mitted through the latter. I heard, however, a remarkable exception to this rule, through the kindness of Dr. Dickinson, who pointed out the case to me as an interesting one. I examined it carefully with Mr. Collins, who has on this and on other occasions kindly joined me in examining cases at St. George's Hospital, and we found that the doubling was markedly audible at and as far to the left of the septum as the sounds of the heart could be heard; that is, out to the apex; but that the double sound was soon lost when we travelled to the right of the septum over the right ventricle. Here, the first sound of the right ventricle was readily transmitted through the left ventricle, the sound of which was, however, speedily damped by its fellow. I could not explain the occurrence of this exceptional sign; for the left ventricle was large, its apex being felt an inch to the left of the nipple-line; and there was no other sign than this that the right ventricle was large. It may, however, have been so.

When the doubling of the first sound is diffused over the right ventricle, the first of the two first sounds is the louder over and to the left of the region of the sternum and ensiform cartilage, and the second of the two sounds is the louder over and just to the left of the septum, thus demonstrating that the second of the two first sounds is that made by the ending of the systole of the left ventricle.

In those cases in which the doubling of the first sound is well marked over the septum, and is almost limited to that region, if one end of the differential double stethoscope be applied over the lower end of the sternum, while its other end is applied over the apex, at both which points the first sound is originally single, the doubling of the first sound is perfectly audible. It is lost if one end be removed; it is regained if the end be replaced. I have noted this again and again, in concert with some of the best observers in London, including Dr. Donkin, Dr. Greenfield, Dr. Coupland, Mr. Gould, Dr. Humphrey, and we all heard the sign, and we all agreed in every instance that the first of the two sounds was that heard by us at the lower end of the sternum over the right ventricle, and the second of them was that heard at the apex or over the left ventricle.

Doubling of the first sound is audible in all the forms of Bright's disease, whether acute or chronic, with fatty or granular kidney, and it may be, and often is, absent in all the forms of the disease. It is more frequently, I think, present than absent, but it is more often difficult than easy to discover, and it for this reason fails to decipher for us easily and at first sight many cases that it would be easy to read if this sign were readily available.

As a rule, the second sound is single, and the first sound is double, in all cases of chronic or established Bright's disease; but, in acute Bright's disease, we may, and often do, have a double second sound over the arteries, and especially the conus arteriosus, and a double first sound over the septum of the ventricles. In one or two cases, there was a restricted meeting-point over the upper part of the septum, close to the conus arteriosus, at which the two signs meet each other at the borders of their respective areas, and then we had both a double first and a double second sound heard simultaneously. Dr. Donkin was the first to hear this conjunction of those signs.

A SOLVENT FOR QUININE.—The spiritus ætheris dulcis as a solvent for quinine is recommended by Dr. Isaac Smith in the *New York Medical Journal*. He says that one ounce of the solvent will dissolve two drachms of quinine, giving a transparent solution.

USE OF GELSEMINUM IN DILATATION OF THE CERVIX UTERI.—Dr. J. A. Agnew reports to the *Virginia Medical Monthly* three cases in which he employed gelseminum as an adjuvant to mechanical means for dilatation of the cervix uteri. The first patient had retroflexion of the uterus. The cervix was small and conical; the os was at the apex of the cone, and was so nearly closed that he could not introduce the smallest laminaria tent. He had two conical bougies made, the little end of the smaller bougie being not larger than a knitting-needle; even this could not be introduced. The influence of gelseminum over sphincteric action occurred to him, and he determined at once to try it. He gave ten drops of the fluid extract of gelseminum every ten minutes until thirty drops had been taken. Immediately after the last dose, without the slightest difficulty, he passed successively the bougies and a No. 1 sponge-tent up to the point of flexion. On the fourth day thereafter, he failed in every effort to introduce a No. 2 sponge-tent until the fluid extract of gelseminum had been given as before. After this was given, the tent was readily passed up to the point of flexion; and, to his great satisfaction, after a little manipulation, it passed this point, and there was no further trouble in the operation of dilatation. He has tried gelseminum in two other cases, with equal success (*Boston Medical and Surgical Journal*).

REMARKS

ON

THE PATHOLOGY OF CHOREA.

By H. CHARLTON BASTIAN, M.A., M.D., F.R.S.,

Professor of Pathological Anatomy in University College, London; Physician to University College Hospital, and to the National Hospital for the Paralysed and Epileptic; etc.

THE JOURNAL of December 23rd contains an interesting communication by Dr. Hughlings Jackson, entitled "Note on the Embolic Theory of Chorea"; in which, towards the close, after discussing some of the evidence adverse to this theory recently adduced by Dr. Dickinson, he adds, "It may be, I would admit here, that the hypothesis of embolism will be displaced by Bastian's hypothesis of thrombosis as an explanation of many cases of chorea". As the problems involved in the etiology of chorea are of considerable importance, and as I have only made a few casual remarks in my work *On Paralysis from Brain-Disease* (p. 29), on the part played by thrombosis in the production of chorea,* it may not perhaps be out of place for me to state more fully the grounds of my opinion, and refer to some other points in the pathology of chorea, principally in reference to the very interesting and important communication already referred to by Dr. Dickinson, in the last volume of the *Medico-Chirurgical Transactions*. I am the more desirous of doing this because, in a communication to the *Transactions to the Pathological Society for 1869*, on the plugging of minute vessels of the brain by aggregations of white corpuscles, I made a remark which has since led others to regard me as a supporter of the embolic theory. I said (p. 16), "I hope shortly to publish the details of a fatal case of chorea in which such embolisms led to ruptures and obliterations of small vessels throughout the corpus striata, and in the course of the middle cerebral arteries generally, this being a case of bilateral chorea in which delirium was also present". The details of this case have not yet been published, partly because my examination, shortly afterwards, of the brain in two other fatal cases of chorea, in which vascular occlusions were also present, though to a less extent, caused me to reject the notion that they had been occasioned by embolism, and to favour that of their production by thrombosis; and partly, because further reflection convinced me of the need of obtaining some knowledge of the microscopical characteristics of the blood in patients suffering from chorea, as well as other kinds of evidence, before any coherent account could be given of the pathology of this affection.

This additional information was in part acquired when other work absorbed my attention, and the question of the pathology of chorea has not again been made by me the subject of special investigation, though I have always felt a keen interest in the problem. Meanwhile, on the strength of the passage above quoted, I have been quite legitimately cited in one of the text-books on Medicine at least, as an upholder of the "embolic theory" as applied to chorea, though as a matter of fact this is a view which I have long since rejected.

In the following remarks, I propose to make no formal communication, and to refer to some of the many points involved in recent discussions concerning chorea, with as much brevity as may be compatible with a clear exposition of the views which I myself hold as to the pathology of this disease; and therefore shall allude to the many important communications on this subject only so far as may be absolutely needful. I am fully impressed with the value of many communications, though a discussion of their doctrines here and now would be impossible.

Region of Nervous System affected in Chorea.

In common with Dr. Russell Reynolds, Dr. Jackson, Dr. Broadbent, and others, I regard chorea as a cerebral rather than as a spinal affection; and, believe also, that the corpora striata, either alone or with the thalami, are the principal seats of the lesions or disordered actions related to the well known motor phenomena of this disease. In certain cases, however, other nervous complications may arise, and then the area of pathological change may be correspondingly widened, as in the case referred to by me in the *Transactions of the Pathological Society*, in which delirium supervened upon the chorea, and in which, after death, occlusions of minute vessels were also found in the grey matter of the convolutions, and also in a somewhat similar case which has been recorded at length by Dr. Tuckwell. Other occasional complications of the disease need not now be specially referred to.

* Though I have more than once conversed on the subject with my colleague, Dr. Jackson.

My case above alluded to was, I think, the first in which definite multiple lesions were discovered in the corpora striata, after Dr. Reynolds had indicated these bodies as the probable seats of disordered action. And owing to the fact subsequently pointed out by Dr. Jackson, and reinforced by Dr. Broadbent and Dr. Russell, that the distribution of the spasms accords almost exactly with that of paralysis in cases of hemiplegia due to lesions in the corpus striatum or some other part of the territory of one of the middle cerebral arteries, it has of late been very commonly regarded as almost settled, that the seat of the principal disordered actions or lesions in chorea must be in this same arterial territory, either on one or on both sides of the brain.

The truth of this conclusion may seem to some to have been rendered somewhat doubtful, at least since the recently published researches of Dr. Dickinson, owing to his having found lesions widely distributed, not only within the brain, though principally in the sites above mentioned, but also in various parts of the spinal cord. I have, therefore, within the last few days, again carefully studied Dr. Dickinson's recorded cases, in order to see what amount of evidence they seem to furnish against this conclusion, or in favour of a conjoint spinal seat of lesion.

It seems scarcely necessary to say, that the publication of Dr. Dickinson's pathological observations cannot affect the validity of the reasons originally adduced by others for regarding the symptoms of chorea as essentially a cerebral rather than a spinal malady; such reasons being especially strong in relation to hemichorea. This latter evidence, indeed, seems to me quite incapable of being shaken; and, looking to the close relations existing between hemichorea and ordinary bilateral chorea, we could not suppose that the essential seat of the lesions or disordered actions was in the one case cerebral, and in the other to any large extent spinal. Dr. Dickinson's seven recorded cases are instances of ordinary bilateral chorea, and, as in all of them he found more or less distinct pathological changes in the spinal cord, it remains to ascertain in what light these lesions are to be regarded in reference to the sum total of symptoms presented in such cases of chorea. Unfortunately, the clinical details of Dr. Dickinson's cases are recorded so very briefly as not to throw so much light as they might have done upon this part of the problem.

In Case I, chorea had only lasted two days: Death occurred apparently from the effects of some intercurrent pathological accident. There was congestion of almost the whole of the brain and spinal cord, but a similar condition existed in "the liver, spleen, and kidneys". The remarkable size of the central canal of the cord must have been independent of the disease, and can only be regarded as a developmental peculiarity.

In Case II, the only spinal lesion which could by any possibility be regarded as distinctive or pertaining to chorea, would be the perivascular changes "found in the lumbar region, where dilated arteries traversed the grey horns separated from their substance by a wide interval filled with the globular translucent matter which so often marks the contact of nervous tissue with dilated vessels". In the brain, the congestions seem to have been more marked, since actual though small hæmorrhages had taken place in and beneath the pia mater in some places. But the parts of the brain especially referred to as being notable for the impletion of its vessels, and the pathological consequences of this, are the *corpora striata*.

In Case III, the patient died on the twenty-fourth day, with signs of prostration. "She had had but little sleep, and the movements had been violent and general." The brain was remarkable for the wide-spread extent of its congestion, which was associated even with capillary injection in some parts. "The capillary injection was most marked, though by no means confined to the *optic thalami*." Dr. Dickinson adds, "the cord was congested, but to a less degree"; in addition, however, there were actual structural changes in the grey matter, both in the dorsal and in the lumbar regions, which appeared to have been occasioned by antecedent extravasations of blood in these sites.

In Case IV, there were "violent chronic movements of the neck, trunk, arm, and legs.....the face was little affected, but swallowing was difficult, and speech hesitating". The patient subsequently became prostrate, and "lapsed into the condition to which the term typhoid is applied". The necropsy was made under difficulties, so that only "portions of the brain" were examined, together with the spinal cord, and no mention is made as to the state of other organs. The only part of the brain referred to as having been examined are the corpora striata, and these are said to have been notably hyperæmic, though no other structural changes were detected. A similarly general hyperæmic condition existed in the medulla and throughout the cord. That is, each part of the body specially alluded to as having been examined was found to be almost equally hyperæmic—an important fact to bear in mind when we consider that the patient died in

a "typhoid condition"—a state which is generally characterised *post mortem* by wide-spread congestions of organs. There was in addition, in this case, an extravasation of blood into the grey matter of the left posterior horn in the cervical region.

In regard to Case V, Dr. Dickinson's summary is thus given (p. 14): "Sixty-four days. Venous injection of the brain, especially of the corpora striata, wherein were also periarterial exudations. Arteries in the convolutions near Sylvian fissure surrounded by blood-crystals and *débris*. Injection and scattered erosions of the cord. 'Sclerosis' of the grey matter in both the dorsal and cervical regions, placed with bilateral symmetry." In this case, the chorea followed upon an attack of acute rheumatism which had existed for the previous three weeks. The choreic movements had ceased for about two weeks before the death of this patient from heart-disease; and it is worthy of note that the "brain and cord were natural to the naked eye"; that is, the hyperæmic conditions usually associated with chorea during its course and at its close had had time to disappear, leaving their microscopical effects only. The *corpora striata* are noted as being "more minutely injected than the rest of the brain".

In Case VI, there had been two previous attacks of chorea. The changes found "were of two kinds—recent injection and its consequences belonging to the last attack, and ancient changes due probably to congestive processes associated with one of the earlier". The recent changes were most marked in "the bodies at the floor of the lateral ventricles and in the cord", whilst the older changes were confined to the brain in the territory of the middle cerebral arteries. These are described as consisting of "periarterial degenerations and scattered spots of 'sclerosis' in the *substantia perforata* and convolution at the beginning of the left Sylvian fissure". The changes in the cord, beyond fulness of its vessels, only consisted of erosion about the bottom of the anterior fissure, which is common as a result of congestion in many conditions, according to Dr. Dickinson.

Case VII was a chronic and severe case, thus summarised: "Four years. Spots of 'sclerosis' numerous set in the *substantia perforata* and grey matter of corpora striata, symmetrically placed with regard to the two sides. In cord, large exudations into grey matter, and fissures chiefly in cervical region."

Cases I and IV can scarcely be said to throw much light upon the question as to the seat of lesion; whilst, of the remaining five cases, the only point common to all is, that the maximum amount of pathological changes, so far as the brain was concerned, was found in the corpus striatum or some other part of the territory of the middle cerebral artery. The change in the spinal cord presented, however, no such constancy as to its seat of maximum intensity. In some, it consisted of mere general congestion; in others, there were, in addition, local accidents which may have resulted from this state, situated now in one region and now in another of the cord. It seems to me more likely that these lesions were consequences of mechanical congestion, induced, perhaps, by the more severe of the choreic phenomena themselves; or else, as in Case VII, that they were due to irritation (followed by determination or congestion) brought about secondarily from the prolonged over-activity of some portion of the cord in response to the constantly recurring stimuli emanating from the cerebral seats of disorder during a prolonged period.

We must certainly strive to distinguish the secondary from the primary morbid states of the nerve-centres in such affections as chorea; and, in order to enable us to do this with more certainty, we much need certain data (which, I have good reason to believe are now being sought for by a very competent observer) as to the effects which may be produced upon the cord by congestion or general bodily states in cases in which no appreciable symptoms referable to the spinal cord have existed during life. It seems to me, however, that the opinion of those who believe that the seat of disordered action or lesion productive of the phenomena of chorea is situated in the corpora striata and thalami, or, at all events, in some part of the territory of the middle cerebral artery, need not be appreciably shaken by the facts recently recorded by Dr. Dickinson in the able communication to which I have been referring.

My own experience as to the local lesions of chorea is imperfect and fragmentary, but, so far as it goes, it tends in the same direction, and in some respects supplements that of Dr. Dickinson. The case referred to in the *Pathological Transactions* was that of a girl who died in University College Hospital, under the care of Dr. Wilson Fox; and in the following year, 1869, I also received through the late Alexander Bruce parts of the brain from a fatal case of chorea which had been under the care of Dr. Basham in Westminster Hospital; and shortly afterwards I examined the brain in another case of chorea which had been under the care of Sir William Jenner at University College Hospital. I do not propose now to enter into any details, but

merely to say that in each of these cases I found the ganglia at the base of the brain, and more especially the corpora striata, notably hyperæmic; and, proceeding by a method different from that of Dr. Dickinson, I also found occlusions, though not to the same extent in each of them, in the small vessels of these parts; the occlusions being partly fibrinous plugs, and partly composed of rounded concretions, apparently of an albuminoid nature, such as are figured in the right-hand part of the illustration on page 11 of the *Transactions of the Pathological Society* for 1869. In Dr. Wilson Fox's case, where delirium supervened, there was also a minute capillary injection of many parts of the surface of the brain; and in these regions I also found numerous occlusions of minute vessels.

My observations were made in the latter case by separating the convolutions whilst the brain was in the fresh state, cutting with a sharp-pointed scissors through and all round a portion of the pia mater adhering to one side, and removing this, with as little traction or disturbance as possible, to a microscope-slip, where it was immersed in a quarter per cent. solution of bichromate of potash. The portion of pia mater was put into this thin fluid for preliminary examination, in order to prevent shrivelling; and afterwards, if it were to be preserved, glycerine was allowed to run in and replace the bichromate solution without disturbing the specimen. In examining the corpora striata and other internal parts of the brain, after section of the part, a distended vessel was dissected out by means of a needle, whilst its surrounding branches were cut across with a sharp-pointed scissors whilst they were still imbedded in the brain-substance. The vessel and its branches were then removed as before, with as little disturbance as possible, to a weak bichromate solution. In this way, the contents of the vessels and their branches could be fully examined, and I was able to detect the vascular occlusions which I believe to be the principal cause of the minute injections of the parts, but which Dr. Dickinson seems not to have found in his examination of sections of the hardened organs. In future observations, the two methods should be combined; that is, with different parts of the same brain. The examination of the vessels by removing them from the recent brain is good as a preliminary measure best calculated to reveal the fact of the existence and the nature of occlusions; but, in order to study the effects produced by them, we must proceed, as Dr. Dickinson has done, to harden the nervous organs and subsequently examine sections.

[To be continued.]

THE EMBOLIC THEORY OF CHOREA.

By THOMAS STRETCH DOWSE, M.D., F.R.C.P.E.,

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MY experience in that acute angle of the plane of nervous diseases called chorea has not been so extensive as in the remaining superficialities. Yet I think I have seen sufficient of the etiology, clinical features, and pathology of this disease and its kindred congeners to venture a few remarks on this very important question.

I admit, on commencing, that, of the four cases where I have had an opportunity to examine the cerebral vessels, no embola were detected, but thrombi of the vessels of the grey matter of the convolutions of the postparietal and occipital lobes were abundant, as well as perivascular spaces and degenerated nerve-cells. Of this I am sure, that, so long as we constantly write and talk of embolism of cerebral vessels to the exclusion of thrombosis, so long shall we be in error and fall short of a most important and unvarying phenomenon.

It is to the vascular system and the stability and integrity of the fluids in circulation through the body, that we have to look for the induction of primary changes in the nerves and nervous centres. It is the circulation of the blood-governing, as compared with the due formation of the blood-governed, which we have to take into consideration; and, moreover, it is to the due correlation of these that we have to look for a healthy standard. I ask the questions: Is an atheromatous vessel due to blood-change, or to defect in the governing power which regulates its nutrition? Is a cerebral glioma or fibroma of the dura mater due to blood-change or to defect in the governing power which regulates the due nutrition of those parts? Is an ovarian growth or uterine cancer due to primary blood-change or to defect in the governing power which regulates nutrition?

The probable answer will be, that it is sometimes the one and sometimes the other. I cannot meet the question without committing myself to mere conjecture, which I should be very sorry to do. Still, it would be a not uncommon error, in these metaphysical days of advanced pathological research, did I venture to state that, in the habit of body or constitution known as the choreic, we have a family history, a dia-

thesis, a blood-condition, a nervous state; in fact, a type where the governing power, the blood, is easily aborted and becomes, in great measure, governed by nervous agency rather than being itself the governing agent, and it is thus that disease commences.

It might be chorea, pericarditis, valvular or atheromatous changes, acute rheumatism, epilepsy, mental derangement, the various forms of skin-disease, mere albuminuria, or desquamative nephritis, age and circumstances modifying results as influenced by hereditary tendency.

I do not think it would be an easy task to say whether the blood or the nervous system was the factor of thrombosis in the following cases which I find in my note-book.

A woman, aged 50, was sitting at tea in her usual health during a hot sultry summer afternoon; she was suddenly startled by a loud peal of thunder, and immediately she was seized with intense pain at the uppermost part of the thigh. When she came under my care, the limb was of immense size, and a plugging of the iliac and femoral veins was diagnosed and verified by *post mortem* examination.

A woman, aged 36, was standing on shore and saw her son drowned in a gale at sea. She suffered from temporary mental aberration, which was succeeded by general incoördinate muscular movements of the face and extremities, with opisthrothotonos. Here, we had multiple thrombosis of the grey matter of the brain and spinal cord. She recovered with some ataxy of the lower limbs.

A man had been suffering from repeated attacks of erysipelas, but had recovered. There was ankylosis of the joint of the index-finger, which was amputated. In less than three hours, his temperature rose to 104 deg., the respirations to 60, and he died from what is called shock within twenty-four hours of the removal of the finger. At the *post mortem* examination, the heart was found to be empty, but the vessels of the brain and spinal cord were plugged with thrombi.

I have cited these cases, as I could many others, to show (but not to prove) that, in certain conditions of the blood and nervous system, thrombosis is not so uncommon. We hear a great deal of vascular spasm in the various forms of epilepsy, true enough; but I believe ramuscular venous thrombosis has been overlooked. I maintain that no one can give a rational explanation as to the pathology of chorea who does not first make himself acquainted with the causation of the many forms of involuntary, incoördinate movements which are to be found in well known organic changes of the nervous system. Let it be granted that, in chorea, we have something unique: movements, in fact, which can scarcely be called tonic or clonic, whose diverse action partakes of flexion and extension of parts supplied by the same nerves at the same moment of time without rhythm, uniformity, or regularity, with or without loss of sensation, but, unless very profound, are certainly, in some measure, under mental influence.

In Dr. Hughlings Jackson's able defence of the embolic theory of chorea, he states: "All movements must depend on nervous discharge. Disintegrated nerve-tissue cannot discharge; for really the disintegrated matter has ceased to be nerve-tissue. I suppose the excessive movements occurring either in chorea, or epilepsy, or epileptiform seizures, are produced by discharge of grey matter, which, except for great instability from overnutrition (not better nutrition) is healthy."

One can but be in accord with Dr. Jackson, that disintegrated nerve-matter ceases either to generate or conduct nerve-force, yet there are periods of disintegration which cannot be overlooked. The first of these, of the utmost importance, is molecular derangement. It might arise from mere inhibitory action of reflective centres, or from the least possible transudation of serum from overgorged vessels. Nerve-force is essentially vital force, and, as in the inorganic world we have latent force, so much the more have we latent force in the organic. This, I presume, no physiologist is willing to deny. It is in molecular derangement, inducing molecular hyperæsthesia (irritability), that this latent nerve-energy is generated, not from overnutrition.

There is no such thing as overnutrition beyond what is compatible with perfect health. After this nutrition is perverted and degeneration begins, and so surely commences what Dr. Hughlings Jackson calls the positive condition of the motor organs in chorea, the negative follows when disintegration is complete. The questions which arise in reference to the cause, course, and termination of chorea (independent of *post mortem* microscopic evidence) do not to my mind favour the embolic theory. But one is bound to admit, and many cases have occurred in my own practice where extensive valvular disease has given rise to embola of the left Sylvian artery and its branches, which has been productive of defective speech, right facial palsy, jactatory involuntary movements of the arm and paresis of the leg, without spasm.

One case I remember of extreme interest, and would at first sight support the embolic theory; but the movements were unlike those of chorea, and the area of distribution of nerve-change gave evidence of a more limited destruction than is found in hemichorea.