CASES ILLUSTRATING THE CAUSES AND EFFECTS
OF
FIBRINOUS OBSTRUCTIONS IN THE
ARTERIES,
BOTH OF THE BRAIN AND OF OTHER ORGANS.

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SINCE the publication in the 'Transactions of the Society' of the paper by Dr. Kirkes, on "Obstruction in the Cerebral Arteries," numerous cases bearing upon the subject have been brought forward. The opinions expressed by Dr. Kirkes in the paper alluded to have been much canvassed, and although they have in a great measure been adopted by pathologists, yet objections have been raised to some of these views, and reasons have been adduced for doubting the cardiac origin of the fibrinous plugs found in the cerebral arteries. It appeared therefore to the author that it would be desirable to collect together and to analyse as many cases as might be available, and in this way to endeavour to estimate the amount of evidence both in favour of and against the theory which supposes that these obstructions depend on fibrinous concretions or vegetations washed away from the cavities of the heart.

In the following communication accordingly the fatal cases bearing upon this subject which have passed under the author's observation have been collected together, and they include all that have occurred in the Middlesex Hospital during the last eight years. As some of these cases

have been published before in the 'Pathological Transactions' and elsewhere the headings only of these cases are given.

Moreover as the arguments in favour of the cardiac origin of these obstructions depends much on their association of the so termed fibrinous deposits in the internal organs it was necessary to enumerate the examples of these formations. The examples of fibrinous deposits are taken from the post-mortem records of the Hospital from 1853 to 1859.

The cases have therefore been divided into four groups. In the first group (eleven cases) are placed all those examples in which a plug in one of the cerebral arteries has been found associated with disease of the brain or hemiplegia. In the second (three examples) are placed certain incomplete cases in which softening of the brain has been found associated with warty growths on the valves of the heart, but in which the state of the cerebral arteries was not ascertained. In the third group are two instances of cicatrix of the brain. The fourth group (twelve cases) consists of the examples of fibrinous deposits in the internal organs but in which there was no affection of the brain.¹

GROUP I.

Examples in which a plug in one of the cerebral arteries has been found associated with disease of the brain, or hemiplegia.

CASE 1.—*Warty growth on the mitral valve; hemiplegia; softening of the brain; fibrinous plug in the middle cerebral artery.*

A female,² æt. 51, was admitted on June 25th, 1852, under the care of Mr. Shaw. She had fistula for which she

¹ [Only a few of the following cases are given in detail: the majority have been materially abridged by the author at the request of the Council.—*Sec.*]

² Vide 'Med. Times and Gaz.,' vol. v, p. 412; and 'Pathol. Trans.,' vol. iv.

was operated on; four days afterwards she was suddenly seized with syncope and hemiplegia, and died in twenty-four hours.

CASE 2.—*Warty growth on the mitral valve; softening of the brain; fibrinous plug in the middle cerebral artery.*

A man,¹ æt. 42, was admitted June 29th, 1852, under the care of Dr. Crawford. The patient had been ill a fortnight, and had the symptoms of softening of the brain. He died four days after admission.

CASE 3.—*Cancer of the penis; hemiplegia; softening of the brain; fibrinous plug in the middle cerebral artery; deposits in the kidney; disease of the valves of the heart.*

James R—, æt. 65, died November 26th, 1853, under the care of Mr. Shaw. The patient had suffered from cancer of the prepuce for six years, and became an inmate of the hospital three months before his death. On November 5th he fell down in the water-closet, apparently fainting, but did not lose consciousness. He recovered from the faintness in half an hour, and was then found to be hemiplegic on the left side. He gradually became worse, and died in a state of imperfect coma on November 26th.

Post-mortem examination made forty-six hours after death.

—The central part of the right hemisphere of the brain was found to be much softened, but not altered in colour. The arteries of the brain were all perfectly healthy with the exception of the right middle cerebral. At the point where the artery branches, a fibrinous plug, the size of a grain of wheat, was found firmly adherent to its walls. Attached to the distal side of this plug were two slender strings of fibrin about half an inch in length, these extended into each of the branches of the artery, and did not adhere to the walls of the vessels. In the right kidney was a

¹ Vide 'Med. Times and Gaz.,' vol. v, p 412; and 'Pathol. Trans.,' vol. iv.

bright, yellow, fibrinous deposit; in the left, a cicatrix. The spleen was healthy. The lungs were congested; there was a peculiar cartilaginous growth on the pleural surface of the diaphragm; the mitral valve was thickened; the free edge of the aortic valve much calcified. There were no distinct warty growths on the valves, or on the endocardium.

CASE 4.—*Hemiplegia; softening of the brain; gangrene of the feet and nose; fibrinous deposits in the spleen; vegetations on the mitral valve; plugging of the middle cerebral artery.*

Ann T—, ¹ æt. 37, died under the care of Mr. Moore. She had suffered from cancer of the uterus for six months; twenty-two days before her death she became hemiplegic. The gangrene appeared ten days later.

CASE 5.—*Softening of the brain; hæmorrhage; fibrinous plug in the middle cerebral artery; warty growth on the endocardium; fibrinous deposit in the spleen and kidneys.*

Kezia S—, æt. 15 years, was admitted on March 11th, 1858, under the care of Dr. Hawkins. She had been ill a fortnight. She was speechless when admitted, and died four days afterwards.

CASE 6.—*Rheumatic endocarditis; apoplectiform seizure; fibrinous plug in an artery of the brain; extravasation of blood; warty growth on the aortic valve; fibrinous deposit in the spleen and muscular substance of the heart.*

John S—, æt. 27, a bricklayer, was admitted July 14th, 1857, under the care of Dr. Hawkins. The patient had suffered twice formerly from acute rheumatism, and had symptoms of heart disease for twelve months. He was admitted with dropsy. Six days afterwards, he suddenly became insensible, and died on the following day.

¹ Dr. Van der Byl, 'Path. Trans.,' vol. vii.

CASE 7.—*Hemiplegia ; softening of the brain ; fibrinous obstructions in several of the cerebral arteries ; fibrinous deposit in the spleen and kidneys ; warty growth on the valves of the heart.*

Frances C—, ¹ a female, æt. 44, when in apparent health, fell down hemiplegic. She rallied slightly, but died comatose twenty-one days subsequently.

CASE 8.—*Bronchitis ; hemiplegia ; fibrinous plug in the middle cerebral artery ; deposit in the spleen and kidneys ; vegetation on the aortic valves.*

Elizabeth S—, æt. 58, died under the care of Dr. Hawkins, on June 17th, 1858, having been admitted two days previously. She had suffered from cough for a month, and three days before admission she became suddenly insensible and paralysed on the right side. When admitted she was quite insensible, the breathing being almost stertorous ; she was hardly able to swallow.

CASE 9.—*Mitral disease ; hemiplegia ; softening of the brain with slight extravasation on the surface ; fibrinous plugs in the middle cerebral artery, and in one of its branches ; fibrinous deposit in the spleen and kidneys.*

Eliza S—, æt. 46, was admitted under Dr. Stewart, on September 1st, 1858. A systolic mitral murmur was detected. On the morning of the 28th she suddenly became hemiplegic, her consciousness remaining but the power of speech being lost. She became less sensible, but not comatose, and died on October 30th.

CASE 10.—*Rheumatic endocarditis ; hemiplegia ; softening of the brain ; obstructed middle cerebral artery ; warty growths on the endocardium ; fibrinous deposits in the spleen and kidneys.*

¹ Dr. Van der Byl, 'Path. Trans.' vol. ix.

Jane C—, a servant, æt. 23, was admitted on November 22nd, 1860, under the care of Dr. Stewart. She had been out of health for six months, and had suffered from rheumatic pains, and from severe cough for some months; and she had suffered from rheumatic fever once, eight years before. On November 13th she lost the use of the left arm and leg. When attacked she became partially comatose, and remained in this condition for four hours. After this she fully regained consciousness, and became somewhat excited, and wandered a good deal. She then became partially comatose, and became subject to convulsive movements which especially affected the eyeballs, and she died November 24th.

CASE 11.—*Rheumatic endocarditis; hemiplegia; softening of the brain; plugging of the middle cerebral artery; fibrinous deposits in the spleen and kidneys; cicatrix in kidney.*

Fanny N—, a married woman, æt. 27, was admitted under the care of Dr. Stewart, on February 12th, 1861. She stated that she had been attacked a fortnight before Christmas with pain and swelling of the feet. At the time of admission she was suffering from pain in various parts, and a loud, rough, whizzing murmur was heard over the body of the heart. For a week after admission the murmur continued to increase in intensity; on the 25th, friction sound was audible, and on the 27th the murmur had become musical.

On the morning of March 7th she awoke in her usual health, and at 9 a.m., as she was being lifted in bed, she fell back in a state of complete insensibility; there was some foaming at the mouth, but no convulsive movements. In the course of the morning consciousness partly returned, her eyes and face drawn to the left; the right arm and leg paralysed. She gradually sank, and died incompletely comatose on the 9th.

GROUP II.

Examples of softening of the brain associated with disease of valves of the heart, but in which the state of cerebral arteries is uncertain.

CASE 12.—*Rheumatism; endocarditis; hemiplegia; softening of the brain and hæmorrhage; fibrinous deposits in the spleen and kidneys.*

Robert T—, æt. 15, a plasterer's boy, was admitted under the care of Mr. Moore, on October 18th, 1855. At the time it was asserted, by those who brought the boy to the hospital, that he had fallen from a scaffold, but this statement was subsequently found to be incorrect. In the previous winter he had been laid up with pains in his joints. After this illness, he did not regain his strength. On the morning of his attack he went to work as usual, but stated that he felt an odd sensation about the mouth. When on the scaffold he became faint, and was carried down by his fellow-workmen. When admitted, he was partly unconscious, but able to give his name; his face was drawn to the right side, and his left arm and leg were powerless. Subsequently the muscles of the paralysed limbs became contracted; he had one slight convulsive seizure, and passed into a state of profound coma, in which state he died on October 23rd.

Post-mortem examination made thirty-eight hours after death.—Over the right lateral ventricle of the brain there was a clot of blood about the size of a hen's egg. Several small clots and patches of "ecchymosed" brain were found near to the larger clot. The structure of the brain around was much softened. In the spleen and in the kidneys there were several fibrinous deposits, and cicatrices of various ages. In the heart, a large and loose warty growth was found attached to the aortic valves. A small aneurism

was also discovered, communicating with the left ventricle and passing into the septum. The cerebral arteries were not examined.

CASE 13.—*Purpura ; hemiplegia ; softening of the brain and cerebral hæmorrhage ; fibrinous deposits in the spleen ; cicatrix in the kidney ; vegetations on the aortic valves.*

James B—, æt. 23, died November 21st, 1853, under the care of Dr. Hawkins. He had been in the hospital in September, suffering from purpura. He was readmitted on November 8th, having fallen asleep in the middle of the day and having awoke hemiplegic. The amount of paralysis increased, and he died on November 21st, after two violent epileptic fits.

CASE 14.—*Bronchitis ; pneumonia ; hemiplegia ; softening of the brain ; fibrinous deposits in the kidneys.*

Caroline H—, æt. 54, was admitted with bronchitis, under the care of Dr. Stewart, January 20th, 1857. On the morning of January 22nd she became faint, partially unconscious and hemiplegic. After this she became loquacious and then semicomatose, with twitching of the muscles, and died on November 25th.

GROUP III.

The two cases which form the next group exhibit what are probably the ultimate results of obstructions in the cerebral arteries when the patient recovers the immediate effects of this lesion. In the great majority of cases death is no doubt the consequence, and this event it will be seen in the foregoing cases rarely takes place sooner than three or four days, and is rarely delayed longer than three weeks. There is, however, no doubt but that recovery does ensue in a certain number of instances, and chiefly in those in which a small artery only has been obstructed. The ap-

pearance presented by the brain structure under these circumstances closely resembles that which is observed in other organs, and a cicatrix corresponding in all particulars to the cicatrices in the spleen and kidney is formed. In neither of the cases which are here brought forward is the proof absolutely conclusive that the cicatrix originates in the way indicated, but in one instance the patient having survived a transient attack of hemiplegia died twelve months afterwards, and presented after death a cicatrix in the cerebellum, and warty growths on the valves of the heart; and in the second case the mode of seizure corresponds to a sudden obstruction of an artery, but unfortunately the post-mortem examination did not extend to the internal organs. In neither case was there any reason to suppose that the cicatrix had originated from any other cause, such as apopleptic cyst, and the position of the cicatrices in these instances renders it very difficult to ascribe the cicatrix to another cause than obstructed artery.

It need hardly be here remarked that little evidence can be derived from a careful examination of the arteries in such cases, as the obstruction must have occurred so long before that the vessel would have become obliterated and absorbed, the collateral arteries having become enlarged and carrying on the circulation.

CASE 15.—*Ovarian tumour; hemiplegia (imperfect) twelve months before death; cicatrix in the cerebellum; cicatrix in the kidney; warty growths on the mitral valve.*

Sarah G—, æt. about 34, died under the care of Mr. De Morgan, on September 28th, 1853. She had suffered from ovarian tumour and dropsy for six years before her death. In 1849 an attempt was made to perform the operation of ovariectomy, but the adhesions being considerable the operation was abandoned; after this she was repeatedly tapped. About twelve months before her death she became partially hemiplegic for a few hours, but at the end of a few days she regained her usual state of health.

Post-mortem examination made twenty-eight hours after death.—The convolutions of the cerebrum presented the appearance of being somewhat atrophied. On the side of the under surface of the cerebellum was a deep excavation large enough to admit the points of three fingers, the brain tissue being shrivelled and its place occupied by œdematous membranes. There were several warty growths on the mitral valve, there was also a cicatrix in one of the kidneys. The only other disease observed was the ovarian tumour.

CASE 16.—*Sudden loss of power over one leg, followed by partial paraplegia ; death six years after from laryngitis ; cicatrix of the cerebrum.*

A gentleman, æt. 58, died of laryngitis in January, 1857. Six years before his death he suddenly felt as if the left leg had gone through the floor. After a month he recovered some power, but the right leg also became affected. The paraplegia by degrees slowly increased ; the sight of the left eye became impaired. At the post-mortem examination a small cicatrix was discovered on the front part of the right half of the cerebrum.

GROUP IV.

The following are the headings of the cases of fibrinous deposits in the internal organs of the body where there was no affection of the brain. With the exception of case 28 it will be seen that they were all associated with disease of the cardiac valves, or coagula in the heart.

CASE 17.—Sarah D—, æt. 47. General dropsy ; dilatation and hypertrophy of the heart ; warty growth on the aortic valve ; fibrinous deposit, and cicatrix in the kidney.

CASE 18.—Louisa M—, æt. 42. Bronchitis, emphysema, cirrhosis of the liver ; vegetations on the mitral valve ; fibrinous deposit and cicatrices in the kidneys ; deposit in the muscular substance of the heart.

CASE 19.—William J—, æt. 23. Hypertrophy of the heart; vegetations on the aortic and mitral valves; numerous fibrinous deposits in spleen; granular disease of the kidneys.

CASE 20.—Thomas H—, æt. 22. Hypertrophy of the heart; adherent coagula in the cavities of the heart; pulmonary apoplexy; cirrhosis of the liver; numerous fibrinous deposits in the kidneys.

CASE 21.—Sarah Q—, æt. 36. Puerperal metritis; vegetations in the heart; fibrinous deposits in the spleen; fibrinous (?) deposits in the lungs.

CASE 22.—Martha J—, æt. 25. Hypertrophy of the heart; vegetations on the mitral valve; loose coagula in the cavities; pulmonary apoplexy; fibrinous deposits and cicatrices in the kidneys.

CASE 23.—Mary P—, æt. 36. Osteoid cancer (myeloid) of the humerus scapula, &c., with similar tumours in the lungs; vegetations on the mitral valve; fibrinous deposit in the spleen.

CASE 24.—Mary C—, æt. 28. Hypertrophy of the heart; vegetations on the mitral and aortic valves; granular disease of kidneys; fibrinous deposit in the spleen, and in the muscular substance of heart.

CASE 25.—James T—, æt. 28. Hypertrophy and dilatation of heart; endocarditis; fibrinous deposit in kidney.

CASE 26.—Eliz. M—, æt. 39. Loose coagula in the heart; plugging of the left common iliac artery; gangrene of the leg; numerous fibrinous deposits and cicatrices in the kidneys and spleen.

CASE 27.—George K—, æt. 25. Hypertrophy of the heart; thickening and adhesion of the aortic valves; hepatisation of the lung; cicatrices in the kidneys.

CASE 28.—John C—, æt. 30. Grey hepatization of the lungs; several fibrinous deposits in the spleen; heart and its valves healthy.

SUMMARY.

As regards the complete cases in which there was *softening of the brain, and obstruction in the cerebral arteries* (Group I), it is seen that in nine out of eleven cases fibrinous deposits were found in either spleen or kidney, or in both. In the remaining two cases no such appearance is noticed, but as these were the first cases observed, and the records are not perfect, it cannot be absolutely affirmed that no such deposits existed in the spleen and kidneys of these patients.

In ten out of the eleven cases the middle cerebral or one of its large branches was the artery obstructed, but in three out of these ten cases there was a second obstruction in some other artery of the brain.

In two of the cases there was gangrene of some part of the surface of the body, in one of these instances there were several patches of (local) gangrene, in the other there was but a single patch.

In one instance, in addition to fibrinous deposit in other organs, there was a fibrinous deposit in the walls of the heart.

In ten out of the eleven cases there were warty growths on the valves of the heart, similar in structure to the fibrinous plugs in the cerebral arteries. In the remaining instance no obvious warty growth was found after death, but there was extensive and rough atheromatous and calcareous disease of the aortic valves, and of the aorta.

In the other *three cases* (Group II) in which there was *cerebral disease, but in which the state of the arteries was not ascertained*, it is seen that in two there were vegetations on the valves of the heart; in the third case there was hepatization of the lung. In two of the three cases there

were fibrinous deposits in both the spleen and kidney, and in the third there were deposits only in the kidney.

As regards the cases of *fibrinous deposits without cerebral disease* (Group IV) it is seen that out of the twelve cases observed there were vegetations or loose clots in the heart in nine cases. In one of the remaining instances there was a rough condition of the endocardium, but no distinct vegetation. In the eleventh case the aortic valves were thickened and adherent together, but in this instance there was not a recent fibrinous deposit, but a cicatrix of old date. In the last case the valves and the endocardium were healthy, but the lung was in a state of gray hepatization.

It should also be noted that in two of the cases of this group there were fibrinous deposits in the muscular substance of the heart, and in one there was gangrene of the leg.

As regards the *cerebral symptoms* in the cases of the first two groups: No satisfactory history could be obtained of two of the cases. Out of the remaining twelve cases, in five syncope and coldness of the surface was a prominent symptom.

At the time of attack there was no loss of consciousness in three cases, two were attacked it is supposed during sleep and awoke hemiplegic. In one of these latter cases (9) the exact time of attack is somewhat doubtful, in the other case (13) it seems very probable that the seizure did take place during sleep. In all the remaining nine examples there was more or less complete loss of consciousness, but in none was there profound coma. The duration of the insensibility varied from a few minutes to three or four hours. In one instance the seizure was preceded by intense pain in the head.

When an artery becomes obstructed the effect produced on the part to which that artery should supply blood is very similar in the different regions of the body. For a short time the supply of blood is completely cut off from the part. After an interval, however, the collateral circulation

enlarging, enables the part to receive a scanty supply of nourishment. In certain situations it is well known that there are but few anastomoses of the arteries, and the collateral circulation can only therefore be carried on to a very limited extent. This absence of anastomosing vessels is especially noticed in the cerebral arteries (beyond the circle of Willis) and also in the arteries of the spleen and kidneys. It is in these organs more particularly that the evil effects of obstructed arteries are observed. In other parts of the body the collateral circulation is quickly established, and the condition of the affected part undergoes no material alteration, and it is probably only in those cases in which two or more neighbouring arteries become obstructed that any visible effect is produced, and in these instances the part becomes either gangrenous, or, if the circulation does not entirely fail, it may become the seat of fibrinous deposit; the former is no doubt the condition which existed in the instances of local gangrene (cases 1, 4, 26) and the latter in the examples of fibrous deposit in the walls of the heart (cases 12, 18, 24).

A consideration of the alterations which take place in the circulation of a part, of which the nutrient artery becomes obstructed, will explain the structural changes which are met with. At first, simple local anæmia is produced, and this condition may frequently be observed both in the spleen and kidney. The pale patch corresponds accurately with the area to which the artery should distribute its supply of blood, and the form is, therefore, almost always that of an irregular cone, the apex of which is directed towards the obstructed artery. On microscopical examination, these pale patches do not exhibit any deviation from the normal structure of the part. In the next stage, the vessels of the surrounding parts enlarge in order that they may carry a supply of blood to the affected structures; and as it is more especially by means of the minute vessels that this anastomosis takes place, a bright zone of enlarged vessels forms and encircles the pale patch. During the time that nature is making this effort to reestablish the

circulation the pale patch is in a state of commencing gangrene, but it is saved from absolute death by the feeble and irregular supply of blood which it receives from these surrounding vessels. A low form of inflammation follows, during which compound exudation corpuscles are formed (especially in the instance of the brain), the tissue undergoes rapid degeneration, and fat accumulates both in the cell- and the other structures of the part. During this stage, the bright zone formed by enlarged vessels is still observed, and the patch becomes of a bright yellow colour. The change of colour depends upon the accumulation of fat in the form of granules in the tissue of the part; and it should be observed that the colour does not indicate the presence of fibrine as was formerly supposed. The term fibrinous deposit cannot, therefore, be properly applied to this condition, but it has been made use of in the present communication, as it is the term usually employed, and, although the fallacy has been proved by many observers, no other convenient term has yet been invented by which we can designate these appearances.

In this stage hæmorrhage frequently takes place into the degenerated structure, and this complication was observed in five out of fourteen cases of softening of the brain.

After this, the part recovers to a certain extent its normal condition; the circulation among the degenerated structures becomes more perfect; the bright zone of increased vascularity by degrees disappears. The exudations take on more of the plastic character and undergo contraction, the yellow colour gradually fades, and at length a cicatrix is the result. In the instance of the spleen, kidney, or muscular tissue, this cicatrix is clearly formed of an imperfect fibroid tissue, but in the brain there is yet no positive evidence of the formation of this tissue among the brain structure as a result of obstruction in the arteries.

It will be well, in conclusion, shortly to review the various arguments both for and against the theory which supposes that these obstructions have been washed away from the cavities of the heart by the current of the blood. It will

hardly be necessary here to notice the hypothesis which has been adduced by some, namely, that the fibrinous plugs in the cerebral arteries are the effect and not the cause of the softening of the brain. The want of evidence to prove that a similar condition is observed in other parts of the body, and the fact that the obstruction is formed of fibrinous material and not of simple blood clot, are in themselves reasons for doubting the correctness of such an hypothesis. Moreover, the plug is not usually met with in the small arteries of the part affected, but in one of the arterial trunks; the obstruction is therefore at a distance from the softened portion of brain.

Supposing, then, it to be granted that the obstructed artery is the cause and not the effect of the disease in the brain, the plug may have been formed in the situation in which it is found after death, or it may have been washed from a distance. Both of these hypotheses may be maintained; and there appears to be but little doubt that obstructions in the arteries may originate in either way, and it is therefore necessary to distinguish between the appearances presented by plugs formed in each mode.

One chief difficulty which has presented itself in assigning a distant cause to these plugs is the fact that certain cases have been met with in which no vegetations or emboli have been discovered in the heart. In several of these apparently exceptional cases an inflamed lung was noted, and the presence of this lesion satisfactorily accounts for the occurrence of loose masses of fibrin in the blood. In this manner all the cases of obstructed arteries which have fallen under my own observation are accounted for, there having been noticed in each instance either vegetations, loose clots, or some obvious cause in the heart, or an inflamed lung.

There remain, however, certain cases recorded elsewhere in which no such cause can be traced, and the majority if not the whole of these cases are to be regarded as examples of local arteritis. It will at once be seen that however much the causes of these obstructions in arteries

may differ, the effect produced on the part supplied by these arteries must be precisely similar.¹

It is, therefore, necessary to distinguish between the appearances presented by an artery filled with fibrinous matter from arteritis, and a plug washed from the heart or elsewhere; and practically there is, I believe, little or no difficulty in drawing this distinction. In the first place it should be observed there is structurally the greatest similarity between the fibrin which forms on the valves of the heart, and that produced by arteritis. I have carefully examined in several of the foregoing cases both the plugs found in the arteries and the fibrin on the endocardium, and have been unable to discover either by the naked eye or by the microscope, any essential difference between them. Moreover, occasionally, we meet with striking features of analogous structure, which appear conclusively to prove that the plugs have been washed from the heart, as, for instance, in Case 6, in which both the plug in the artery and the warty growths on the endocardium, were gritty owing to their having undergone partial calcification; and it should also be observed that in this instance the artery was free from atheromatous or calcareous disease. Except in such an instance as this, the argument derived from identical structure is, however, not of any great weight; for we should hardly expect to find any essential difference in the histological characters of fibrin produced in the interior of an inflamed artery from that presented by the material which forms vegetations on the valves of the heart.

The condition of the plugs found in the arteries depends upon the length of time they have rested in their present site; and as they usually give rise to inflammation, the opportunity frequently occurs of comparing the appearances of a plug thrust into an artery with that produced by local

¹ An excellent example of this statement was observed in a case of general arteritis, recorded by Dr. Van der Byl in 'Path. Trans,' vol. ix. In this instance there were fibrinous deposits in the spleen, kidneys, in the muscular substance of the heart, and also softening of the brain.

arteritis. If it happens that the patient dies quickly after the attack, the plug is found loose and non-adherent to the wall of the artery; if, however, death does not ensue so rapidly, the plug is generally found to be slightly adherent to the artery. In several instances, the amount of inflammation produced by the presence of the plug has been so great as to cause the effusion of lymph around the artery at the seat of the obstruction.

A very forcible reason for attributing a distant cause to these plugs is the fact that frequently several arteries become blocked up, the plugs usually being met with at points of bifurcation. In all these instances the part of the artery intervening between the plugs was perfectly healthy, and exhibited no evidence of inflammation. Virchow has supposed that these secondary plugs are produced by fragments of a large plug washed away, but it is more probable that the smaller artery became obstructed first, and then the larger. If the larger artery were the first blocked up the circulation in the branches would of course be arrested, and therefore there would be no force to wash a fragment into one of its branches.

The fact that plugs are almost invariably formed at those points where an artery divides and suddenly becomes smaller, tends strongly to prove that the obstruction has originated from the arrest of a solid body suspended in the blood.

It has been urged by Dr. George Johnson¹ that in the instance of the so-termed fibrinous deposits in the kidney there is evidence to show that the obstruction takes place in the inter-tubular capillaries, and not in a branch of the renal artery. There is, however, certainly, strong reason for doubting the correctness of the inferences from these observations, in the fact that fibrinous deposit associated with arteritis of the renal artery² precisely corresponds in all particulars with fibrinous deposit associated with vegetations on the endocardium; whilst, on the other hand, the

¹ "On the Minute Anatomy of the so-called Fibrinous Deposits in the Kidneys," by Dr. G. Johnson, 'Path. Trans.,' vol. ix.

² "A case of Arteritis," Dr. Van der Byl, 'Path. Trans.,' vol. ix.

appearance presented by a kidney affected with inflammation of the renal vein¹ is altogether of a different nature.

As an additional reason for believing that portions of vegetations may be washed away from the valves of the heart, those cases might be adduced in which large emboli have obstructed one or more of the great arteries of the body, as in Case 26. In these instances the emboli in the artery, corresponding in every particular to the loose clots found in the hearts of the same patients, leave no doubt as to the origin of the bodies in the arteries. If, then, it is allowed that these bodies may be carried to a distance by the circulating blood, it can scarcely be denied that small and loose vegetations from the valves of the heart may be carried in like manner.

The common origin of these obstructions is also supported by the almost invariable association of fibrinous deposits in the spleen and kidney with these instances of softening of the brain. Such a constant companionship can hardly be the result of accidental causes, and it is difficult to imagine that local arteritis can occur in so many and such distant parts of the body, whilst all the remaining portions of the arterial system remain free from disease.

If any additional testimony were required to establish the fact of the washing away of these bodies from the heart, it would be found in a consideration of the symptoms of this form of brain disease. The sudden seizure, the faintness, and the rapid development of the ordinary symptoms of softening of the brain, are all explained on the supposition that a large artery of the brain has suddenly become obstructed, whilst they do not meet with solution on the hypothesis that the obstruction has taken place in the more gradual mode, which would probably have been the case had the artery been obstructed from inflammation of its coats.

I have to express my great obligations to the physicians

¹ "A Case of Extensive General Phlebitis," by the author, 'Path. Trans.,' vol. ix.

and surgeons (both past and present) of the Middlesex Hospital, for their extreme kindness and liberality on all occasions, in affording me every facility for observing the patients under their care, and for placing at my disposal their notes of the cases.