diminution of volitional power, as would practically leave the right side of the body paralysed. Doubtless other cases have been recorded in which injuries of surface grey matter have been met with quite as extensive, and yet which have been unaccompanied by hemiplegia. But the mode and rapidity of production of the morbid change must always be taken into account, as well as its extent. It seems possible that chronic changes, as extensive as those met with in the present case, might gradually supervene, and yet produce no paralysis; whilst, if the lesions were suddenly, and, as it were, violently, produced all at the same time, it is quite conceivable that the result might be different. A violent and sudden interference with such subtle functions as are presumed to be carried on in the surface grey matter, may not only annul the functions of the part injured, but may also extend a perturbing or inhibitory influence over the remainder of the grey matter of this hemisphere. Thus, there would be a supplementary reflex influence, such as Brown-Séquard considers adequate for the production of the most definite and varied combinations of cerebral symptoms.

It is interesting to call attention to the fact that, although, in this case, there was a sudden and extensive injury to the surface of the brain,

no convulsions of any kind were produced. In other cases of disease at the surface of the cerebrum, convulsions are frequently met with; and Dr. Wilks strongly insists upon the fact that in many instances of cerebral disease, accompanied by epileptiform convulsions, some morbid change is met with in this region. Also, it is interesting to note that the onset of a sudden destructive change in the convolitional grey matter, not only gave rise to no motor phenomena, and seemed unaccompanied by pain, whilst it did produce marked mental phenomena which revealed themselves by unmistakable symptoms of terror, agitation, and bewilderment. This is as might have been imagined, so far as the absence of sensation is concerned, and is fully in accordance with the results of experiments, which have shown that the substance of the cerebral hemispheres may be cut or handled without the production either of sensation or of movement.

It is reasonable to expect, also, that irritation of a part concerned in the carrying on of intellectual operations, should lead to mental perturbations of some kind, just as an irritation of the optic nerve, for instance, leads to subjective sensations of flashes of light.

It is quite certain, moreover, that, in this case, there was, from the first, not the slightest trace of that partial paralysis of the facial and hypoglossal nerves, which almost invariably exists when hemiplegia is produced by a lesion in the central ganglia. Dr. Alderson told me that this particularly struck him during the life of the patient, and that he called the students' attention to this fact. Of course, if the paralysis in this case had been produced after the manner indicated by Dr. Hughlings Jackson, then the symptoms, if due to an interference with the functions of the corpus striatum, should have resembled those ordinarily produced by a hæmorrhage into or softening of this body. But the absence of facial paralysis may have been exceptional in this case, even though the paralysis were due to the lesions in the convolutions; and such paralysis may perhaps occur in other cases where the superficial lesions are more extensive. Still the absence of such paralysis in this case must not be lost sight of. Unfortunately, no definite observations were made as to the condition of sensation in the paralysed extremities, or as to their temperature. Physiological data would lead one to expect that in a paralysis so produced, sensation would be unimpaired, and the temperature of the paralysed parts unaltered instead of raised.

In cases, therefore, where hemiplegia exists from the first without any paralysis of the facial or of the ninth nerve—more especially where the onset of the symptoms has been sudden, and attended with some evidence of mental agitation—where perhaps sensation is unimpaired in the paralysed limbs, and their temperature is normal, may we not suspect the existence of extensive disease of the grey matter of the convolutions of the opposite half of the cerebrum?

A CASE OF FRACTURE OF THE TIBIA AND FIBULA IN A PATIENT AGED 88.

By JOHN GRANTHAM, F.R.C.S. (Hon.), Crayford, Kent.

In relating the following case of fracture of the tibia and fibula, I am prompted to do so by the fact that tubular bones can unite at such an advanced age as 88 years. If my memory serve me rightly, I think it is the only case of fractured tibia on record as having been completely united so as to support the body without assistance at that age. Mr. Erichsen says, in his work on *Surgery*, that he has witnessed two cases of fracture of the femur in patients upwards of 90 years of age, but does not state the results: only, that the bones united.

My case occurred to a lady, of short stature, very temperate in her habits throughout life, an early riser. She had slipped off a step that had just been washed, causing a transverse fracture just above the ankle-joint, representing the appearance of a dislocation of the ankle-joint backwards, but which, on extension being made, proved to be a fracture of the tibia and fibula. Knowing how much is due in effecting the union of a fractured bone by maintaining tranquillity of the general system so as not to depress the law of life, I resolved in the treatment to leave no principle that could produce ease and quietude unperformed; I applied, therefore, Dr. Neil Arnott's method of making equal pressure (as reported in my work *Facts and Observations in Medicine and Surgery*, page 2), by means of an India-rubber case of the shape of a box, of the length of the leg, without ends or top, supplied with air from the upper end into three compartments; the same secured on the limb with Cline's splints; the whole suspended on one of my suspension apparatus from the top of the bed, the four ropes converging to one point, not being checked by any other machinery, thereby giving full effect to the simple action of the "ball and socket joints".

The accident occurred on Jan. 26th, 1867. The patient is at this time in perfect health, taking carriage exercise and walking without aid.