TWO HARVEIAN LECTURES

ON

BRIGHT'S DISEASE AND ITS TREATMENT:

CONSIDERED MAINLY IN RELATION WITH ARTERIAL
TENSION FROM BLOOD-CONTAMINATION.*Delivered before the Harveian Society of London, November 1875.*

BY

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of the British Medical Association; etc.LECTURE I.—*Concluded.*

Arterial Tension.—It may be accepted as a clinical fact, that there is tension or continuous fulness of the arteries in every case of Bright's disease, whatever the form and stage of the affection, unless, owing to this very tension, the aortic valve have yielded, so as to allow the blood, when it cannot advance forwards, to make its way backwards, and so to relieve the distension of the arteries; or the mitral valve has given way, owing to the increasing size of the left ventricle, with the effect of obstructing the stream of blood from the lungs and lessening the supply to the system, and so transferring the tension from the arteries of the body to the arteries of the lungs; or when a like effect is induced by bronchitis.

When the arteries are tense, and the amount of blood they contain has not been lessened by the weeping away of albumen, the loss of red corpuscles, and wasting diseases, the whole arterial tree is lengthened and every arterial tube is widened. The root of the aorta consequently descends deeper into the chest, displacing the heart somewhat downwards, and the ascending aorta leans to the right; the arteries become slightly tortuous, but do not present a visible pulsation; the radial artery stands out, and, in extreme cases, seems to leave its bed; the amount of blood leaving the artery between the beats of the heart is so small that the tube scarcely relaxes, and so remains full and tense during that interval; the left ventricle, owing to the difficulty with which it sends its blood into the aorta, takes a longer time than usual to empty itself. On the other hand, the return stroke or reflux of the wave of blood backwards, when the contraction of the ventricles has ceased, begins more quickly, completes itself more rapidly, and is made with much greater force, than in health; so that it sends back the blood upon the root of the aorta and the arch with greater and more sudden force than in health. The left ventricle becomes enlarged, and its walls become thickened, owing to the increased force that it necessarily employs to send its blood into the arteries already filled, and to distend those arteries still more; and to the imperfect emptying of the cavity. The left auricle also, and the pulmonary veins, become unduly full. The arteries of the lungs partake of the general tension; but, owing to the difficult transit of the blood through the smaller vessels of the body, the supply to the vessels of the lungs is lessened; and, as the pulmonic arterial tree is much smaller than the systemic arterial tree, the right ventricle tends to complete its work more quickly than the left ventricle. During the early stages of acute Bright's disease, when the whole arterial system is suddenly called upon to do a disproportionate amount of work, and when, therefore, that system has not adapted itself to its additional labour, and is taken, so to speak, by surprise, the reflux of the blood in the pulmonary arteries, after the right ventricle ceases to contract, begins and ends sooner than the reflux of the blood in the aorta, so that the return wave of blood in the pulmonary artery falls back sooner upon the root of that vessel than the return wave of blood in the aorta falls back upon the root of that vessel.

At a later period, when the acute disease becomes chronic, the kidney being enlarged, the systemic and pulmonic arteries adapt themselves more nearly to each other. The systemic arteries give back their return-wave more rapidly, when, perhaps, the pulmonic arteries give theirs back less rapidly, so that the wave of each beats simultaneously upon the root of its respective artery; and this, although the ventricle may find a greater and greater obstacle to the emptying of its contents, and may, in consequence, continue to work for a greater length of time after the right ventricle has ceased its stroke. This state of things exists throughout in cases of granular kidney; for that disease, instead of making a sudden start in the acute form, begins slowly, and the vessels of the system educate themselves gradually and

imperceptibly to the new order of things. They are given, indeed, to a much greater degree of distension than are the arteries in fatty kidney; for, while in that disease there are a constant weeping of albumen and general waste and lessening of the volume of the blood, and especially of its solid particles, in cases with granular kidney the solid ingredients of the blood are retained, and the watery part of it passes rapidly off through the kidneys, which let the water through more easily, at the same time that the lightness of the action and the high pressure send out the water more rapidly. The same arterial tension that expels the water of the blood out of the vessels through the kidneys in cases of granular kidney, expels the water out of the smallest vessels into the surrounding tissues in cases of fatty kidney, and so establishes general dropsy. If from any cause, such as an acute attack, the water be expelled scantily through the kidneys in cases of granular kidney, the same effect necessarily results, and the high pressure drives the water out of the small vessels into the tissues, with the effect of inducing general dropsy, just as in cases of fatty kidney.

Arterial tension by no means implies in all cases arterial distension. It does so, as I have just said, in the typical cases of granular kidney, but does not do so in the typical cases of fatty kidney. In them, the weeping of the albumen lessens the volume of the blood; and the lowering of the vital forces of the tissues due to the loss of the solid ingredients of the blood and ulceration of the tissues, with general dropsy, lead to the establishment of wasting diseases, phthisis, diarrhoea, sickness, and so forth, that still further lessen the volume of the blood. The result is that, instead of having in those cases a radial artery cord-like and starting from its bed, we have a much contracted radial artery, the beat of which is often difficult to feel. But, although these arteries contain little blood, they are never empty. The blood finds the same difficulty in getting on through the smaller vessels; the muscular walls of the arteries contract down with all their force upon the blood, and with thickened walls and impaired power, owing to their narrowed calibre. The arteries, however small and contracted, are still characteristically tense.

I need not point out to you, for it is self-evident, the great, the vital importance of being able to recognise arterial tension and the absence of it; for, by ascertaining it, we often can at once fathom the nature of such a case. This is usually less important in cases of acute Bright's disease and of fatty kidney; for the general dropsy, the aspect, the pallor, stamp such a case at once. But it is far otherwise with cases of granular kidney; for they often remain in the apparent possession of full health even when the disease has made a deadly advance. A man in full work drops down in the street unconscious and in abiding coma, and enters the wards, perhaps never to regain consciousness. In these cases, the poison circulates through the blood; but the disease begins and continues so stealthily and slowly, that the system becomes accustomed to and consumes its narcotic poison, just as De Quincey and Coleridge did to their large doses of opium. In such cases, it is important to have an additional means of diagnosis, and, what is of equal, perhaps greater importance, an additional test of the depth of the disease; and this test we have with precision, if we can truly read the presence of arterial tension.

The signs of arterial tension are to be observed over the arteries themselves that are the actual seat of the tension, and over the heart, the action and rhythm of which are modified to meet the great strain of additional work thrown upon the left ventricle during its endeavour to overcome the tension. The arteries themselves which are the immediate seat of the tension present naturally the most important and immediate signs of this condition. The radial artery in extreme cases, owing to the distension and lengthening of the vessel, stands out and even seems to leave its natural bed, so as to be quite visible. It may even become tortuous; but when it is so, and especially when its pulsations are visible, there is reason to anticipate that arterial degeneration is joined on to arterial tension. The artery in typical cases feels hard, like a cord or tendon; it, indeed, sometimes feels exactly like the adjoining flexor tendon. It is not the pulse that you wish or ought to feel, but the artery. This is a very important distinction. You do not press the fingers into the artery, so as to compress it, but you glide them backwards and forwards from side to side of the artery; and you endeavour, if the case be one of extreme arterial tension, and especially if it be joined to arterial degeneration, to gently poise it up and lift it from its bed. You thus feel whether the artery is soft between the beats and hard during the beats, or whether it is continuously hard—whether the vessel is pulsating or not. You notice whether you can feel and count the pulse by simply placing the tips of the four fingers with a gentle firmness, without the least compression, over the artery. If the vessel soften between the strokes of the heart, and harden during or just after them, you know that the case is not one of great arterial tension. I find myself naturally taking this by comparison

and feeling my own artery and pulse in this manner after and between thus feeling the patient's artery and pulse. As I have just said, in the first or highest degree of tension, the artery feels like a cord or tendon; in the second degree, it is like an India-rubber tube distended with fluid; in the third degree, it resembles a rather firm empty India-rubber tube. In the majority of cases, the arteries do not present the typical self-asserting character of which I have just spoken. Indeed, when an artery does so assert itself, it is well to consider whether arterial degeneration is not joined to arterial tension, and even whether that partly local condition is not the only one. An atheromatous artery stands out, just as an India-rubber tube does, by its own intrinsic firmness. It can be moved backwards and forwards and lifted out of its bed, and it does not present to the gently applied fingers a very definite pulsation. Such pulsation is, however, to be perceived by a little attention, and such pulsation is always definitely visible even when there is no aortic regurgitation, for the artery even then can be seen crossing and meandering under the influence of the beat of the heart. The pulsation is not absolutely invisible in cases of extreme arterial tension, for the addition of blood during the transmission of the wave of fluid infinitely increases the size of the artery; but this increase is by no means so marked in extreme cases of arterial tension as in cases of simple arterial atheroma. When that condition of the artery is marked, it may be at once perceived by laying the tips of the fingers upon the artery, and moving them not across the vessel, but upwards and downward along and upon the line of the artery. A feeling like a row of rough irregular small shot is conveyed to the finger in extreme cases; but between this, and the comparatively slight case of which I have just spoken, there is every gradation of mammillary roughness.

The pulse, when felt by compressing the radial artery, is distinctly gradual and soft, and contrasts remarkably with a hard tube-like character of the uncompressed artery. The collapsing radial artery and pulse, and artery of aortic regurgitation, audible when the arm is raised, is the exact opposite to the pulse of arterial tension.

The presence of a sharp second beat over the enlarged ascending aorta to the right of the upper portion of the sternum, and mainly in the second space, is perceived in a few rare cases of acute Bright's disease on the one hand, and of granular kidney in its advanced stage on the other. This sign was well seen in a case of acute Bright's disease in University College Hospital under the care of Dr. Wilson Fox, who kindly invited me to see the case, which I examined with his clinical assistant Mr. Gould. There was distinct sharp second beat over the aorta that coincided with the second sound in the first and second right space close to the sternum, and extending outwards for fully an inch. I have observed the presence of this second beat in several cases of advanced granular disease of the kidney, but have not noticed it in cases of fatty kidney. The sharp second beat of the aorta to the right of the upper sternum is best felt, not by applying the tips of the fingers, but by steadily pressing over that region with that part of the palm of the hand that is close to the fingers.

The presence of a loud metallic sound, intensified, and a muffled or feeble first sound over the ascending aorta, with a feeble muffled and even absent first sound to the right of the upper portion of the sternum, is one of the most constant signs of arterial tension. It is audible in every form of Bright's disease—the acute form, the fatty kidney and granular, the lardaceous kidney, and those cases in which that again may be infected with a calculus, or the seat of suppurative disease from affection of the bladder or urethra. This intensified second sound has been observed in several cases by Traube and Rosenstein, and Dr. Grainger Stewart. Each gave a case in which this sign was predominant. Sometimes this intensified second sound is, however, extending only about two inches beyond the sternum, but sometimes it is audible over the whole upper part of the right side of the chest, as in a case observed the other day by Mr. Rossiter in St. Thomas's Hospital, who kindly called my attention to the case.

If the second sound over the aorta in the second right space be compared with the second sound over the pulmonary artery in the second left space, in each about an inch from the sternum, the sound is much less loud and more flat over the pulmonary artery, wanting in a metallic note, unless the metallic character of the aortic second sound drowns the natural character of the pulmonic second sound, and is heard over both places, though much louder over the aorta.

The muffling, or indeed sometimes silence, of the first sound over the aorta, gives increased emphasis to the loud metallic second sound. The cause of this muffling of the first sound is evident. The arteries are already full when the ventricle forces its blood into them, and the fluid already in the aorta prevents the ventricle from giving a sudden distension and shock to the walls of the arch. In cases of aortic regurgitation and of anæmia, when the arch is flaccid, a loud shock is often heard with the first sound owing to the hammer-like action of

suddenly expanding waves of fluid which strike against the walls of the artery. In cases, indeed, of aortic regurgitation, when the arm is raised and the vessels are thereby crippled, a shock may be heard with each beat of the pulse over the radial, which ceases to be audible when the arm is brought below the level of the heart, and the artery is thereby occupied with blood.

The region directly over the enlarged aorta, to the right of the upper half of the sternum, is not the only region where the intensified metallic second sound is audible in these cases. It is also heard over the region of the conus arteriosus at the left third and fourth spaces, below the region of the pulmonary artery, and above that of the tricuspid valve, which extends naturally from the third over the fourth and fifth spaces, but is somewhat lower in cases of Bright's disease with enlargement of the aortic arch, owing to the root of the aorta being lower than usual in the chest because of the lengthening of the arch. In some cases, the metallic aortic second sound is as loud over the conus arteriosus as over the aorta in the second right space; in some it is even louder, and in others it is less loud. The reason for the aortic second sound being audible in these cases over the conus arteriosus, instead of the second sound of the adjoining pulmonary artery, is obvious. The root of the aorta is situated immediately behind the conus arteriosus, where it fills up the space between the root of the pulmonary artery and the tricuspid orifice. The conus arteriosus presents but a small flat space, containing the blood of the right ventricle between the third space and fourth space and the root of the aorta, the second sound of which vessel is consequently heard in these cases over that region with great intensity. I need only remind you that the diastolic murmur of aortic incompetence is for the same reason heard as a rule with greater intensity less over the conus than over the ascending aorta to the right of the upper sternum, unless that artery is so enlarged as to pulsate against the surface in that region; and I need not say that it is louder there than over the left ventricle at the apex, since there the diastolic aortic murmur is rarely audible, although the stream of regurgitation pours down into the left ventricle, the reason being that the stream of blood entering the ventricle from the left auricle through the mitral orifice, is interposed over the outer part of the left ventricle at the apex between the ear and the regurgitative stream of blood pouring down from the aorta into the inner part of the left ventricle.

The presence of the second beat of the aorta, and of this intensified metallic second sound to the right of the upper sternum, and the muffling of the first sound, show the very great extent to which the arterial tube is stretched by its contained blood. The ascending aorta in health is limited within the right edge of the sternum, being entirely covered by that bone, and by a couch of lung of some thickness that interposes itself between the artery and the sternum. In Dr. Fox's case, the ascending aorta completely left its natural bed, moving outwards for at least an inch beyond the edge of the sternum—for to that extent Mr. Gould and myself felt it—and advancing forwards for fully an inch from it, came to the front of the chest, displacing the lungs, and hitting our hands when applied over it with a short sharp tap. Is this great enlargement of the arch of the aorta due to the great distension of the arteries from the blood tending to leave the vessels more slowly at its distant extremities, than it enters it from the ventricle at its root? or is it from the constant back hammer of the reflux blood which produces the second sound and second shock felt by the hand to the right of the upper sternum? Both of these agents or factors are at work. The repeated injection of the blood from the ventricle into the arterial lines readily expands the arteries and enlarges the whole tree. This is a great powerful distending agency of the Bramah press order. It works silently, without blow, and slowly. It detains its force, gently diffusing it noiselessly over the whole of the arterial tree, from its root to its finest branches.

The other force of which I have spoken is the reflux blow of the blood, driven backwards like the water-hammer, upon the walls of the root and arch of the aorta, including the aortic valve and the sinuses of Valsalva, with the whole stored up force of the lesser arteries increased by the last injection of blood from the left ventricle. I have again and again felt the force with which this reflux blow of return blood from the arteries, contracting after the systole, tells upon the walls of an aneurysm of the ascending aorta. This blow is delivered unceasingly. After the end of the beat of the heart, telling to expand the whole arterial tree by its great quiet injection of blood, we have the sudden blow of the arterial beat telling upon the root of the artery. Both these forces, the gradual and the sudden, the still and the noisy, tell to produce the common effect. But that the sudden return blow must have a second expanding effect, and a power of giving an injurious blow to the surface against which it beats, is most probable.

The following narrative of the effect of an analogous backstroke in

again and again bursting a pipe, has been kindly sent to me by Sir William Armstrong, who discovered the nature of the disease, and supplied the case. I give you the history in his words.

"A curious case of a water-pipe bursting without pressure occurred at the railway-station in Newcastle-upon-Tyne. The pipe was of considerable length, and was employed to carry off the water discharged from a hydraulic press used for revolving a large turntable. This pipe repeatedly burst, although it was a mere open-ended conduit. Upon investigation, it was found that the water ejected from the press attained sufficient velocity in the discharge-pipe to cause the column to run forward after the outlet valve of the press was closed. A momentary vacuum was thus occasioned at the back end of the pipe next to the press, and the column of water, recoiling by the external pressure of the atmosphere, delivered a blow which burst the pipe. The effect was counteracted by allowing a little air to be sucked into the pipe at a point near to the press, so as to prevent the formation of vacuum and cushion the return-stroke. With an elastic pipe, no such provision would have been necessary."

We have seen that in acute Bright's disease, and in cases of fatty kidney, atheroma sometimes affects the aorta and the aortic valves; and that concretions form on those valves evidently from inflammation. It appears to me, that in this reiterated reflux blow of the diastolic beat which is coincident with the second sound, we have an adequate cause for the excitement of inflammation, and of atheroma of the aortic valves, and aorta, and in conjunction with the systolic injection of blood, for the widening of the aortic arch. I would here remark, that the second sound is not caused by the closure of the aortic and pulmonic valves, which must close the tenth of a revolution of the heart's action before the period of the second sound, or the blood would then regurgitate from the arteries into the ventricles; but that this second sound is due to the reflux beat of the blood driven backwards after the systole by the contraction of the arterial walls, in the manner in which Dr. Burdon Sanderson has explained the second wave of the dicrotic pulse. This wave of the reflux blood is not indeed limited to the arch of the aorta, but is diffused over the whole arterial circulation short of its finest branches.

A remarkable sign appears, in the majority of cases of acute Bright's disease, in the form of doubling of the second sound over the region of the conus arteriosus, and less vividly over that of the pulmonary artery, and less still over that of the ascending aorta. I examined this point carefully with Dr. Donkin, Dr. Coupland, and others, fully two years ago, and we made up our minds that the second second sound was the louder and more metallic of the two second sounds; thus evidencing that the reflux blow of the aorta, with its second sound, was delivered after that of the pulmonary artery. This is the reverse of what takes place when there is doubling of the second sound over the pulmonary artery, owing to an obstacle to the flow of blood through the lungs. Then the second second sound over the pulmonary artery is the louder sound. I have traced doubling of the second sound lately in some cases of granular kidney with mitral murmur, and in these cases the order of the two sounds was the reverse of what it was in the cases of Bright's disease not so affected, the second second sound being evidently, at least in one case, that of the pulmonary artery. I examined this case at Guy's Hospital, through the kindness of Mr. Jones, the physician-assistant there. In these cases, the great arterial tension has been transformed by the mitral incompetence from the systemic to the pulmonic circulation.

The signs of arterial tension audible over the heart are less constant, and therefore less valuable, than the signs heard and felt over the ascending aorta and those felt over the pulse. These signs are, however, of considerable interest, and sometimes are of great importance and value, for they demonstrate sometimes the existence of the disease when it might be otherwise doubtful, and speak to the serious condition of the arterial tension that gave rise to these signs.

The heart's impulse occasionally, but not often, presents features of value. In Dr. Wilson Fox's important case of acute Bright's disease, of which I have already spoken, the apex-beat, at the time of the intensity of the disease, was fully an inch beyond the mammary line, or five inches by measure from the centre of the sternum, thus manifesting the immediate influence of the Bright's disease with its poisoned blood, its resisted capillary circulation, its arterial tension, and the increased force of the contraction of the left ventricle, to cause the great enlargement of that cavity and the thickening of its walls. At a later period in this case, the apex-beat had struck inwards, so that it was well within the mammary line.

Sometimes, as in this case of Sir William Jenner with fatty kidney observed by me with Mr. Gould at University College Hospital, the impulse is absent from the spaces and appears in the epigastric space, to the left of the ensiform cartilage. It is natural that this should be

the position of the impulse, when we consider that, in these cases, as in those of very great tubular dilatation and lengthening of the ascending aorta, the heart is pushed downwards into a lower position in the chest, owing to the lengthening of the ascending aorta and the lowering of its root.

In the cases of fatty kidney, and generally, perhaps, in acute Bright's disease also, the position of the impulse gives but little information as to the amount of the arterial tension and its effects upon the size of the left ventricle, and the general lowering of the heart. In cases of fatty kidney, as we have seen, the exhausting character of the disease and its secondary affections, tend to keep down the size of the heart and to diminish the volume of the blood, so that the ascending aorta is not lengthened downwards, and does not, therefore, displace the heart downwards also. We have seen, in one case of acute Bright's disease, that the seat of the impulse may give important information in the direction I am speaking of; but, practically, I have not found this sign to be of much avail in those cases. In patients affected with granular kidney, the position of the impulse is often of great moment; thus, in one case of the kind that I saw some time ago, the apex-beat could be felt low down in the sixth space, nearly or quite three inches to the left of the mammary line, and fully three inches below the level of the nipple. In that case, there was marked doubling of the first sound over the septum, the sign of which it still remains to me to speak; and the patient alternately breathed rapidly, deeply, and with great apparent distress for about forty or fifty seconds, and ceased to breathe, falling asleep the while, for about fifteen or twenty seconds. The cycles of alternately hurried and arrested respiration were tolerably, but not quite, regular. I may say that, in my second and concluding lecture, I shall bring before you, Mr. President, some important cases of this class, and especially one, a patient in St. Mary's Hospital, from whom I obtained respiratory tracings on Foucault's register somewhat on the method ingeniously contrived by Dr. Burdon Sanderson.

There are two influences outside the heart, one above and the other below that organ, that tend materially to modify the position of the impulse. I allude to the lungs in the higher region, which tend, by their increased expansion, owing to distress in breathing, often to coerce the heart in front, and perhaps to displace that organ downwards; the other, a very serious force directly and too strongly contending with the natural downward direction of the heart and lungs to displace them upwards; I allude to flatulent distension of the stomach and bowels, perhaps added to by ascites. This incident frequently occurs in the worst cases of kidney-disease, whether fatty or granular, and is of most serious import and requiring efficient help to counteract it from the physician. I shall briefly address myself to this question in my next lecture.

The last sign of arterial tension of which I have to speak is doubling of the first sound. The most extensive diffusion that I have yet met with of this sign was in Dr. Wilson Fox's important case, already alluded to, of acute Bright's disease. I heard, as Dr. Wilson Fox had already found, that there was marked and very neatly accentuated doubling of the first sound. There were three distinct sounds with each revolution of the heart's action, the space being longer before and after the second sound than between the two first sounds. Mr. Gould and I examined this case carefully together, and we found that the doubling of the first sound was audible over the whole of both ventricles, over the aorta and pulmonary artery as high as the first cartilage, at the root of the neck, over the innominate or right carotid artery, and, downwards, below the heart as low as the eighth cartilage. I have not heard in any other case anything like so extensive an area of doubling of the first sound as we heard in this. We found that, over and to the right of the right ventricle, the first of two first sounds was the louder, but it was the reverse over the left ventricle at and to the left of the septum; for there the second of the two first sounds was the louder sound.

As a rule, the doubling of the first sound is more or less limited to the region of the septum, or a little within the mammary line, and from one to two inches below the level of the nipple. Sometimes, under these circumstances, it is easy to find; but the sign, when present, and even audible to all the listeners, often requires a little time for its discovery and acknowledgment. When the doubling is markedly accentuated and extensive, it is usually, as in Sir William Jenner's case just quoted, audible over the whole right ventricle, where it may be especially emphasised over the epigastric space, that being the space at which the heart is most uncovered and is nearest to the surface; and it is lost over the left ventricle and its apex, being usually inaudible to the left of the nipple-line. This, under the circumstances, is natural; for the left ventricle is large and extensive, while the right ventricle is relatively small, so that the sounds from the former are readily trans-

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.

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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.