AN ADDRESS ON THROMBOSIS AND EMBOLISM.

Delivered at the opening of the Liverpool Medical Society.

By JAMES TURNBULL, M.D., F.R.C.P.,

Physician to the Liverpool Royal Infirmary; President of the Medical Institution; etc.

I HAVE selected as the subject of this address the coagulation of the blood in the heart and blood-vessels during life; and I have chosen it partly because I have met with several cases during a comparatively recent period, and also because the effects of coagulation are so widely spread in their influence on the various organs of the body, that it must interest those who are engaged in every branch of practice or medical inquiry.

From an early period, clots and concretions were from time to time observed in the vascular system, which led to speculations similar to those which have occurred in more recent times as to whether they had formed during life or after death. The great German pathologist Virchow was, however, the first to examine the whole subject in a complete and systematic manner, and to put together and explain the isolated facts seen by previous observers without being fully understood in their relation to each other. He placed the whole subject on a firm foundation, and applied the term "thrombosis" to coagulation of blood in the heart or vascular system, and "embolism" to the plugging of vessels which results from the transportation of such clots or thrombi by the current of the blood.

The light thrown on pathology by these investigations was regarded by the late Dr. Parkes as one of the great recent advances in medical science; and, to prove that such is the case, let me point to the embolic pneumonias which arise from plugging of branches of the pulmonary artery, to the sudden deaths which occur in puerperal and other conditions from more complete obstruction of the artery, to the paralytic seizures from plugging of the cerebral arteries, and to the gangrenous attacks in the extremities consequent on heart-affections: all of which were at one time involved in much obscurity.

Systematic treatises on this subject have been published in the German language by Conheim and Cohn, but the researches of British medical men are chiefly to be found in the medical periodicals and in papers in the *Transactions* of the societies, which abound in cases illustrating the subject, and showing that the profession in this country is fully alive to its importance. In referring to thrombosis of the veins, Mr. Prescott Hewett, in his address to the Clinical Society in 1873, stated that it is of greatly increasing frequency, but this probably arises from the fact that the true nature of this condition is now more generally recognised.

Before directing your attention to the clots and concretions which form in the vascular system, I would remind you that the coagulation of the blood is produced by the passage of the fibrine from the soluble to the insoluble fibrinated condition. This seems to be dependent on some change in the vital condition of the blood which is not fully understood. Dr. Richardson's theory of its dependence on the escape of ammonia has not been sustained; but the fact remains that the volatile alkali as well as the fixed alkalis have the power of retarding or even preventing coagulation.

In the normal condition, the blood contains from 2 to $3\frac{1}{2}$ parts of fibrin in 1,000, but the proportion is altered in many diseases, and is increased greatly in all the acute inflammatory diseases, especially in pneumonia and acute rheumatism. This increase is the cause of the buffy coat formerly so carefully looked for in reference both to diagnosis and treatment. In the puerperal condition, the blood contains an increased quantity of fibrin, which contracts slowly and presents generally the buffy coat. In chlorosis, it occurs also from there being some absolute increase of the fibrine, together with a large relative increase, owing to the great diminution of red corpuscles. In many cachectic diseases, such as cancer, there is a condition of the blood similar to that of chlorosis, and in all the diseases now referred to the condition of the blood itself predisposes to the formation of clots in the vascular system. In persons of gouty constitution, there is a remarkable tendency to thrombosis in the veins. This may be due to the presence of an acid or other morbid matter in the blood; but Sir James Paget, who has drawn attention to this form of thrombosis, seems to think

that there is a local phlebitis. It is, however, worthy of observation that, in the allied disease acute rheumatism, supposed also to be connected with an acid diathesis, there is likewise a tendency to the deposit of fibrin, especially on the valves of the left side of the heart.

With regard to thrombi, they may form in any part of the vascular system. In the heart, we meet with three kinds of clots or thrombi:—1. The loose, soft, dark-coloured clots, which form ordinarily after death; 2. The firmer yellowish-white thrombi, often adherent to the walls and entangling the valves, which form while death is taking place, or shortly before; 3. The firm laminated concretions, made up of layers of fibrin with colourless corpuscles, which have existed so long, that they have often undergone partial softening, especially towards the centre of the concretion.

The second variety of concretions, with which most of us are familiar, form at a variable but short period before death, and they occur chiefly in cases where there has been a severe and protracted struggle, more especially in pulmonary and cardiac diseases. But little attention has been paid to these concretions, and they have scarcely been looked upon as a cause of death, and have been regarded as merely an evidence of how life has terminated. I am, however, disposed to look upon them as in some cases a cause of death which might possibly be warded off if we could prevent their formation, and so gain time for resolution of pneumonia or other diseases with which they are associated. I had lately an opportunity of seeing a *post mortem* examination in a case of acute pneumonia, where the right cavities of the heart were filled with firm whitish concretions enveloping the valves and cordæ tendineæ, which must have formed a considerable time before death, and where the left ventricle contained also a firm white flattened concretion of still older date. In this case, I think that the immediate cause of death was as much in the blood itself as in the condition of the lung, and that it must have begun with the coagulation of the blood in the cavities of the heart.

In the treatment of this disease, I think we should not overlook the use of any means that can be used to prevent the formation of such fibrinous concretions in the heart. The use of stimulants, which urge on the languid circulation, is clearly indicated; but the alkalis, and more particularly the volatile alkali ammonia, are the only agents we are acquainted with which have a direct power in diminishing the tendency to coagulation, and they should, therefore, be freely administered when we have reason to dread the formation of concretions in the heart. The value of ammonia for this purpose, I may add, was long ago pointed out by Dr. Richardson.

With regard to the class of diseases in which this kind of concretions occur, Dr. Bristowe found that, in a series of forty-one cases, in all but one, which was a case of sloughing sore-throat after scarlet fever, they were cases of either heart-disease, arterial disease, chronic bronchitis, renal disease, tuberculosis, or such a complex of two or more of these affections as we are constantly meeting with in hospital practice.

The third kind of concretions are not always separated by a distinct line of demarcation from the previous one. The concretions, whether in the heart or blood-vessels, which have begun to soften and break down in the centre are often of long standing. I had clear evidence in one case of a concretion in the right ventricle, which was partially softened, of its existence for more than one month; and, in a case recorded by Lancereaux (Observation cxxii), where several concretions had so softened as to assume the form of fibrinous cysts, the duration was probably much longer. The softening of these concretions produces the puriform appearance often seen in the centre, and the washing away of the detritus is a cause of capillary embolism in various organs: in the lungs, when the thrombus or concretion is in the venous system or right side of the heart; in the liver, when the concretion is in the portal system; and in the brain, spleen, and other organs of the body, when the concretion is in the left side of the heart or larger arteries.

Dr. Ogle reports eight cases of concretions in the cavities of the heart of old standing, some partially converted into a puriform fluid. Of these, five were in the right auricle, one in the right ventricle, and two in the left ventricle.

The thrombi which form in the veins may undergo the same kind of softening, but they also undergo other changes, and may be partially or completely absorbed.

With respect to the causes of thrombi, we have seen that the clotting of blood by which thrombi are formed is caused by an alteration in the state of the fibrin, and the conditions which determine this in the heart or vessels have been fully studied by pathologists, and they may be arranged in two principal groups:—1. Those which produce interruption, stagnation, or slowing of the blood-current; 2. Those which produce changes in the vessels or their walls. In many cases, however, these conditions are combined and act together.

To the first group belong the thrombi from ligature of vessels, from the pressure of tumours or abscesses, tubercular or carcinomatous disease, and from surgical injuries. The pressure or stagnation from consolidation of an organ, as in pneumonia or nephritis, may first cause coagulation in the capillary veins, from which it may extend by continued deposition till it reaches the larger vessels. Dilatation, by producing stagnation of blood in the part, causes fibrinous deposits: in the veins, when these are in a varicose condition; in the arteries, where there is aneurism; and in the heart, where the cavities are dilated.

The long continuance of any chronic disease may, by weakening the power of the heart and the vessels and by lessening the respiratory force which aids the circulation, produce what has been called marasmus or debility thrombosis. In this way, clots form in the sinuses of the brain in chlorosis, and also in other cachectic diseases where the blood is detained by wasting of the cerebral substance. Thus, too, thrombi form in the veins as a consequence of febrile diseases, such as typhus and typhoid, and in tubercular and carcinomatous affections.

The second group of conditions which produce clotting—those, viz., within the vessels, and where the blood comes in contact with an abnormal surface deficient in vitality—comprise phlebitis, endocarditis, endarteritis, and also the roughness of the vascular coats from degenerative changes of fatty or calcareous nature. Also the projection of any foreign body into the vessels or the blood, such as needles, bone, shot, or blood-clots themselves. The passage of pus into the vascular system acts in the same way; and, after fractures, there is sometimes absorption of marrow or fat by the veins, which becomes a source of thrombosis and pulmonary embolism. (On Fat Embolism, see *Medical Times and Gazette*, January 8th, 1876). Purulent matter may find its way into the veins, not only by the opening of an abscess into them, but also from suppurative phlebitis; the occurrence of which, though rare, cannot be questioned. There are thus two kinds of thrombi—those which are caused by mere mechanical stasis, and those which are produced by suppurative gangrenous or other inflammation in or around the walls of the veins. Thrombi of the latter kind, transported by the blood current, carry with them the embolic seeds of disease to the distant part in which they are arrested, and thus we have pyæmic thrombosis and embolism. It is not improbable that pyæmia may sometimes result from the puriform softening of fibrinous concretions formed in the heart or veins; but no doubt it far more frequently arises from those thrombi having access to the external air, and connected with wounds or with the uterus.

When we see the great variety of conditions which may thus lead to clotting of the blood, we are enabled to appreciate the importance of thrombosis in a practical sense, and in its relations to other diseases. Time presses, however, and we pass on to view the subject in some of its clinical aspects.

In doing this, I shall first direct your attention to thrombosis of the venous system, and of the right side of the heart, in causing embolism of the pulmonary artery, embolic pneumonia, etc.; 2nd, to thrombosis and embolism of the portal system and the liver; 3rd, to thrombosis of the left side of the heart and embolism of the arterial system.

1. The following case is one where a fibrinous concretion formed in the right ventricle of the heart; and the course of the symptoms showed that another coagulation subsequently took place in the left subclavian vein at its junction with the internal jugular vein. A robust young man, twenty years of age, steward of a ship, was admitted into the infirmary on November 16th; ill five weeks, and off work nine days. His legs and abdomen were dropsical, but there was no albumen in the urine. The pulse was feeble, and the heart acted with great irregularity. There were extreme cardiac distress and difficulty of breathing, which symptoms were aggravated by being raised, so that he lay with his head as low as possible. An indistinct systolic murmur was heard at the apex, which afterwards became inaudible. The dropsy disappeared from the abdomen and legs; but he had frequent vomiting, and the cardiac distress and weakness were most severe for three weeks. The pulse then became more regular and stronger, the distress at the heart abated, no murmurs could be heard, but new symptoms appeared. The left arm began to swell; he complained of pain in the left shoulder; there was tenderness on pressure above the left clavicle, œdema of the surrounding parts, and signs of effusion in the same side of the chest. From the 10th to the 18th of December, these new symptoms continued, whilst the heart symptoms abated; and on the 18th, while sitting up in bed and eating a biscuit, he suddenly expired.

On examination, an old fibrinous concretion, partially softened and broken down, was found in the apex of the right ventricle; and this must have been the cause of the unusually distressing cardiac symptoms, as the heart did not appear to be diseased. A firm fibrinous thrombus was found in the left subclavian vein, at its junction with the

jugular, which completely obstructed the former, and was the cause of the œdema of the arm. It must have formed long after the cardiac concretion; and as we can scarcely suppose that the softened particles from this could pass through the pulmonary capillaries, and then through the systemic capillaries, to form the nucleus of the concretion in the vein, it appears more probable that there may have been a morbid tendency in the blood to coagulation, the persistence of which caused the formation of the second as well as the first thrombus.

I was called to see in consultation a gentleman, about fifty-six years of age, a large robust man, whose illness began with a pain and slight swelling in the calf of the right leg, which he could attribute to nothing except a slight jerk in getting into an omnibus. This was followed by severe pain in the abdomen, then pain in the chest, and pneumonic signs in the right lung, and expectoration of blood and sputa mixed with blood. The left leg next began to swell, and both continued painful, œdematous, and the superficial veins were hard and painful to the touch. He sank in about three weeks with increasing pulmonary obstruction, pleuritic effusion, and œdema of the legs. No examination could be obtained.

The next case was so similar to the preceding in the succession of symptoms, as well as in the slightness of the injury from which it was supposed to arise, that I ventured, from the description given by the medical attendant, to suggest before seeing the patient the probability of its being a case of venous thrombosis and embolic pneumonia.

An active, healthy-looking gentleman, forty-five years of age, rode out ten miles on horseback. The horse, being spirited, gave him trouble; and he felt stiff in his legs after the exertion. Next day, there were slight redness and pain on the inside of the right leg, with œdematous swelling. This was followed by rigor and pain in the right side of the chest, cough, and expectoration of bloody mucus more fluid than the rusty expectoration of ordinary pneumonia. When I saw him on the third day, there were some dulness on percussion, and rather coarse crepitation over the lateral and central portion of the right lung, with moderate febrile disturbance; but the right leg had also begun to be swollen, with painful streaks of redness in the course of the internal saphena vein, and pain also on pressure in the groin. The embolic pneumonia quickly subsided, but the venous obstruction increased, and required surgical treatment. The case passed, therefore, from my care and observation; and, after having made partial recovery, the gentleman died about four months after his first attack, ulceration of the cartilages of the right ankle having supervened after erysipelas, on account of which he underwent amputation.

In the first of these cases, the thrombosis could not be traced to its cause; and the injury or cause to which it was attributed in the two other cases was of so trifling a nature that we can scarcely doubt that there must have been some predisposing condition in the constitution or the blood itself.

Sir James Paget has shown that similar cases occur in persons of gouty constitution, and he inclines to the view that the coagulation is caused by gouty phlebitis. He tells us it affects chiefly the superficial veins of the lower limbs, and shows a disposition towards being metastatic and symmetrical, characters which, in his opinion, are strongly in favour of the belief that the essential and primary disease is not a coagulation of the blood, but an inflammation of portions of the venous walls. On the other hand, however, we must not forget that, whilst phlebitis is a cause of coagulation, thrombi likewise produce adhesive phlebitis.

Mr. Prescott Hewett, Dr. Tuckwell, and Dr. George Johnson have likewise observed the occurrence of thrombosis in the gouty condition. Mr. Hewett observes: "In a very few cases only did the clotting of the blood appear to be connected with an injury—a slight contusion, a sudden strain of the limb. In some cases, it followed fever, especially typhoid fever; and in one remarkable case, in which both external iliac veins and both axillary in the course of months became permanently blocked, it followed small-pox. In most cases, the patients were over forty. In three of his cases, there was embolism, with pleuropneumonia. The limb usually remained more or less swelled, but one gentleman recovered so as to return to deer-stalking."

Dr. George Johnson (*BRITISH MEDICAL JOURNAL*, November 30th, 1872) found in two of his cases that the symptoms of cardiac thrombosis preceded those of venous obstruction, resembling in this respect the first case I have detailed; and from this he infers that fibrinous debris from clot in the pulmonary artery may pass through the capillaries of the lungs and thence into the arterial capillaries, and so reach the veins of the limbs. I have, however, already observed that we can more readily conceive the occurrence of secondary venous clotting from continuance of the original cause than from the passage of the solid particles from the debris of fibrine through two sets of capillaries.

It has been observed that in chlorosis the state of the blood predisposes to thrombosis; and a case came under my observation at the Infirmary this summer, where there was a great amount of clotting in many of the veins, and apparently confined entirely to the venous system. There was also clear evidence from the symptoms of two distinct attacks of clotting in different parts of the venous system. A pale, delicate girl, aged 21, was admitted on June 28th. She had not menstruated for two months; and, after bathing in the sea, was seized with pains in the lower part of the abdomen and the sacrum. The left leg, and then the right, became painful, hard, and swollen. She had afterwards an attack of dyspnoea. On July 14th, head symptoms suddenly showed themselves; she became unconscious; hemiplegia on the right side followed; and she died on the 17th. A great amount of blood-clotting of dark colour was found in both femoral veins, in those behind the uterus and bladder; and where the iliacs terminated in the inferior vena cava there was a firm whitish old thrombus adherent to the venous walls; higher up, the thrombus was softer and of a dark colour; but, still nearer the heart, it became again white and firm, with a conical termination. There were thrombi in some of the branches of the pulmonary artery, and a small portion of lobular pneumonic consolidation in the left lung. A branch of the pulmonary vein from the left lung was obstructed by a firm thrombus, and soft recent concretions were found in the left auricle and ventricle. No infarction of the viscera or appearance of arterial embolism was discovered. There was a firm white thrombus obstructing the straight sinus of the brain; and the veins on the surface of both hemispheres were filled with dark clots. There was an extensive softening and diffusion of dark clots of blood in the left hemisphere, and similar less advanced appearances on the right side.

What was the cause of the venous clotting in this remarkable case? Did it arise from phlebitis, seeing there was some pelvic irritation, or from some condition of the blood itself? I take the latter view, and partly for this reason, that there was a second clotting in the cerebral veins, the symptoms of which were separated by about fifteen days from those marking the first attack in the pelvic and femoral veins.

In the medical journals, and more particularly in the *Transactions* of the Pathological Society, there have been published numerous cases illustrating thrombosis and embolism; and Lancereaux gives, in his work, *Atlas d'Anatomie Pathologique*, many cases which throw light on the subject. In two cases, he shows the danger from sudden movement or excitement in displacing venous thrombi, and thus causing them to be carried into the pulmonary artery.

A woman sixty-six years of age, who had thrombosis of the femoral vein and pneumonia, from which she was recovering, got out of bed in a passion, and died suddenly from embolism of the pulmonary artery. Another woman, twenty-three years of age, was suffering from venous thrombosis and oedematous thickening of the left leg, but was better and about to be discharged. After a fit of laughing ("à la suite d'un accès de rire"), she died quite suddenly. The popliteal and tibial veins were found obliterated with clots; and embolic concretions from the femoral vein were found in the right ventricle, and also obstructing the branches of the pulmonary artery. Dr. Browne has reported (*Lancet*, June 27th, 1874) the case of a man suffering from an injury to a varicose vein, for which an elastic stocking was applied. In running a short distance to avoid a shower, he was seized with faintness and dyspnoea, and died within three-quarters of an hour, from complete obstruction of the inferior vena cava. These cases show the importance of complete rest in the treatment of thrombi, and the danger of displacing them from the situation in which they have formed. Lancereaux has given a series of seventy cases showing with what diseases thrombosis and embolism of the venous system and right side of the heart are most frequently associated, from which it would appear that they are most frequently met with in connection with carcinomatous, tubercular, and scrofulous diseases, the puerperal condition, varicose veins, and syphilis. In six of these seventy cases, death occurred suddenly.

Disease of the heart is a frequent source of concretions in the right cavities, especially in the auricle, leading to pulmonary embolism; and this is one of the ways in which cardiac disease may prove suddenly fatal. Lancereaux tabulates eighteen cases to show the cardiac affections most prone to produce these concretions. In seven, there was contracted mitral orifice; in two, insufficiency of the mitral valve; and in nine, dilatation of the right side of the heart from pulmonary lesion.

In the puerperal condition, there is not only an increased quantity of fibrin in the blood, but with this there is also venous stagnation from pressure and dilatation—a combination of causes prone to induce thrombosis; and it is well known to obstetric practitioners that some of the most formidable accidents consequent on delivery are due to

thrombosis and embolism. Dr. Barnes and Dr. W. S. Playfair have both described, in papers in the *Transactions* of the Obstetrical Society, the appalling symptoms of dyspnoea, etc., which from this cause suddenly attack patients after confinement. They do not, however, differ materially from the symptoms which mark the occurrence of complete or partial pulmonary embolism from other causes. Dr. Barnes has shown that the puerperal condition disposes to clotting of blood in the arterial as well as the venous circulation, and in two of his cases the veins and arteries were both obstructed.

When a fibrinous clot from the peripheral veins or from the right side of the heart completely obstructs the pulmonary artery, death must of course follow rapidly; and the same result must ensue where the larger branches are completely obstructed by smaller clots. But, when some of the smaller branches only are plugged, the parts supplied by the vessels become the seat of embolic pneumonia or infarction, formerly known as pulmonary apoplexy. Recovery not unfrequently takes place from this condition, the dark wedge-shaped portions of consolidated lung undergoing fatty transformation and absorption; but, on the other hand, the pneumonic abscesses which result from septic emboli are probably always fatal.

I must necessarily be brief with the two remaining divisions of my subject, each of which would suffice for a single paper or address.

2. Thrombosis and embolism of the portal system and liver have been very fully investigated by Frerichs, who has shown that the same causes which operate in the venous system act here too. Thrombosis and embolism of the vena porta are, perhaps, not unfrequently overlooked for the same reason that they are not observed in other organs; viz., because the organs are more carefully examined than the vessels. But the study of pathology has become a laborious work, and microscopic examination is also necessary to discover the smaller capillary embolisms. If we are, therefore, to advance pathological knowledge, we must continue to uphold our Microscopical Section, and to encourage those who devote their energies to this laborious work.

3. The third and last branch of our subject comprises thrombosis of the cavities of the left side of the heart, thrombosis of the arteries, and embolism of the branches of the arteries; also the effects of embolism on the viscera and limbs in producing infarction, softening, gangrene, and loss of function.

Concretions form in the cavities of the left side of the heart from conditions similar to those which produce them in the right side, especially in the left auricle from contraction of the mitral orifice; and Lancereaux describes two cases of concretions in the auricle from mitral contraction consequent on acute rheumatism. He has also reported two cases of fibrinous concretions in the left ventricle. In one, where this cavity was much hypertrophied, there were five concretions, varying from the size of a nut to a pigeon's egg. The larger, adherent to the internal surface, were softened at the centre, and contained a milky-looking fluid. In the other, a case of weak, fatty, dilated heart in an intemperate man, there were also several concretions embedded in the fleshy columns, which were softened in the centre.

We know that the left side of the heart is liable to endocardial inflammation, and to valvular affections, which rarely affect the right side; and these conditions cause the formation of smaller fibrinous concretions, which are deposited like granulations on the edges of the valves; and they, as well as the larger concretions resulting from stasis, are a frequent cause of embolic obstructions in the arteries. Rheumatic fever is known to be one of the great causes of endocarditis, and it may produce either valvular contraction or these warty concretions on the edges of the valves. There is, however, another form of endocarditis—the ulcerative—from which embolic matter may be carried away; and this is much more injurious in its effects, being of septic or pyæmic nature; and Lancereaux, who has collected eleven cases, tells us it always proves fatal.

With regard to arterial thrombosis, concretions form less commonly in the arteries than in the veins; but they are produced, as in aneurisms, by dilatation and contraction of the vessels, and also by degeneration of the coats. Last session, Dr. Davidson brought before this Society a case of fibrinous obstruction of the arteries of the brain, which were thickened by syphilitic disease, and this he regarded as thrombosis. Dr. Bristowe (*Transactions* of the Pathological Society) reports a similar case, where the right hemisphere was softened from obstruction of the middle cerebral artery. He has also reported seven cases of fibrinous deposits in the arteries of the brain, which, he thinks, were not of embolic nature, and must have been due to spontaneous coagulation of blood or local arteritis. In the arteries as well as in the veins, there must often be this difficulty in deciding whether a fibrinous clot has formed in the situation where it is found, or has been transported from a distant part. In

either case, however, the effects on the obstructed organ or part will not be materially different.

With respect to arterial embolism, not only the large concretions which form in the left auricle and ventricle, but also the smaller ones which are deposited on the valves, as well as the degenerating matters which escape from both forms of concretions, are liable to be swept away by the blood-current to form embolic plugs in the arterial branches. The clots also which form on the coats of diseased arteries, and likewise the fatty or calcareous matters which form there or on the valves, are carried away and block up the arteries of the spleen, the kidneys, the brain, or the limbs; the relative degree of liability to be affected being in this order, and being due to the particular conditions of the circulating system of these organs.

I need not describe the appearances of infarction and softening presented by each of those organs in consequence of embolic obstruction. We are all, however, I believe, in the present day aware of the fact that embolic obstruction of the arteries of the brain is a frequent cause of softening and paralysis, which cannot be removed by any active depleting and counter-irritant treatment, but is more likely to be benefited by rest and gentle stimulation. And I would remark that, just as Paget, so far back as 1844, drew attention to coagulation of the blood in the pulmonary artery, ably illustrating the subject by a series of carefully observed cases, so also Dr. Kirkes was, in 1852, the first to demonstrate clearly by another series of cases that fibrinous deposits in the interior of the left side of the heart are a frequent cause of softening of the brain, which always results when the obstruction is beyond the circle of Willis, owing to the absence of anastomosing branches. In his very original paper, he also illustrated the effects of embolic obstruction in producing infarctions of the spleen and kidneys, and also the violet-coloured petechial spots on the mucous membrane of the stomach and bowels, which are produced by the same cause.

I have condensed from the Infirmary Clinical Medical Prize Reports of this year a case which occurred lately in the Infirmary, which shows well the close connection between valvular disease of the left side of the heart and cerebral softening.

A woman, twenty-one years of age, admitted with disease of the heart consequent on rheumatic fever, became suddenly unconscious, with right hemiplegia, the day before her death. The middle cerebral artery was found plugged by white fibrine; and on the mitral and aortic valves were found numerous fibrinous and calcareous vegetations, which showed the source from which the embolic matter was derived. The cause of the obstruction in this case is sufficiently clear; but in another case, taken from the same reports, and which occurred in the hospital practice of my colleague Dr. Glynn, the cause of the arterial obstruction was more obscure, and was probably from thrombosis, seeing that there was also venous thrombosis.

A girl, nineteen years of age, was carried helpless into the Infirmary on January 4th, having had a sudden seizure, followed by left hemiplegia and anæsthesia, which had been preceded by pain in the right side of the head. She died on March 13th. There was extensive softening of the right middle cerebral lobe. The right internal carotid artery was plugged by what appeared to be an embolus at the bifurcation of the anterior and middle cerebral arteries; the higher part being firm and of yellowish colour; but lower down, of dark brown colour, and having a conical termination in the external carotid. The thyroid body was enlarged; the heart sound. There was also a thrombus in the lower end of the vena cava, and in the left common and external iliac veins adherent to the walls.

Here, again, we have coagulation of blood in both the arterial and the venous system which has been observed in so many other recorded cases.

I might have alluded to some other forms of embolism, to the effect of embolism of the coronary artery in arresting the heart's action, to the effect of plugging of the central artery of the retina in destroying vision, and to the connection between embolism and paraplegia, chorea, and some other nervous affections. I might also have alluded to the view taken by Dr. George Johnson, that the lobular pneumonia which often follows extensive burns arises from the passage of coagulated blood from the injured parts to the pulmonary vessels and the duodenal ulcers from a similar cause. We can readily understand the wide range of effects produced by the blood distributed throughout the whole system; but yet we must be careful in not ascribing to the effect of embolism anything that is not proved by rigid observation. Sufficient has, however, now been adduced to show how great are the consequences that may result from the coagulation, the lessened vitality, or it may be the death of a minute portion of the blood passing through the heart or circulating in a vein or an artery, and to prove the importance of a knowledge of this subject to all who are engaged in the practice of medicine or surgery.

BULBAR PARALYSIS.

BY THOMAS S. DOWSE, M.D., F.R.C.P.E.,

Physician Superintendent of the Central London Sick Asylum, Highgate.

[Concluded from p. 583 of last number.]

BEFORE entering upon the pathology of this disease, I wish to draw attention to two typical cases, which are at the present time under my care—one of complete cure, and the other a retrogressive case of reflex bulbar paralysis.

CASE III. *Bulbar Paralysis of five years' duration: Cure.**—Daniel Megan, aged 22, of neurotic tendency, and with a history of convulsive seizures in infancy, had fairly good health in childhood, but became subject to a series of epileptic fits when sixteen years of age. These were succeeded by paralysis of the seventh, eighth, and ninth nerves, and by increasing failure of motor power all over the body. He came under my care three years afterwards. When first seen by me, the countenance was calm and intelligent; he was unable to bring the lips together, to spit, or blow out a candle. The lower jaw could not be fully depressed, neither could the lateral movement of mastication be effected. Saliva was constantly dribbling from the mouth, and the tongue lay flaccid, furrowed, and helpless behind the lower row of

Fig. 4.

teeth. He was able only, in a very limited degree, to retract it or to move it in a lateral direction. The want of power in deglutition showed itself chiefly, though not entirely, in the first act, which was performed apparently with great difficulty. The palate muscles were more or less involved, and he would occasionally suffer from partial asphyxia, from particles of fluid entering the larynx. There was some inhibition of the vagus, evidenced by respiratory and cardiac irregularities; and, like many other cases, his head seemed fixed so that he moved it with difficulty. This, doubtless, arose from palsy of the spinal accessory nerve, whose origin, according to Dr. Lockhart Clarke, is intimately associated with that of the vagus and hypoglossal. The vocal chords were found to act feebly. He was quite dumb, and unable to make a sound. There was complete motor paralysis with impaired sensation of the upper limbs, and entire paralysis of the lower to motion, and partially to sensation. The muscles of the latter were greatly atrophied, especially the extensors, so that the heel was drawn up, as in talipes equino-valgus. There were no tremors or muscular fibrillations. There was dulness at the apex of the right lung, with crepitation. Altogether, I thought the case most unfavourable, so I merely fed him well, and gave him steel, with cod-liver oil. He gained flesh, and his general health improved; saliva ceased to flow from the mouth; the act of deglutition was performed fairly well; the palate and vocal muscles also gained power; yet the tongue remained as motionless as ever.

In December of last year, I determined to galvanise the tongue regularly; and, to show how much its sensibility was impaired, I could get no response to an induced current of Stohrer's two-celled battery. I persevered daily for three weeks, and at the end of this time electric sensibility was aroused; and, to his own surprise as well as mine, he protruded the tongue, and went through the alphabet, with the exception of R, O, F, P, W, and S. The following day, he was unable to protrude the tongue until it was galvanised; but from this time he exercised voluntary control over it, and he now has perfect use of lips and tongue, can swallow, and speak with almost perfect accuracy. The tendo Achillis has been divided, and by the daily use of the constant current, and injection of a quarter of a grain of strychnine, he is fast regaining the use of his lower limbs. In this case, which is of unusual interest (and in English medical literature I do not find another recorded), we have good evidence of lesion of the medulla, and possibly

* Patient shown at Clinical Society last session.