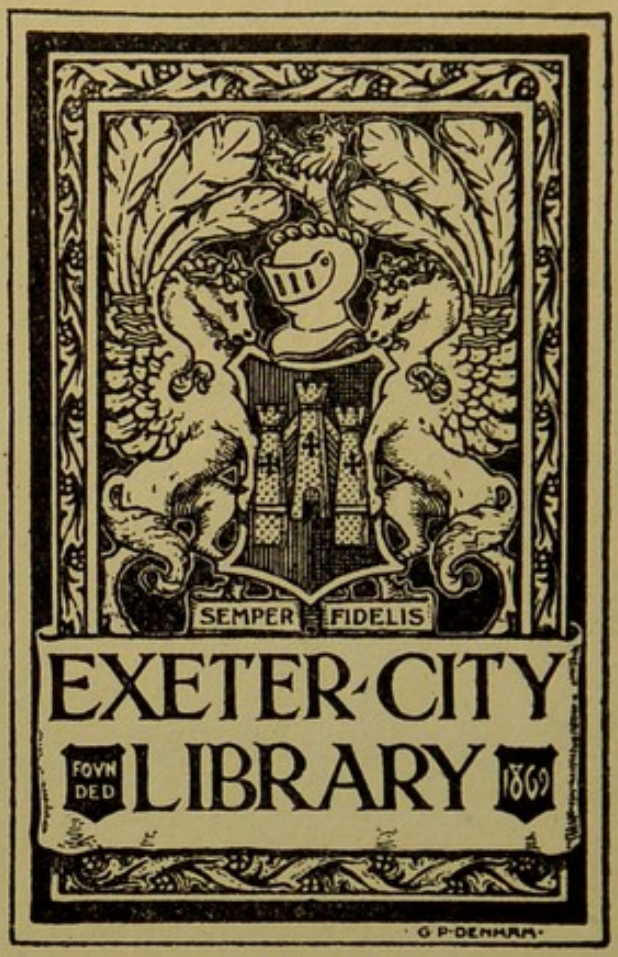
**Phlebitis and thrombosis / by Warrington Haward.**

# PHLEBITIS AND THROMBOSIS

*WARRINGTON HAWARD*

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# PHLEBITIS AND THROMBOSIS

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## The Hunterian Lectures

DELIVERED BEFORE THE ROYAL COLLEGE OF SURGEONS OF ENGLAND IN MARCH, 1906

BY

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# LECTURE I

Causes of thrombosis.—Disturbances of physiological relations between blood and vessels by: (1) Changes in vessel—(*a*) traumatism; (*b*) inflammation; (*c*) degeneration; (*d*) dilatation. (2) Changes in blood—(*a*) invasion of micro-organisms; (*b*) physical changes; (*c*) chemical changes. (3) Retardation or arrest of blood-current.

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Septic phlebitis.—Phlegmasia dolens.—Pylephlebitis.—Suppurative phlebitis.—Symptoms of septic phlebitis.—Sources of infection.—Treatment.

MR. PRESIDENT AND GENTLEMEN,—It has always seemed to me that our interest in a disease should be in proportion, not to the rarity, but to the commonness of its occurrence. It is true that the careful investigation of an unusual affection may throw a light upon some pathological process which is common to other diseases, and thus give a wider significance than would at first appear to belong to such observations; but surely the study of conditions which we are frequently called upon to treat must possess

for us a still greater importance. I have therefore ventured to devote the lectures which I have the honour to deliver here to the subject of thrombosis and phlebitis, conditions which are of no uncommon occurrence, and concerning which there yet appear to me to be some points worthy of consideration.

I am the more encouraged to ask your attention to this topic because, having for some years been making and collecting observations relating to thrombosis, I have found that the remoter effects of the disease do not seem to have attracted much attention from surgeons.

Coagulation of blood within the living vessels occurs under a variety of conditions, of which the mode of origin is in some cases well understood and sufficiently obvious, while of others it is obscure and difficult to explain.

A thrombus may be the result or the cause of inflammation of the containing vessel; it may be determined by a change in the blood or by a change in the vessel which contains the blood.

As the fluidity of the blood and its proper circulation depend upon the maintenance of the normal physiological relations between the blood and the vessels, conditions which disturb these relations will give rise to thrombosis. The most obvious of such disturbing conditions are traumatic. A wound, laceration, or contusion of a blood-vessel may lead to the formation of a thrombus by interfering with the integrity of the vessel wall. Other injuries, such as burns and fractures, neighbouring irritation or suppuration, or anything giving rise to inflammation of the vessel, are causes of thrombosis. So also degeneration of the arterial or venous coats, dilatations, aneurisms, and varices, pressure or other causes of retardation or arrest of the blood-stream in the vessel and in the vasa vasorum, may give rise to the formation of a thrombus.

Other causes of thrombosis are changes in the blood, of which some of the most important are due to the invasion of micro-organisms. There is an increased tendency to coagulation of the blood in anæmia and chlorosis; in phthisis and other debilitating diseases; in certain acute diseases; after fevers, hæmorrhage, and long-continued suppuration; and in conditions of exhaustion.

The process of coagulation has been much discussed, and the relative importance of the factors in its production is still undetermined. The presence of fibrinogen and fibrin ferment is certainly essential, and probably also the presence of a soluble salt of calcium; but the source of the fibrin ferment remains a matter of dispute. Cohnheim and others have maintained that the leucocytes play the chief part in the process of thrombosis; Schmidt of Dorpat and his followers stating that the leucocytes by disintegration form fibrino-plastin or paraglobulin and the ferment, the fibrinogen existing naturally in the blood-plasma; while, on the other hand, Eberth and Schimmelbusch, followed by numerous other observers, attribute the greater importance to the blood-platelets.

This much may, I think, with some confidence be stated: that healthy blood contained in normal vessels consists of a fluid (the plasma) which holds in solution a proteid material (fibrinogen). In this fluid are suspended the red corpuscles, the various colourless corpuscles, and the blood-platelets. That if the normal relations between the blood and the vessels are disturbed, coagulation may occur, and this involves the appearance in the blood (probably from changes in the colourless corpuscles and platelets) of a nucleo-proteid, which, with a soluble salt of calcium, forms fibrin ferment; and this, acting upon the fibrinogen of the plasma, leads to the formation of fibrin, which, with the entangled corpuscles, forms the clot.

There has been a good deal of difference of opinion as to the origin and function of the platelets, some observers maintaining that they do not exist in normal blood, but are the result of the disintegration of the corpuscles; others that they may exist in normal blood, but that they are derivatives of the leucocytes or of the red corpuscles; others, again, that they are the progenitors of the red corpuscles. But the tendency of recent researches has been to regard the platelets as independent elements of the blood, and to attribute to them an important influence in the process of coagulation. The evidence in this direction seems to me to be convincing.

Although the blood-plates were described as long ago as 1842 by Donné, in a paper on the origin of the corpuscles of the blood,[1] and various theories concerning them were promulgated from time to time, it was Bizzozero's[2] paper, published in 1882, which aroused a fresh interest in these bodies and led to much new investigation. Bizzozero maintained that the blood-plates were definite and regular elements of the normal circulating blood, and though this was disputed by other observers, his observations have since been abundantly confirmed.

Professor Osler,[3] in his 'Cartwright Lectures on the Physiology of the Blood Corpuscles,' published in 1886, records a number of interesting observations upon the blood-plates. He agrees with Bizzozero that they are normal elements of the blood, and he points out that their tendency to agglutinate and to undergo rapid changes when the normal conditions are disturbed renders them

[1] *Comptes Rendus de l'Académie des Sciences*, 1842, p. 366.

[2] Bizzozero, *Virchow's Archiv*, vol. xc., p. 261.

[3] Osler, 'Cartwright Lectures on the Physiology of the Blood Corpuscles,' *Medical News*, Philadelphia, 1886, p. 365. See also Osler on the 'History of Blood-platelets,' *Johns Hopkins Hospital Bulletin*, May, 1905.

extremely difficult to observe, and that this may account for the discrepancies in the descriptions of various observers. This agglutination is quite different from that of the red corpuscles; they do not form rouleaux, but become fused together in a granular mass. Professor Osler gives the relative number of the plates as about 1 to 18 or 20 of the red corpuscles (a lower estimate than most observers), and he found that the number varied in conditions of disease. In the formation of a thrombus he observed that the blood-plates were the first elements which collect upon the vessel wall, and that in the process of coagulation the fibrin filaments spread chiefly, though not exclusively, from the plate masses as centres. He saw no indication whatever of the disintegration of leucocytes.

Dr. Robert Muir,[1] in his papers on 'The Physiology and Pathology of the Blood,' published in the *Journal of Anatomy and Physiology* for 1891, has given a most careful description, with beautiful drawings, of the blood-plates, together with directions as to the best way of observing them. In fresh undiluted normal blood, examined a few seconds after withdrawal from the vessels, he found numerous blood-plates among the red and white corpuscles. He describes them as round or oval disc-like bodies, about one-fourth the diameter of a red corpuscle, slightly convex, faintly granular, and colourless. His observations show that in their staining reactions, in their structure, and in their chemical composition they differ from all the other elements of the blood, and that they have definite characters peculiar to themselves. In shed blood they rapidly undergo certain changes connected with the process of coagulation. They become adhesive, stellate, more granular and refractive, and are soon aggre-

[1] 'The Physiology and Pathology of the Blood,' by Dr. Robert Muir, *Journ. of Anat. and Physiol.*, 1891, vol. xxv., p. 256.

gated into small groups. After about three minutes the formation of fibrin commences, delicate threads of which appear round the groups of altered blood-plates. These changes can be prevented or retarded by various methods which prevent the coagulation of the blood, such as the addition of neutral salt solutions, or exposing the blood to a low temperature. On the other hand, if a foreign body is introduced into the living vessels, or if the vessel by injury or otherwise loses its normal condition, the blood-plates undergo the same changes that they do outside the vessels, adhering to each other, to the foreign body, and to the vessel wall, and becoming fused together into a granular mass which is the starting-point of a thrombus.

Professor G. T. Kemp and Miss H. Calhoun communicated some important observations with regard to the blood-plates to the International Congress of Physiologists at Turin in 1901.[1] As the result of seventy-five observations on nineteen different individuals, they estimated the average number of platelets in normal blood as 778,000 per cubic millimetre.[2] The number bore no definite relation to the number of leucocytes, but the ratio to the number of red corpuscles was fairly constant. The connection of the blood-platelets and the leucocytes with coagulation was determined by a method which the authors proposed to name 'fractional defibrination.'

A certain fraction of the calculated blood of an animal was drawn, defibrinated, filtered, and returned to the

[1] Reported in *Brit. Med. Journ.*, 1901, vol. ii., p. 1539.

[2] It will be remembered that in normal blood the number of leucocytes per cubic millimetre is about 10,000; the number of red corpuscles per cubic millimetre is about 5,000,000. Messrs. Brodie and Russell estimated the number of blood-platelets as 635,300 per cubic millimetre, or 1 : 8·5 of red corpuscles. Dr. J. H. Pratt, of Boston, United States of America (*Journ. of Amer. Med. Assoc.*, December 30, 1905, p. 1999), gives the average number of platelets per cubic millimetre as 469,000.

circulation. This process was repeated until fibrin was no longer formed. At each stage the red corpuscles, the leucocytes, and the platelets were counted. The platelets disappeared progressively with each defibrination, and after the blood ceased to be coagulable (from the sixth to the tenth defibrination) they were no longer present. The leucocytes disappeared to some extent, but never completely. The red corpuscles suffered some diminution, but much less than the leucocytes.

The platelets were seen to be bi-concave, but they were never found to contain traces of hæmoglobin, and therefore could not be regarded as hæmatoblasts in Hayem's[1] sense. Their micro-chemical reactions resembled to some extent those of red corpuscles, and also those of the nuclei of leucocytes, but were identical with neither. The authors agreed with Bizzozero that they were independent elements of the blood.

Dr. A. Petrone (of Naples) at the same Congress related researches pointing to the same conclusions as to the independence of the blood-platelets as separate elements of the blood.

Wooldridge remarks in this connection: 'Blutplättchen are regarded by all observers who have attacked the problem as definite form elements, by which I understand them to mean organized bodies; and it is difficult to know how else to regard them, for they have just as much right to be regarded as form elements as the red corpuscles have. Many attribute to these bodies, in regard to coagulation, the powers which Schmidt has referred to the white corpuscles. I think they are perfectly right.'[2]

[1] Dr. Robert Muir came to the same conclusion as to Hayem's theory (*op. cit.*, p. 500).

[2] Wooldridge, 'The Chemistry of the Blood, and other Papers,' edited by Horsley and Starling, 1893, p. 177.

But Wooldridge[1] maintained that fibrin could also be produced as the result of an interaction between two or more fluid fibrinogens or proteid-lecithin compounds of different chemical composition, of which interaction the fibrin ferment is a by-product; and he drew attention to the important part played by lecithin in the process of coagulation.

Professor W. D. Halliburton and Mr. T. G. Brodie[2] have shown that nucleo-albumin, free from lecithin, obtained from the tissues of various organs, when injected into the circulation of rabbits, produces instantaneous and fatal coagulation of the blood; and they consider it probable that in cases of fatal and extensive thrombosis occurring in the human subject, death may be due to the entrance into the blood of the same material.

> '. . . with a sudden vigour it doth posset
> And curd, like eager droppings into milk,
> The thin and wholesome blood.'[3]

Moreover, Professor A. E. Wright has proved that the addition of lymph to blood notably accelerates coagulation,[4] 'and that the administration of chloride of calcium

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After I had written these lectures, my friend Dr. G. A. Buckmaster kindly allowed me to read the proofs of part of a lecture in which he treats of the origin of the blood-platelets, and records his investigations into the subject ('The Morphology of Normal and Pathological Blood,' by G. A. Buckmaster, M.D.; Murray, 1906). Dr. Buckmaster has come to the conclusion that blood-platelets do not exist in normal living blood, but are pathological or artificial products. I have the greatest respect for Dr. Buckmaster's views, but I do not understand how his negative observations invalidate those of observers (Eberth and Schimmelbusch) who have seen the platelets in the vessels of the living mesentery, or (Laker) in the membrane of the wing of the young bat.

[1] Wooldridge, *op. cit.*, pp. 190, 241. This statement was based on observations made with 'peptone plasma,' and was controverted by Halliburton and others. See *Journ. of Physiol.*, vol. ix., p. 270.

[2] *Brit. Med. Journ.*, 1893, vol. ii., p. 682.

[3] 'Hamlet,' I. v. 68.

[4] *Journ. of Physiol.*, vol. xxviii., p. 514.

has a most marked effect in increasing the coagulability of the blood in the living subject.'[1]

Evidently, therefore, the chemical composition of the blood may have an important influence in diminishing or increasing the tendency to coagulation.

Wooldridge[2] showed that the injection of laky blood—*i.e.*, blood in which the red corpuscles have been dissolved—gives rise to intravascular clotting, and that the active agent is not, as used to be thought, the hæmoglobin, but the stromata of the red corpuscles, the protoplasmic framework in whose meshes the hæmoglobin is contained, and which is a complex proteid-lecithin compound.

Wooldridge's work in relation to the chemistry of coagulation is extremely suggestive, and although his conclusions are not all universally accepted, his papers are well worth careful study. The chemistry of the blood is an investigation of extreme difficulty, because directly the blood is removed from the bloodvessels it undergoes important changes; so that it is difficult to reason from experiments in the laboratory to the vital chemistry of the living body.

As Wooldridge said: 'Genetically considered, both blood and endothelium are differentiations of one and the same protoplasmic mass. The blood is merely the more fluid central part of the originally solid protoplasmic cord. The blood and the vascular wall may, then, be looked upon as a protoplasmic unit. That the vascular wall exerts a great influence on the blood is evident from the fact that the blood undergoes changes which finally terminate in coagulation, so soon as it leaves the vascular wall.'[3]

[1] *Brit. Med. Journ.*, July 29, 1893, p. 223, and July 14, 1894, p. 57: *Lancet*, October 14, 1905, p. 1096.

[2] Wooldridge, 'The Chemistry of the Blood,' p. 167.

[3] *Ibid.*, p. 180.

Hunter[1] said: 'It must be evident that the fluid state of the blood is connected with the living vessels, which is its natural situation, and with motion.' And again: 'While the blood is circulating it is subject to certain laws to which it is not subject when not circulating.'[2]

Recent investigations show that the first stage in the formation of a thrombus is the accumulation and viscous change of the blood-platelets, which adhere to each other and to the wall of the containing vessel; to these are soon added numerous leucocytes; fibrin ferment is set free, and fibrin appears, entangling more or less of the red corpuscles.

It is evident that this process will be favoured by a retardation of the blood-stream; while, on the other hand, in the large arteries near the heart, where the current is powerful, small quantities of clot will be easily swept away, and a thrombus less easily formed. This, moreover, is in accordance with clinical experience, for thrombosis is more common in veins than in arteries, and it is specially prone to occur in positions in which the circulation is apt to be impeded.

Moreover, von Recklinghausen has directed attention to the manner in which thrombosis is favoured by the eddies produced in the blood-stream by cross currents, irregularities of surface, and variation in the size of the vessels, and by obstruction to the blood-flow. Retardation or arrest of the blood-current, although favouring the occurrence of thrombosis, is not in itself sufficient to cause it; for it has been shown by Baumgarten that healthy blood will remain fluid for weeks or months, though shut off from the circulation in a vessel between two carefully applied aseptic ligatures.

[1] 'Treatise on the Blood,' etc., p. 24, 1794.

[2] *Ibid.*, p. 85.

But free circulation is necessary for the nutrition both of the blood and the vessels. Slowing of the current, therefore, by diminishing the normal influence of the lining membrane of the vessels upon the blood-cells, and by lowering their vitality, may set free the fibrin ferment and thus lead to thrombosis.

Eberth and Schimmelbusch,[1] moreover, have observed that in the normal blood-current the platelets travel with the red corpuscles in the axial stream, while the white cells move in the outer, slower stream. A slight retardation leads to an increase in the number of white cells in the outer zone, but not to thrombosis; but on a further slowing of the current the platelets are found in the peripheral stream and in contact with the endothelium, and it is upon these collections of platelets that the fibrin is first deposited which is the commencement of a thrombus.

It would seem, then, that thrombosis may depend upon a variety of conditions, and is usually due to a combination of several.

Lesions and degeneration of the vessel walls, impaired nutrition of the endothelium, retardation of the blood-current, changes in the composition of the blood and in the proportion of its formed elements, the invasion of micro-organisms—any or all of these may play a part in the process, the predominant factor varying.

If the movement of the blood in a vessel is arrested, as by a ligature, the coagulum formed from the stagnating blood consists of fibrin, together with both red and white corpuscles, and resembles microscopically a clot formed in blood outside the vessels; it is of red colour and is spoken of as a *red thrombus*.

If the thrombus is formed from blood in motion it is usually of gray colour (the so-called *white thrombus*), and

[1] *Virchow's Archiv*, vol. ciii., p. 39.

consists of fibrin, leucocytes, and blood-platelets. A clot thus formed may, however, entangle a variable number of red corpuscles, which will give to it a more or less red colour.

Professor Welch[1] has described the characters of thrombi produced experimentally from the circulating blood: 'The material composing the youngest of such thrombi appears macroscopically as a soft, homogeneous, gray, translucent substance of viscid consistence. Microscopically, it is made up chiefly of platelets, which are seen as pale, round, or somewhat irregular bodies, varying in size, but averaging about one quarter the diameter of a red corpuscle.' Then, leucocytes collect at the margins of the platelet masses and between them. 'These leucocytes are nearly all polynuclear, and usually present no evidence of necrosis or disintegration.' With the accumulation of leucocytes, fibrillated fibrin makes its appearance at the margins of the masses of platelets. At the end of half an hour a thrombus may be composed of platelets, leucocytes, and fibrin, with entangled red corpuscles.

Professor Osler[2] also examined the structure of white thrombi, and found that they consisted chiefly of blood-plates. The thrombi, for instance, which form upon rough patches upon the aorta, are, he says, 'without exception composed, not of colourless corpuscles, nor of a reticulated fibrin network, but almost exclusively of plaques, which in the deeper parts have undergone granular disintegration, but in the superficial parts still retain their normal shape and appearance.' Bizzozero and Hayem have confirmed this statement. The softening which occurs in the centre of these thrombi is due

[1] 'The Structure of White Thrombi,' *Trans. Path. Soc. of Philadelphia*, 1887, p. 281.

[2] 'Cartwright Lectures on the Physiology of the Blood-Corpuscles,' *Med. News* (Philadelphia), 1886, pp. 365, 421, 424.

FIG. 1.—OBLITERATING ARTERITIS: SHOWING COATS OF VESSEL MUCH THICKENED, AND ORGANIZING AND ADHERENT CLOT.

[*To face p.* 13.

to the liquefaction of the blood-plates. Such a thrombus, starting in a vein from some point of injury, or, as often happens, forming above the point of attachment of a valve, extends to a variable distance chiefly in the direction of the blood-current. Thrombosis may be simultaneously of wide extent, or the process may spread gradually along the vessel towards the heart, obstructing long tracts of vein and giving rise to very extensive propagation of the thrombus. Extensive thrombi thus formed will present alternations of white and red thrombus. The original white thrombus, extending across the vessel, forms a barrier to the blood-current; stagnation occurs as far as the next branch, and thus a red thrombus is added to the white; upon this the circulating blood deposits fresh white thrombus, until stasis and coagulation again take place.

A third variety of thrombus is the *hyaline thrombus*, which consists of colourless, translucent, homogeneous material, which stains after the manner of fibrin. This is found chiefly in the capillaries and small vessels, and is usually associated with infective diseases.

The small vessels of acutely inflamed tissues may be found obstructed by thrombi consisting almost entirely of polynuclear leucocytes; or sometimes, as in hepatized lung, the vessels may be occupied by purely fibrinous coagula. But these are of *inflammatory origin*, and of quite different significance to the thrombi above described.

If there is no infection of the blood a thrombus may become organized, or it may undergo disintegration and be carried away in the blood-stream without doing any harm.

The organization of a thrombus is accomplished by the shrinking and cracking of the clot, into the fissures of which leucocytes pass from the vessel wall, and capillaries extend from the vasa vasorum. By this means the clot

is gradually absorbed, and its place taken by newly-formed connective tissue, which is adherent to the lining of the vessel, and which, shrinking after the manner of scar tissue, becomes less and less vascular, until only a fibrous cord remains (Fig. 1).

The thrombus, thus organized, may adhere in a layer of varying thickness to the vessel wall, or may form a plug completely occluding the vessel; or a channel may be formed through its centre, or between the clot and some part of the vessel wall, leaving the vessel with a thickened wall and diminished lumen (Fig. 2).

If disintegration of a thrombus occurs, softening begins in the centre or at the end of the clot, and extends until the whole is broken up into a finely granular débris, which is carried away in the circulating blood.

This is a process which is frequently to be observed, especially in varicose veins, and does not usually give rise, when the clot is not infective, to any serious symptoms; but if the whole, or a considerable fragment of a clot, becomes dislodged and carried into the circulation, dangerous embolism may occur, with results that will be described later on.

Softening commencing in the centre of a thrombus does not always lead to its complete disintegration, but may result in the production of a thick, greasy, brown fluid, completely shut in by the outer layers of the thrombus. The enclosed fluid, somewhat resembling, and formerly mistaken for, pus, consists of the granular and oily débris of the broken-down corpuscles, coloured by blood pigment.

Occasionally calcareous changes occur in thrombi, giving rise to phleboliths, concretions consisting principally of phosphate of lime with about 20 per cent. of proteid matter, and a little sulphate of lime and potash. Although

FIG. 2.—ARTERIAL THROMBOSIS: COATS OF ARTERY NOT THICKENED, BUT VESSEL CONTAINING ADHERENT AND CHANNELLED CLOT.

[*To face p.* 14.

sometimes met with in arterial thrombi, they are found chiefly in the dilated pouches of the veins of the lower extremities and pelvis.[1]

In septic thrombi softening, disintegration, and true suppuration may occur ; and fragments of the infected thrombus are carried in the blood-stream to distant parts, where, as septic emboli, they become centres of fresh infection.

In the heart, thrombi form upon the inflamed valves, forming the vegetations of endocarditis. Thrombi are also found in the cavities of the heart, especially in the auricular appendices, sometimes attached and sometimes free, in the latter case giving origin to the so-called 'ball thrombi.'[2]

Cardiac thrombosis, however, falls under the care of the physician, and I shall not further allude to it.

## Arterial Thrombosis.

This is met with in connection with wounds, injuries, and degenerations of the arterial walls. It may occur gradually, as in aneurisms, or suddenly, as a result of embolism or other mechanical obstruction to the blood current.

It is also caused by chronic or acute arteritis, such as is

[1] Dr. Rolleston has recorded an instance of numerous small phleboliths under the skin of both shins of a woman aged fifty. The phleboliths formed discrete, hard, painless, movable nodules, of the size of minute shot or less ; they were not attached to the bone or to the skin, and they somewhat resembled the subcutaneous nodules seen in connection with the acute rheumatism of childhood. See the *Lancet*, January 6, 1906, p. 29.

[2] For description of which see Dr. J. W. Ogle in *Trans. of Pathol. Soc. of London*, vol. xiv., p. 127, 1863. See also Dr. Wickham Legg, *ibid.*, vol. xxix., p. 49, 1877, and St. George's Hospital Museum, Series vi., 47*f*.

seen in the course of syphilis and other infective diseases, and is occasionally met with in cases of anæmia and wasting diseases, in which no changes in the arterial walls can be detected. In wounds and injuries of arteries the resulting thrombus is caused partly by mechanical obstruction to the blood-stream owing to the projection into the lumen of the inner coat, and partly in consequence of the interference with the normal influence of the intima. The same may be said of the application of a ligature. In the case also of atheromatous and other degenerations of the arterial coats, and in aneurisms, the destruction of the intima and the roughening of the inner surface of the vessel are largely concerned in the deposition of mural clot, but some retardation or irregularity in the blood-current seems to be also necessary.

In obliterating arteritis the obstruction of the vessel, though partly due to the arterial sclerosis, is also (as has been shown by Zoege-Manteuffel)[1] largely produced by the deposition and organization of blood-clot. No doubt this condition is frequently due to syphilis, but it also occurs in the course of other infective diseases, as the specific fevers, rheumatism, influenza, and pneumonia. Rare instances are also recorded of arterial thrombosis in connection with phthisis, and Professor Welch[2] describes a specimen showing a tubercular focus in the intima of the aorta, on which a thrombus has formed containing tubercle bacilli.

Besides these changes in the arterial wall, certain conditions of the blood, in combination with a slow and feeble circulation, will lead to coagulation in the arteries; as is seen in chlorosis, anæmia, and towards the end of

[1] W. Zoege-Manteuffel: *Deut. Zeit. f. Chir.*, 1898, vol. xlvii., p. 461.

[2] Allbutt's 'System of Medicine,' vol. vi., p. 196, 1899.

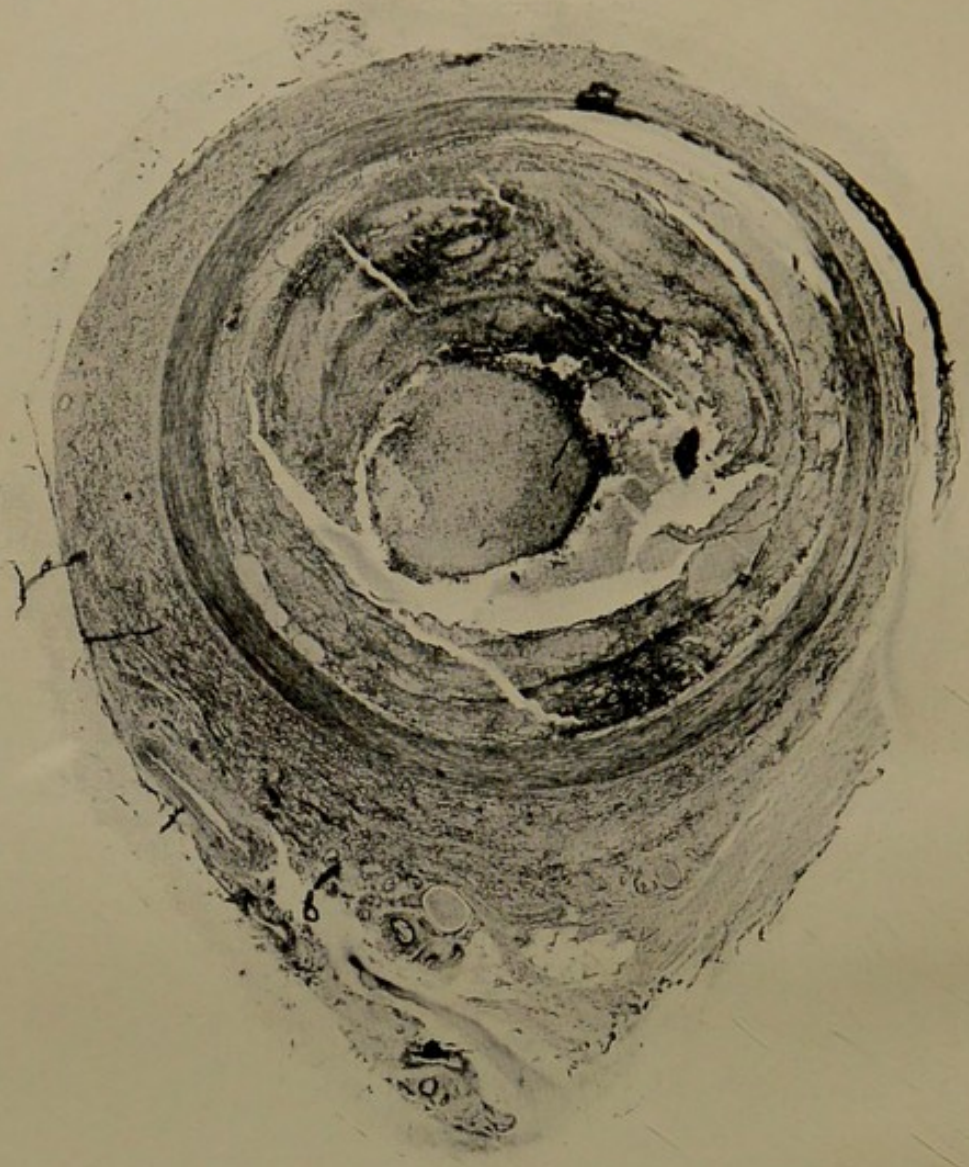

FIG. 3.—SECTION OF BRANCH OF FEMORAL ARTERY FROM A WOMAN, AGED TWENTY-THREE YEARS, WHO DIED FROM EXTENSIVE THROMBOSIS OF THE PULMONARY ARTERY: THE ARTERY FULL OF CLOT; COATS NATURAL.

[*To face p.* 17.

exhausting diseases, but this is much less common in the arteries than in the veins.

Such a condition is illustrated by a specimen in the museum of St. George's Hospital (Series VI., 61*d*), taken from the body of a woman aged thirty-seven years, who died from gangrene of the lower limbs. The specimen shows part of the aorta and the iliac arteries filled with old coagula adherent to the internal coat of the artery, but the coats of the vessel are not thickened (Fig. 2). The general tendency to thrombosis is shown by the fact that similar coagula were found in the main arterial trunks and in the veins of both lower limbs, in the iliac veins and lower part of the vena cava, in the arteries and veins of the left kidney, and in the left ventricle and right auricle of the heart. A specimen from a similar case is shown in Fig. 3.

Thrombosis of arteries has been observed in connection with various acute diseases, especially influenza, enteric fever, typhus fever, and pneumonia. Here again it is much less common than in the veins, and is found chiefly in the arteries of the lower extremities.

Cases have also been recorded by Dr. Dickinson[1] of death from rapid thrombosis of cerebral arteries in which there was no disease of the vessels and no acute illness.

'It appears,' says Dr. Dickinson, 'that in most of these cases two influences have been in operation. There has been disease of the heart, particularly contraction of the mitral opening. This occurred in four of the five cases. The general circulation has thus lost freedom, and a liability to venous and capillary congestion has been established. Besides this, there has been some especial cause by which the cerebral vessels have been overloaded or the circulation in them disturbed.'

[1] *St. George's Hospital Reports*, vol. i., 1886, p. 257.

Mr. Jonathan Hutchinson[1] has also recorded instances of the sudden occlusion of the femoral and other large arteries by thrombosis, in which he could discover no evidence of disease of the vessels.

Professor Osler[2] has related a remarkable case of a labourer, aged twenty, who was attacked with diarrhœa, loss of appetite, and epistaxis, followed by abdominal pain, fever, and delirium. Beneath the skin of the anterior thoracic region and the abdomen were many localized blue spots, but no characteristic eruption. On the tenth day of the illness symptoms of gangrene of the lower limbs appeared; the pulse was 120 to 140; temperature, 101° to 103°; there was great restlessness, persistent delirium, and abdominal tenderness. No pulsation could be felt in the femoral or popliteal arteries. The blood was examined with negative result; the urine was scanty and albuminous. The man died about two weeks from the beginning of his illness.

The case was regarded as one of typhoid fever, but the autopsy negatived this: the ileum was normal. There was thrombosis of the lower two inches of the abdominal aorta, with plugging of iliacs and femorals, the clots firm, reddish-brown, and closely adherent. The mesenteric vessels were free, but two large branches of the splenic artery were plugged. There were infarcts in the right kidney and spleen, from the latter of which spread a general peritonitis. Heart, lungs, and brain were normal. No otitis, no bone lesions. No micro-organisms were found in the blood during life. After death numerous micrococci were found in the infarct of the spleen and the lymph covering it.

In septic conditions it has been observed that the blood-platelets are abnormally numerous, and a primary arterial

[1] *Archives of Surgery*, vol. vii., p. 29, and vol ix., p. 100.

[2] *Transactions of Association of American Physicians*, 1887, p. 135.

thrombosis may occur, to which the infection of the vascular wall is secondary.

Arterial thrombosis occurs most frequently in the lower limbs, but does not exhibit the preference shown by the veins for the left side.

The symptoms are chiefly those due to obstruction of the vessel, and will depend very much upon whether this takes place rapidly or slowly, and also upon the position and importance of the artery concerned. When the artery is rapidly blocked the symptoms resemble those of embolism, from which it is often impossible to distinguish them. When the thrombus is of gradual formation there may be sufficient opportunity for the development of a collateral circulation to prevent any serious results, though this will, of course, depend upon the condition of the arteries generally, and their capacity to respond to the call upon their development.

The gradual obliteration of an artery may only be recognisable by the increasing hardness and thickness of the vessel, its want of elasticity, and the feebleness of its pulsation. There is often, however, some pain and tenderness felt in the course of the artery. When more rapid arterial thrombosis occurs there is usually acute pain, and the artery is tender to pressure; the nutrition of the limb is seriously imperilled, and, as in embolism, unless a collateral circulation is soon established, gangrene results. The danger of gangrene is less in the upper than in the lower extremities, but is greatly increased if the veins are also thrombosed, as in the case quoted on p. 17.

Rapid obstruction of visceral and cerebral arteries is most often of embolic origin, although it may sometimes be due to thrombosis.[1] Dr. W. H. Brown has recorded a

[1] See Dr. Dickinson's cases, *St. George's Hospital Reports*, vol. i., p. 257.

case of thrombosis of the abdominal aorta, iliac and femoral arteries, in which gangrene of the intestine occurred probably, I suppose, from thrombosis of the mesenteric artery.[1]

Obstruction of the coronary arteries is commonly due to a combination of arterial degeneration and thrombosis, the sudden development of symptoms (as, *e.g.*, angina pectoris) depending upon the completion of obstruction by thrombus of a vessel already narrowed by thickening of its coats.

Thrombosis of the pulmonary arteries will be considered in connection with venous thrombosis and pulmonary embolism.

Senile gangrene is usually caused by thrombosis of the small terminal arteries, the first symptom of which is often acute pain. Doubtless arterio-sclerosis and feeble circulation play an important part in its production, and the mischief is often started by some slight injury or inflammation, but the thrombosis is the immediate cause.[2] It is important to recognise this because, if the condition is seen and appreciated at its commencement, the danger may be averted by appropriate treatment. The reason why thrombosis is not more common in connection with arterio-sclerosis is that the heart is usually hypertrophied, so that the arterial resistance is overcome by the increased heart power.

## Venous Thrombosis.

The conditions under which coagulation of the blood occurs in the living veins are very similar to those which

[1] *Transactions of Clinical Society of London*, vol. xxvi., p. 1.

[2] Diabetic gangrene is probably caused by the same conditions. See remarks by M. Barthélemy at the Fifth International Dermatological Congress (*Transactions*, vol. ii., part i. p. 252).

lead to the formation of coagula in the arteries, but venous thrombosis is more common than arterial.

It has already been pointed out that any impairment of the nutrition of the endothelium of the bloodvessels leads to the formation of thrombi upon the vessel wall, and such an impairment of nutrition occurs whenever the rapidity of the circulation is materially diminished for any length of time. 'This,' as pointed out by Dr. Lazarus-Barlow,[1] 'depends upon the fact that the intima, including the endothelial cells, unlike the rest of the vessel wall, derives its nutriment from the blood in the lumen of the vessel, and not from that conveyed by the vasa vasorum. In most cases a diminution in velocity of blood-flow is the proximate cause of the thrombosis. Thus, in the heart the circulation is slowest in the appendices auriculæ, behind the flaps of the auriculo-ventricular valves, and between the columnæ carneæ. Normally it is rapid enough even here to maintain the nutrition of the cardiac endothelium; but when old age or wasting disease or any lesion of the valves has impaired the musculature of the heart, and it is no longer able to maintain the circulation at its normal velocity, the endothelium in these situations suffers first and to the greatest extent, and it is just in these situations that thrombi are found. For the same reason thrombosis more commonly occurs in veins than in arteries.'

In wounds, injuries, and ligature of veins, coagulation of the blood occurs, partly as a consequence of the mechanical disturbance of the blood-flow, and partly from the interference with the integrity of the endothelium. It occurs more readily than in wounds of arteries, because of the slower and less forcible blood-

[1] 'A Manual of General and Experimental Pathology,' by W. S Lazarus-Barlow, 1904, p. 117.

stream, and perhaps also because of the greater coagulability of the venous blood.[1]

Degeneration of the venous walls, dilatations, varices, and anything producing obstruction or retardation of the blood-current, are causes of thrombosis by disturbing the normal relations between the blood and the venous endothelium.

Inflammation of veins is a common cause of thrombosis, and, owing to the thinness of the venous coats, any surrounding inflammation invading the outer tunic of the vein is easily conveyed to the endothelium. This is one reason for the greater frequency of venous as compared with arterial thrombosis. The presence of micro-organisms either in the blood or the venous wall will give rise to thrombosis, as is frequently seen in septic inflammations. Venous thrombosis also occurs in consequence of changes in the blood; in connection with various acute and chronic diseases; and in conditions of great debility.

When coagulation occurs in a living vein, it is often difficult to say whether the thrombus is the cause or the result of phlebitis. If a vein is obstructed by a thrombus the nutrition of the endothelium at once suffers, and if the clot contains micro-organisms they will soon invade the intima, the resistance of which will be already lowered, and an endophlebitis will ensue.

But, on the other hand, there are numerous cases in which it is certain that the changes in the vein precede the thrombosis; for instance, in the chronic inflammation which occurs in connection with varix, in syphilitic endophlebitis, in tubercular invasion of the vein, and in

[1] Wooldridge has shown that the plasma of peptonized blood, which does not coagulate on the addition of fibrin ferment, coagulates freely if in addition a stream of carbonic acid is passed through it. ('The Chemistry of the Blood,' p. 294.)

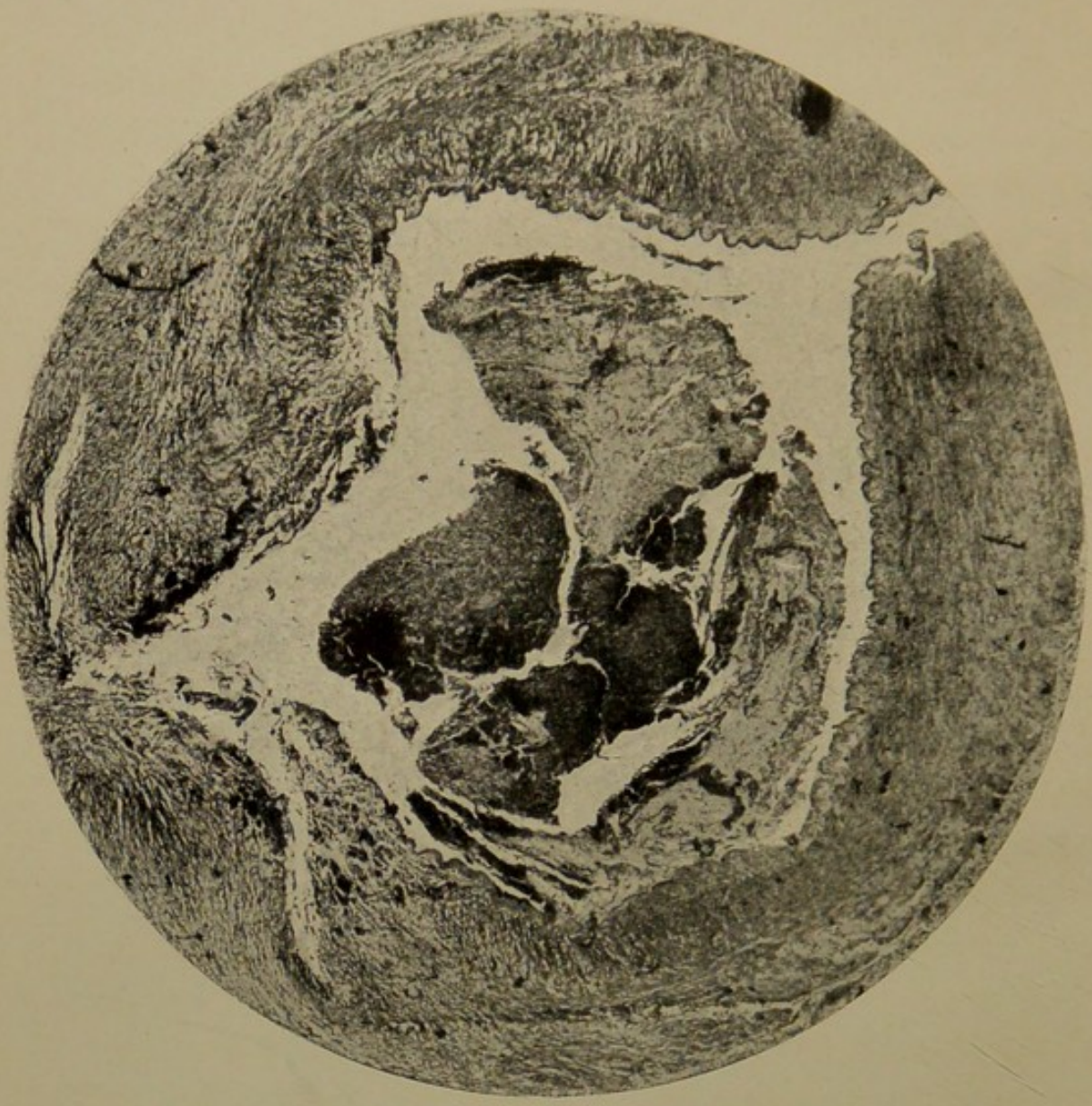

FIG. 4.—FEMORAL VEIN FROM A CASE OF INFECTIVE PHLEBITIS: SHOWING GREAT THICKENING OF COATS OF VEIN AND DISINTEGRATING CLOT.

[*To face p.* 23.

other degenerative changes. So also in septic inflammation spreading to the outer coat of a vein from a neighbouring focus, invasion takes place from without inwards, and thrombosis does not occur till the intima is reached.

The most serious of these conditions are those of septic origin. Here the presence of micro-organisms gives rise to coagulation, and the clot has an infective character which it communicates to any part to which it is carried. The process may begin in inflammation of the outer coat of a vein originating in a septic or suppurating wound, or in an infective focus, as in middle-ear disease or acute necrosis of bone; this, spreading to the inner coats, leads to endophlebitis and consequent thrombosis; or the organisms may invade the intima from the circulating blood or from an infected thrombus brought from a distant part. In these septic cases, whichever the mode of origin, the coats of the vein are always found much thickened (Fig. 4).

*Phlegmasia dolens* is an example of septic phlebitis extending from the uterine veins, through the iliacs, to the femoral and other veins. Thrombosis beginning in the uterine veins may extend from the uterus to the iliac and femoral veins, and even to the vena cava. Sometimes, however, the phlebitis would appear to be the result of the transmission of infecting organisms by the blood, and not of direct extension from the uterus.

There is an admirable paper on phlegmasia dolens, by Dr. David D. Davis, in the *Transactions of the Royal Medical and Chirurgical Society* for 1823,[1] in which the author describes several cases of the affection, and proves by dissection that its proximate cause is an inflammation

[1] *Transactions of the Royal Medical and Chirurgical Society*, vol. xii., p. 419, 1823.

and obstruction of 'one or more of the principal veins within and in the immediate neighbourhood of the pelvis.' But he did not trace the disease of the veins to its source in the uterus, though he recognised its connection with parturition and with disease of the pelvic organs.[1] The paper is illustrated by beautiful coloured drawings showing the condition of the iliac veins and the contained clot.

Dr. Robert Lee[2] in the year 1829 contributed two papers, with excellent coloured illustrations, to the same Society, in which he reported thirteen cases of phlegmasia dolens, in six of which the actual condition of the iliac and femoral veins was ascertained by dissection. 'From this I was led to infer,' says Dr. Lee, 'that inflammation of the iliac and femoral veins gives rise to all the phenomena of that disease in puerperal women, and that in phlegmasia dolens the inflammation commences in the uterine branches of the hypogastric veins, and subsequently extends from them into the iliac and femoral trunks of the affected side.'[3]

In the second paper Dr. Lee[4] reported three cases of cancerous ulceration of the uterus in which there was inflammation of the internal, common, and external iliac and femoral veins, with all the characteristic symptoms of puerperal phlegmasia dolens.

In a third paper, published in 1853,[5] Dr. Lee reported forty-three additional cases in confirmation of his explanation of the pathology of the disease, and he adds that it has been demonstrated by morbid anatomy that phlegmasia dolens is a disease which may take place in women who have never been pregnant, and in the male

[1] *Transactions of the Royal Medical and Chirurgical Society*, vol. xii., p. 445.

[2] *Ibid.*, vol. xv., p. 132, and p. 369.

[3] *Ibid.*, vol. xxxvi., p. 281.

[4] *Ibid.*, vol. xv., p. 369.

[5] *Ibid.*, vol. xxxvi., p. 281.

sex, and that under all circumstances the proximate cause is the same—namely, inflammation of the iliac and femoral veins.

In the same volume of transactions is an elaborate paper by Dr. F. W. Mackenzie,[1] containing 'Researches on the Pathology of Obstructive Phlebitis, and the Nature and Proximate Cause of Phlegmasia Dolens,' and recording a number of experiments on animals made with the view of studying the effects of inflammation of the iliac veins. From these experiments Dr. Mackenzie concluded that in a healthy animal the results of obstruction of the common iliac vein, produced by the application of a ligature or other irritants, were confined to the immediate seat of injury, and showed no tendency to spread beyond it. There was no attendant fever or constitutional disturbance, and only a slight and transient œdema of the limb. But, on the other hand, Dr. Mackenzie ascertained by another series of experiments, that if the blood was vitiated from local or constitutional causes, large portions of the venous system may become obstructed and inflamed independently of any injury of the veins; and he assumed that these phenomena depended upon 'a disturbance of the relations which normally exist between the blood and the lining membrane of the veins.' Finally, applying the results of his experiments upon animals to an analysis of 100 cases of phlegmasia dolens in the human subject, he concluded that the obstruction and inflammation of the iliac and crural veins, which is an essential feature of the disease, depends upon the presence of abnormal material in the blood.[2]

Here, it will be observed, was a very close approach to

[1] *Transactions of the Royal Medical and Chirurgical Society*, vol. xxxvi., p. 169.

[2] *Ibid.*, vol. xxxvi., p. 240.

the recognition of a microbic origin for the disease; and it may be noticed that the important and accurate advance in the knowledge of the causation of phlegmasia dolens, made by the three authors to whose work I have drawn attention, was the result of laborious pathological investigation, of experiments upon animals, and of careful clinical observation—a striking contrast to the wild and unfounded theories which had previously prevailed.

*Pylephlebitis* is an example of suppurative inflammation and thrombosis of the portal vein, originating in an infective focus in some part of the area belonging to the branches of the vein. The most common origin is suppuration in connection with the vermiform appendix, a point to be borne in mind in relation both to the diagnosis and treatment. The symptoms are often obscure, but are chiefly suggestive of pyæmia. Rigors, fever of the remittent type, sweating, jaundice, and enlargement and tenderness of the liver, would make the diagnosis probable, especially if there were evidence of intestinal disease, for which a careful search should be made.

In the light of recent investigations it seems probable that many of the so-called idiopathic or spontaneous thromboses are of microbic origin; but there are some in which no micro-organisms can be found in the thrombi: while, on the other hand, bacterial invasion of the wall of a vein may occur without the formation of a thrombus.

Septic phlebitis is a condition attended by serious symptoms and grave danger. The severity of the symptoms will depend upon the virulence of the infection, the susceptibility of the individual, and the position of the mischief.

In the acute form of suppurative phlebitis the wall of the vein is invaded by pyogenic organisms; coagulation occurs in the inflamed vein, and the thrombus is itself

infected, and by its softening carries infection into the blood-stream, giving rise to septicæmia and pyæmia. An admirable paper on this subject was communicated by Mr. Arnott to the Royal Medical and Chirurgical Society in 1828, in which he showed the relation between the primary and secondary affections in phlebitis.[1]

If a superficial vein is affected it is observed to be swollen and tender, and the skin over it shows a red line. These conditions tend to spread rapidly along the vein in the direction of the blood-stream. If a deep vein is attacked pain is felt in the part, soon followed by swelling, tenderness and suppuration. The constitutional symptoms are severe; the temperature and pulse rise rapidly; rigors supervene, followed by profuse sweating; the tongue is dry, appetite is lost, and there may be delirium. To these symptoms are soon added those of general pyæmia: rapid oscillations of temperature, rigors, sweating, and disseminated suppurations. Such cases usually end fatally, partly by the general poisoning of the blood and the consequent fever and exhaustion, and partly by the occurrence in important organs, especially the lungs, of centres of inflammation and suppuration.

Chaucer described the condition very accurately in the 'Knighte's Tale.'

> 'The clothred blood, for eny leche-craft,
> Corrumpith, and is in his bouk i-laft,
> That nother veyne blood, ne ventusyng,
> Ne drink of herbes may ben his helpyng.
> The vertu expulsif, or animal,
> Fro thilke vertu cleped natural,
> Ne may the venym voyde, ne expelle.
> The pypes of his lounges gan to swelle,
> And every lacerte in his brest adoun
> Is shent with venym and corrupcion.

[1] *Transactions of the Royal Medical and Chirurgical Society*, vol. xv., p. 1.

Him gayneth nother, for to get his lyf,
Vomyt up-ward, ne doun-ward laxatif ;
Al is to-broken thilke regioun ;
Nature hath now no dominacioun.
And certeynly wher natur will not wirche,
Farwel phisik ; go bere the man to chirche.'

When the infection is less virulent the symptoms are correspondingly less severe ; and many gradations are met with down to cases in which, though there is a well-marked phlebitis, the symptoms of infection are but slight. I have no doubt that some of the cases of so-called idiopathic phlebitis are of this nature. The infection may be of a low degree of virulence, and the subject may have a high resisting power or immunity. Moreover, the infected thrombus may be isolated from the general circulation, and so produce only local effects.

In other cases the phlebitis may become manifest, while the source of the infection may be obscure. For instance, I have seen a case in which a slowly-forming ischio-rectal abscess, accompanied by so little pain that the patient disregarded it, gave rise to a septic phlebitis, manifested by rigors, fever and sweating, as well as by severe local symptoms.

In some of the cases of chronic pyæmia in which large superficial collections of pus occur I have found indubitable evidence of thrombosis, and on opening the abscesses the pus was seen to be mixed with broken-down blood-clot.

In all cases of phlebitis it is therefore well to seek for a source of infection, for in many instances in which the disease begins with but slight constitutional disturbance symptoms of severe infection subsequently develop. These symptoms probably arise coincidently with the disintegration of the infected thrombus and the entrance into the blood-stream of septic organisms. The successful

treatment of such cases will, of course, depend very largely upon the removal of the focus of infection. Notable examples of this are the cases of chronic suppuration of the middle ear giving rise to endophlebitis and purulent thrombosis of the lateral sinus, in which life may be saved by ligature of the internal jugular vein, and the thorough removal from the sinus of the infected clot.

In other cases of phlebitis the focus of infection may be found in a deeply-seated abscess, a suppurating wound, or an osteomyelitis, and the first essential in the treatment is the removal or disinfection of such focus. Abscesses must be freely opened and thoroughly cleansed and drained; suppurating or foul wounds must be disinfected; bones the subject of osteomyelitis must be removed by resection or amputation; an accessible vein suspected to contain a septic clot should be ligatured above and below the thrombus, and the portion between the ligatures excised. Beyond these local measures the treatment must be adapted to the general condition of the patient. In the severe and advanced cases it will be that of pyæmia. Quinine should be given in frequently repeated doses, and the strength supported by appropriate food and stimulants. The exclusion of all insanitary conditions should be carefully looked to, and the patient should be kept in a well-ventilated and light room, and removed as soon as possible into fresh and healthy surroundings. He should not remain

> 'in populous city pent,
> Where houses thick and sewers annoy the air';[1]

but should seek

> 'airs, vernal airs
> Breathing the smell of field and grove.'[2]

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[1] Milton, 'Paradise Lost,' ix. 445.
[2] *Ibid.*, iv. 264.

He should go where he can

> 'look into the fair
> And open face of heaven."[1]

For pure air and sunshine are valuable aids to recovery, and the high moorlands are often more beneficial than the sea-coast.

[1] Keats, 'Sonnets,' x.

# LECTURE II

Varieties of phlebitis and thrombosis.—Thrombosis in connection with (*a*) varix ; (*b*) gout ; (*c*) syphilis ; (*d*) enteric fever ; (*e*) typhus fever ; (*f*) chlorosis ; (*g*) influenza ; (*h*) pneumonia ; (*i*) appendicitis ; (*j*) gastric ulcer and other abdominal diseases.—'Idiopathic' thrombosis.—Preference of venous thrombosis for left lower limb.

Thrombosis of upper limb.

Pulmonary embolism and thrombosis.

Symptoms of non-infective phlebitis and thrombosis.—Explanation of occurrence or absence of œdema.—Results of venous obstruction and obliteration.—Obliteration of venæ cavæ.

LEAVING now the cases of undoubted septic origin, we come to the consideration of a class in which the phlebitis is of quite different character, and of which the dangers are of a quite different kind.

Many of these undoubtedly depend upon changes in the blood which favour the occurrence of thrombosis, to which the phlebitis is secondary.

It seems possible that in some of the diseases with which venous thrombosis is associated, toxic or other chemical changes in the blood may affect the nutrition of the leucocytes and red corpuscles, or favour the production and increase of the blood-plates, and thus, aided by an enfeebled circulation, lead to the formation of a thrombus.

Professor Osler[1] has observed that in acute fevers the

[1] Osler, 'Cartwright Lectures,' *op. cit.*, p. 365.

blood-plates do not become more numerous in the early stage of the disease, but increase in number as the patient becomes weaker and more debilitated. In typhoid fever, for instance, there is no increase during the first week, but a notable increase in the third and fourth weeks.

It will be observed that the increase in the number of the blood-plates occurs just in that stage of disease which is especially liable to thrombosis.

Dr. J. H. Pratt,[1] of Boston, has reported a series of observations on the relative number of the platelets in various diseases. There was a marked increase in chlorosis; the greatest diminution was observed in a case of purpura hæmorrhagica.

In other cases in which the slowness or feebleness of the circulation would seem to be the chief factor, it is difficult to say how far the diminished nutrition of the vessel wall is the immediate cause of coagulation, and how much is due to the invasion of micro-organisms.

Mr. W. C. C. Pakes[2] has recorded the case of an anæmic and phthisical girl of thirteen years who was admitted to Guy's Hospital with thrombosis of the left iliac vein and swelling of the left lower limb. A week before death the right leg became swollen, owing to thrombosis of the right iliac vein. Post-mortem there were thrombi in both iliac and femoral veins; and in the thrombus of the left femoral vein, which was carefully excised and examined, was found the *Bacillus proteus vulgaris*.

Mr. Pakes remarks that 'in health the bactericidal power of the blood is probably sufficiently great to destroy this organism if it should gain access to it; but when from any cause this action is diminished, the

[1] *Johns Hopkins Hospital Bulletin*, May, 1905, p. 201.

[2] *Transactions of Pathological Society of London*, vol. li., p. 47.

organism becomes relatively pathogenic. The bactericidal power may be reduced in many ways, of which a wasting disease and a dose of ptomaines are two.'

Dr. F. C. Turner[1] has published a case of thrombosis of the iliac vein in a man, aged thirty-six, who died of Hodgkin's disease, a condition in which there are important changes in the leucocytes, possibly due to micro-organisms.

Thrombosis is a common occurrence in *varicose veins*, and is usually the result of injury; but in gouty persons the subject of varix it is often seen in association with eczema, when no injury can be traced.

Coagulation is especially apt to occur in the cyst-like dilatations and tortuous veins so frequently met with in the lower part of the thigh and the neighbourhood of the knee. Such varices are very liable to become inflamed because of their prominence on a part of the limb often exposed to injury, and because in this situation they are particularly subject to irritation from pressure and friction, as in riding and other exercises.

In *varicocele* also thrombosis is not infrequently caused by injury, and in some cases the resulting obliteration of the veins produces a complete cure of the varicocele.

In gouty persons it is more often the smaller nævus-like patches of varicose veins in the lower part of the leg, or the muscular branches of the calf, which are affected.

Considering the frequency with which thrombosis occurs in varicose veins, it is remarkable that it is comparatively seldom attended with serious symptoms. There is usually but little constitutional disturbance, and the local discomfort is often but slight; the chief danger is of displacement of the clot and consequent embolism. This serious event is fortunately not of very common

[1] *Transactions of Pathological Society of London*, vol. xxix., p. 344.

occurrence, but it is a danger which ought always to be borne in mind in the treatment of such cases, particularly those in which the blocked vein is near a joint or in direct communication with the main trunk, as, for instance, in the neighbourhood of the knee or of the saphenous opening.

*Gouty Phlebitis.*—Since the publication in 1866 of Sir James Paget's well-known lecture, gouty phlebitis has been a well-recognised disease. There can be no doubt that persons of gouty habit or ancestry are more than commonly liable to phlebitis, and that in them the affection has usually certain distinguishing characters. To quote Sir James Paget's description: 'Gouty phlebitis is far more frequent in the lower limbs than in any other part; but it is not limited to the limb that is, or has been, the seat of ordinary gout. It affects the superficial rather than the deep veins, and often occurs in patches, affecting, for example, on one day a short piece of a saphenous vein, and on the next day another separate piece of the same, or a corresponding piece of the opposite vein, or of a femoral vein. It shows herein an evident disposition towards being metastatic and symmetrical—characters which, I may remark by the way, 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.'[1]

Sometimes gouty phlebitis begins in the deep veins of the calf, of which the first symptom may be a sudden and acute cramp-like pain, which is soon followed by deeply-seated tenderness to pressure. There is a tendency to

[1] 'Clinical Lectures and Essays,' p. 293, 1875. See also Sir Prescott Hewett in Presidential Address to the Clinical Society (*Transactions*, vol. vi., p. xxxvii), who relates cases of gouty phlebitis, and points out that it often begins in the back of the leg, midway between the heel and the ham.

frequent recurrences of the attacks, which are especially apt to occur when the patient is fatigued or below the usual standard of health. It is sometimes the only manifestation of gouty inheritance, but is frequently combined with other obviously gouty symptoms. It is not usually attended with much constitutional disturbance.

An example of troublesome and frequently recurring phlebitis occurred in a patient from whom I had on two occasions removed uric acid calculi by lithotrity. He lived most abstemiously and on a most carefully regulated diet, but nevertheless had frequent attacks of phlebitis, alternating with eczema, cystitis, and other symptoms of the uric acid diathesis.

It seems to me, however, that it is the fashion at the present time to attribute many cases of phlebitis, as of other diseases, to gout or the uric acid diathesis, when there is no evidence whatever of either the presence or the inheritance of gout.

Phlebitis is sometimes seen in association with, or during the convalescence from, acute rheumatism, but I do not think that this is sufficiently common to justify the inference that there is any special connection between the two diseases.

*Syphilitic phlebitis* occurs in two forms. In one variety the superficial venous trunks are attacked, chiefly those of the lower, but sometimes those of the upper, extremities. The walls of the veins undergo inflammatory thickening, and thrombosis ensues. This is seen in the early eruptive period. Later in the course of the disease a nodular phlebitis occurs, attacking chiefly the subcutaneous veins of the lower limbs, especially those which are varicose or sclerosed. The condition is characterized by nodular thickenings of the venous coats, with limited thrombosis. It is said to occur also in the corpus cavernosum and in

the spermatic cord. In neither form is there usually any considerable constitutional disturbance, but the affected veins are painful and tender. The disease yields to the usual antisyphilitic treatment.[1]

Thrombosis is frequently met with during convalescence from *enteric fever*, and is not uncommon in other conditions of exhaustion such as result from prolonged or serious illness, and in the late stages of phthisis. It occurs occasionally, but not often, in the acute stage of enteric fever.

Professor A. E. Wright and Dr. H. H. G. Knapp[2] have proved that the coagulability of the blood is diminished in the acute stage, but increased in the convalescent stage of typhoid fever; the coagulation time being four and a half minutes in the convalescent stage, as compared with twenty minutes during the pyrexia. They show, moreover, that the blood of these convalescents is not only much more coagulable than the normal, but that it also contains twice the normal amount of lime salts; whereas, if the lime salts are brought within the normal limits, the coagulability is reduced to even less than that of normal blood.

The authors suggest that the increased coagulability of the blood of typhoid convalescents is dependent upon an excess of lime salts, and that this excess is derived from the milk upon which typhoid patients are chiefly fed. Cow's milk, it is noted, contains 1 part in 600 of lime, as compared with 1 part in 800 contained in lime-water.

The remedy indicated is the administration of citric

[1] See Papers by Jullien, Barthélemy, E. Hoffmann, etc., in *Transactions of the Fifth International Dermatological Congress*, 1904, vol. ii., part i., pp. 225-265.

[2] 'On the Causation and Treatment of Thrombosis occurring in Connection with Typhoid Fever,' *Transactions of the Royal Medical and Chirurgical Society*, vol. lxxxvi., p. 1, 1903.

acid as a decalcifying agent; and in seven patients, in all of whom the blood was found to be abnormally coagulable, it was observed that the administration of citric acid (36 grains three times a day) was followed by a decalcification of the blood and a corresponding diminution of its coagulability. It is suggested that, with a view to restricting the intake of lime salts, the milk given might be partially decalcified, and thus rendered more easily digestible and less constipating. This can be effected by adding to each pint of milk from 20 to 40 grains of citrate of soda.

These valuable suggestions appear to me to be applicable to many other conditions besides typhoid fever.

Dr. W. W. Keen, of Philadelphia,[1] in his work on 'The Surgical Complications and Sequels of Typhoid Fever,' collected 128 cases of venous thrombosis following fever. Of these, 'only four involved the upper extremity alone, two involved both the arm and leg, and all the other 122 cases were limited to the lower extremities.' He observes that, whereas arterial thrombosis occurs with almost equal frequency on the two sides of the body, two-thirds of the cases of venous thrombosis were on the left side; and that both forms of thrombi, arterial and venous, 'form most frequently during or just after the period of greatest cardiac weakness—a weakness felt most at such distant points as the legs.' Of 148 cases, 58 occurred in the second and third weeks.

The influence of the enfeebled circulation is also shown in the location of the thrombus. 'The coagulation takes place at points mechanically favourable to slowing of the current—*e.g.*, the bifurcation of arteries and the valves of the veins.[2] Dr. Keen also gives reasons for thinking that

[1] 'The Surgical Complications and Sequels of Typhoid Fever,' by W. W. Keen, M.D., LL.D., London, 1898, p. 74.

[2] *Op. cit.*, p. 363.

some at least of the cases of necrosis of bone following typhoid may be due to thrombosis of the arteries or veins.[1]

Dr. Murchison[2] recorded a remarkable case in which, during the course of a severe attack of *typhus fever*, gangrene of both legs occurred in consequence of thrombosis of the iliac and femoral arteries, and of the femoral and popliteal veins of the left limb. The patient, a woman forty-five years of age, died on the forty-first day of illness. Dr. Murchison points out that, although there was gangrene of both legs, there was swelling only of the left leg, in which the vein, as well as the artery, was obstructed. He also remarks that in the venous thrombosis occurring in connection with fever it is almost invariably the left lower limb which is affected.

*Chlorosis.*—This is a disease in which it is well known that there is an especial liability to thrombosis. This depends, no doubt, upon the condition of the blood, in which there is a great diminution both of the number of red corpuscles and also of their contained hæmoglobin.

The relatively greater number of white corpuscles in chlorosis and their slow movement along the walls of the bloodvessels are conditions favourable to the occurrence of thrombosis, as also is the increase in the number of platelets which has been observed. It would seem, also, that the chemical composition of the blood is altered, the potassium being diminished and the sodium and chlorine increased.[3]

Dr. Lee Dickinson[4] has pointed out 'that the intravascular coagulation brought about by the injection of foreign substances (snake venom and nucleo-proteid) into

[1] *Op. cit.*, p. 111.

[2] *Transactions of the Pathological Society of London*, vol. xvi., p. 93.

[3] Biernacki, *Wiener Medicinische Wochenschrift*, 1893, pp. 1721 and 1765.

[4] *Transactions of Clinical Society of London*, vol. xxix., p. 63.

the circulation of animals, takes place by preference in the venous system, and is greatly favoured by excess of carbonic acid in the blood. Chlorotic blood, by reason of its poverty in hæmoglobin, is certainly deficient in oxygen, and probably equally overloaded with carbonic acid. The comparative infrequency of thrombosis in the cerebral sinuses, where the mechanical conditions seem so favourable, is perhaps explained by the observation of Dr. Leonard Hill 'that the blood obtained from the torcular Herophili contained far less carbonic acid than that from the femoral vein.'

Dr. A. E. Wright[1] has shown that an increase of the carbonic acid in the blood much increases its coagulability, and he relates a case of hæmophilia in which hæmorrhage was arrested by the inhalation of carbonic acid gas.

Chlorotic thrombosis is apt to be extensive and recurrent. When its seat is the cerebral sinuses, it is a condition of extreme gravity; when affecting the extremities, its chief danger is pulmonary embolism.

[1] *Proceedings of Royal Society*, vol. lv., p. 279; and 'On Methods of Increasing and Diminishing the Coagulability of the Blood,' *Brit. Med. Journ.*, July, 14, 1894, p. 57.

[2] Professor W. H. Welch, in an admirable article in Allbutt's 'System of Medicine' (vol. vi., p. 200), analyzes a collection of 78 cases of venous chlorotic thrombosis, from which he deduces some instructive facts. These may be tabulated thus:

Of 78 cases of venous chlorotic thrombosis—

There was thrombosis of the cerebral sinuses in 32 (39 per cent.). Six of these had also thrombosis of the veins of the lower extremities (19 per cent.); in 4 the thrombus extended into the internal jugular vein.

In 50 there was thrombosis of the veins of the lower extremities. (Bilateral in 46 per cent.; unilateral in 54 per cent.—34 left, 20 right. 64 per cent. began in left limb; 29 per cent. in right limb; 7 per cent. in both limbs simultaneously. 25 per cent. had pulmonary embolism—all but 2 fatal.)

In 2 there was thrombosis of the veins of upper and lower extremities.

In 1 there was thrombosis of the veins of upper extremities only.

Thrombosis may occur in the condition of debility sequential to *influenza*, as to other febrile diseases; but it is also not uncommon during the acute stage of the attack, when it is possibly due to the influenzal bacillus.

Dr. T. J. Horder[1] has recorded two cases of influenzal endocarditis in which the *Bacillus influenzæ* from the blood was cultivated during life. In both cases there was marked leucocytosis.

Thrombosis is also sometimes associated with *pneumonia*. It has been observed that inflammatory exudations that are associated with the presence of the pneumococcus show a marked tendency to coagulate.[2]

Peripheral thrombosis is a well-recognised complication of *appendicitis*.

Notes of 1,000 cases of operation for appendicitis at the London Hospital were furnished by Mr. Hugh Lett at the discussion at the Royal Medical and Chirurgical Society in February, 1905. Among these 1,000 cases there were twelve of thrombosis of the veins of the lower extremities, and one case of pulmonary embolism.

Of 442 cases of operation for appendicitis at St. George's Hospital, of which notes were furnished by Mr. Laurence Jones for the same discussion, nine had thrombosis of the lower limbs and three had pulmonary embolism.

Of 863 cases reported by Dr. H. P. Hawkins, of

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Professor Welch says: 'After making due allowance for the undoubtedly disproportionate representation of embolism of the large pulmonary arteries in published records, this catastrophe remains sufficiently frequent to impart a certain gravity to the prognosis even of simple femoral thrombosis in chlorosis.'

[1] *Transactions of Royal Medical and Chirurgical Society*, vol. lxxxix., p. 1.

[2] Lazarus-Barlow, 'Manual of Pathology,' second edition, 1904, p. 197.

St. Thomas's Hospital, there were two cases of thrombosis of the veins of leg and two cases of pulmonary embolism.

Mr. Aslett Baldwin reported 234 cases from the Middlesex Hospital, with one case of pulmonary embolism and thrombosis of iliac veins.

Mr. Lockwood, of St. Bartholomew's Hospital, contributed 200 cases, with two cases of thrombosis of mesenteric veins and one case of thrombosis of the iliac vein.

Mr. G. E. Gask reported 795 cases at St. Bartholomew's Hospital, with five cases of venous thrombosis.

Of 125 cases reported by Mr. H. S. Clogg and Mr. H. A. T. Fairbank, of Charing Cross Hospital, there was one case of thrombosis, both femoral veins being affected.

Three hundred and fifteen cases were collected by Mr. Ralph Thompson, of Guy's Hospital, among which were six cases of femoral thrombosis and one of pulmonary embolism.

Mr. G. R. Turner, of St. George's Hospital, reported 140 cases, with one case of thrombosis of the femoral vein and one case of pulmonary embolism.

## TABLE I.

OPERATIONS FOR APPENDICITIS, SHOWING NUMBER OF CASES OF THROMBOSIS AND OF PULMONARY EMBOLISM.

| | No. of Cases. | Thrombosis. | Pulmonary Embolism. |
|---|---|---|---|
| London Hospital ... ... | 1,000 | 12 | 1 |
| St. George's Hospital ... | 442 | 9 | 3 |
| St. Thomas's Hospital ... | 863 | 2 | 2 |
| Middlesex Hospital ... | 234 | 1 | 1 |
| St. Bartholomew's Hospital | 795 | 5 | — |
| Charing Cross Hospital ... | 125 | 1 | — |
| Guy's Hospital ... ... | 315 | 4 | 1 |
| | 3,774 | 34 | 8 |

Thrombosis is also met with in connection with operations for gastric ulcer and other abdominal diseases. Of fifty cases of operation at St. George's Hospital for perforated gastric and duodenal ulcer recorded by Mr. T. Crisp English, there were three cases of thrombosis, all of the veins of the left lower extremity.[1]

Dr. A. H. Corder, of Kansas, has collected 232 cases of phlebitis following abdominal and pelvic operations. In 213 cases the left saphenous or femoral vein was affected.

Dr. Corder asserts that phlebitis occurs in about 2 per cent. of all abdominal operations, and is especially frequent after operations on patients anæmic from hæmorrhage, as, *e.g.*, abdominal hysterectomies for bleeding fibroids.[2]

There still remain a certain number of cases of thrombosis and phlebitis in which no association with any precedent disease or injury can be traced, but which occur in apparently healthy individuals. This is the class usually spoken of as 'idiopathic.' In the light of recent researches and increasing facilities for the detection of micro-organisms, it seems probable that some of these are really of infective origin, although the source of infection has not always been discovered. But there are others in which neither clinical nor pathological evidence of infection can be obtained, and of which it must be admitted that the origin is obscure. Sir James Paget related two such cases.[3] In both of these the upper extremity was affected, and the patients were healthy men.

In the first case 4 inches of the axillary vein could be

[1] *Transactions of the Royal Medical and Chirurgical Society*, vol. lxxxvii., p. 27.

[2] *Journal of the American Medical Association*, December 9, 1905, p. 1792.

[3] 'Clinical Lectures and Essays,' p. 305.

felt blocked. The arm was swollen, and there were enlarged superficial veins over the upper part of the chest. Sir James Paget says: 'No cause whatever could be traced for this condition—no injury or pressure, no known inheritance of disease, no disturbance of the general health, past or present.' With the help of the hot douche, warmth, and friction, recovery took place in the course of a year.

In the second case the arm was in a similar condition, and no cause for it could be discovered. 'It was uncertain how long this state of the arm had existed; it had been observed only a week; its rate of increase was unknown.' The patient remained in the same condition for a month, and was then treated by leeching and mercury, after a fortnight of which he had an attack of scarlatina, and while this was running its course all signs of the affection of the arm disappeared.[1]

I will add another case.

A healthy man, fifty-four years of age, living a temperate and healthy but busy life, was suddenly seized with acute cramp-like pain in the left calf. This was on the evening of a somewhat fatiguing day, but no unusual exercise or exertion had been undertaken. The pain subsided after a few hours' rest, but next day recurred, when walking and standing were somewhat painful. A careful examination revealed some deep-seated tenderness in the calf, but no swelling. The patient showed no sign of illness; the temperature and pulse were natural, the urine clear and of normal acidity. He was not anæmic; he had never had any signs of gout, nor was he of gouty ancestry; he was not aware of having received any injury. Complete rest was prescribed; the patient was confined to bed, and the limb covered with a layer of wool and lightly bandaged

[1] 'Clinical Lectures and Essays,' p. 307.

to a splint. Nevertheless, the phlebitis spread upwards to the femoral vein, with severe pain and moderate constitutional disturbance. Subsequently the right femoral vein was attacked, and in the second week symptoms of pulmonary embolism occurred. The patient, after a tedious illness, gradually recovered.

I have seen other analogous cases, and I must admit that I cannot explain them.

A paper was published in 1905 on this class of cases by Dr. John Bradford Briggs, of Washington.[1] He describes a variety of phlebitis affecting the veins of the extremities, and occurring in the absence of all conditions that are commonly recognised as predisposing to inflammation of the veins. The condition occurs suddenly in persons apparently in perfectly good health, and, without fever or other disturbance, leads to obliteration of the affected vein. It is apt to recur, and to spread from the point at which the vein has previously become obstructed.

Dr. Briggs quotes cases in which the saphena, the femoral, and the axillary veins were respectively the seat of the disease. He admits that the cases are 'obscure alike in their pathology and in their remote and immediate etiology,' but thinks that the affection is due to sclerosis of the veins, and is concerned with the wall rather than with the contents of the vessel.

Dr. Briggs refers to a French thesis by Dr. Daguillon,[2] in which is described what the author calls a primitive form of phlebitis—*i.e.*, a phlebitis without any immediate determining cause. It has a special clinical picture—that of limited, localized, superficial phlebitis, affecting the lower limbs, causing slight local and no general reaction.

[1] 'On Recurring Phlebitis of Obscure Origin,' by J. B. Briggs, M.D., *Johns Hopkins Hospital Bulletin*, June, 1905, p. 228.

[2] Paris, 1894.

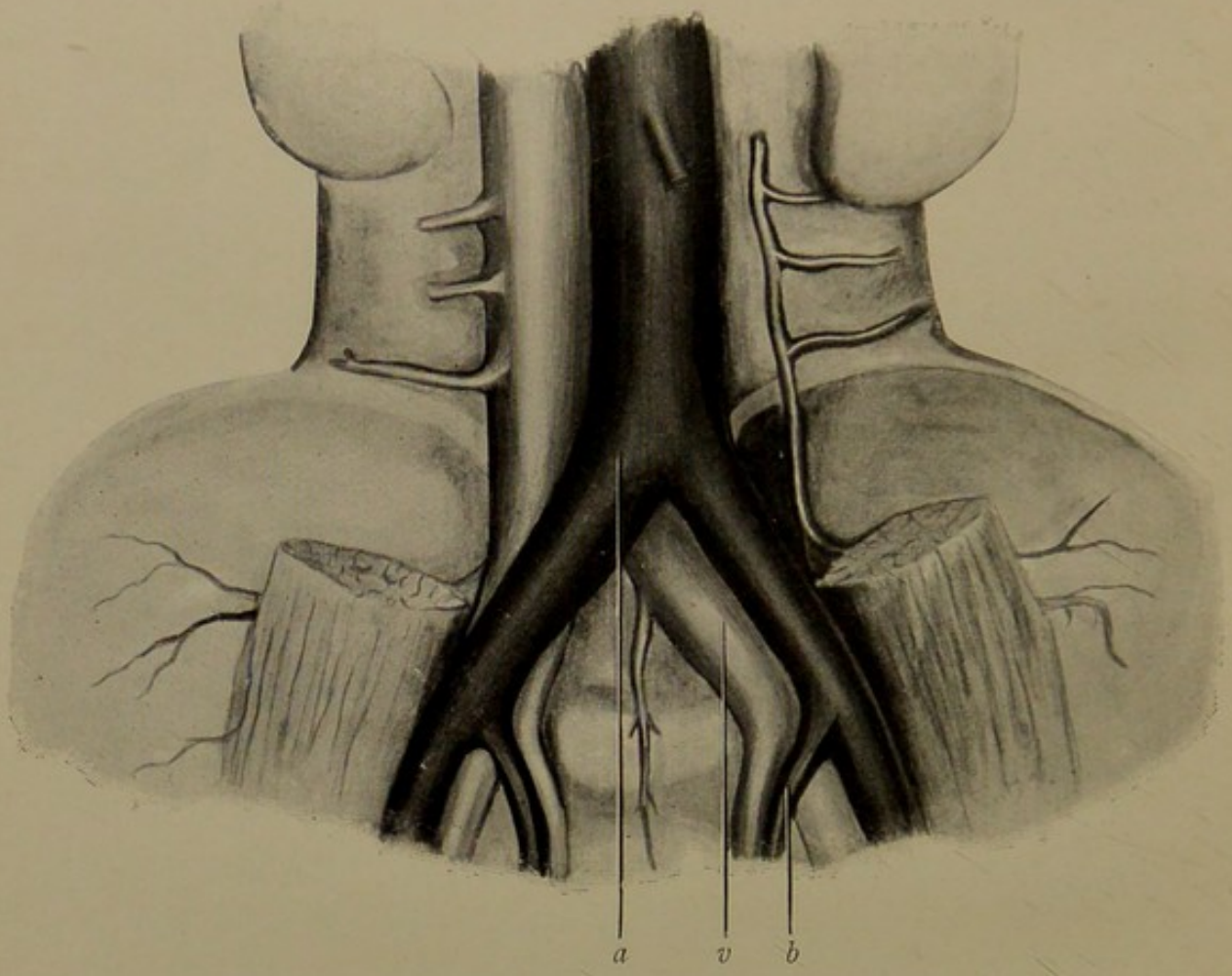


FIG. 5.—THE RELATIONS OF THE ILIAC ARTERIES AND VEINS. THE LEFT COMMON ILIAC VEIN (*v*) IS SEEN CROSSED AT A RIGHT ANGLE BY (*a*) THE RIGHT COMMON ILIAC ARTERY AND (*b*) THE LEFT INTERNAL ILIAC ARTERY.

[*To face p.* 45.

It is of slow evolution, and shows a progressively ascending march by successive attacks and relapses. The prognosis is serious, owing to the facility with which embolism occurs. This differs somewhat from the cases described by Dr. Briggs, and Dr. Daguillon believes that the process is mainly one of parietal thrombosis, and 'is an indication of a general diathetic influence, shown in an arthritic constitution, with or without actual gout'—a statement which does not appear to me to throw much light upon its causation or pathology.

It will be observed that thrombosis occurring in connection with fever, chlorosis, phthisis, and other debilitating diseases, after operations, and, indeed, whatever its cause, shows a curious preference for the left lower limb. The only reason for this preference which, so far as I know, has been suggested is the position of the left common iliac vein, the current in which may possibly be somewhat obstructed by the pressure of the right common iliac artery, under which the vein passes. On looking carefully at the anatomy of the vessel, I noticed that not only was the vein crossed by the right common iliac artery, but also by the left internal iliac artery as it passes downwards to the sacro-sciatic foramen, both of the arteries crossing the vein almost at a right angle, and in marked contrast to the relations on the other side (Fig. 5).

This may not seem a very strong reason for the great predominance of thrombosis in the left vein, yet if the blood is in a condition in which a slight retardation of the current would be sufficient to turn the balance towards coagulation, this anatomical difference may be enough to determine the thrombus to the left side. It is possible also that the pressure of a loaded rectum may to some extent interfere with the venous circulation of the left side of the pelvis.

Thrombosis may, however, occur in the upper limb. I have alluded to two cases of Sir James Paget's.

Sir Prescott Hewett[1] recorded a case in which, after small-pox, both axillary veins, as well as both external iliac veins, became permanently blocked. The patient, 'an officer in a heavy cavalry regiment, was nevertheless able to remain in the service and efficiently to discharge his duties, for a vast collateral circulation had been developed, and there was a mass of large tortuous veins spreading over the belly and chest.'

Dr. Ormerod[2] has published a case in which 'there was complete obstruction of both innominate veins, internal jugulars, subclavians, and anterior and external jugulars. They were filled with adherent clot. The clot was rather firmer on the right side than on the left. A projecting end of clot hung into the superior cava, but was not adherent there. The clot ceased at the opening of the azygos. The azygos, and the superior intercostal opening into it, were pervious and dilated. There was no clotting in the cerebral sinuses. The patient was under Dr. Gee's care, and was admitted for mitral stenosis. There was much dilatation of the left auricle and right chambers. The symptoms of thrombosis, which developed in the hospital, pointed to its commencement in the right subclavian vein. No local cause could be found for it post-mortem.'

I have seen thrombosis of the veins of the upper arm occurring in, and associated with, simple debility.

Dr. F. C. Turner[3] has recorded a case of thrombosis of the innominate, internal jugular, and subclavian veins, occurring in an anæmic man, aged forty-four years, who had suffered from severe hæmorrhage from a malignant

[1] *Transactions of Clinical Society of London*, vol. vi., p. xxxvii.
[2] *Transactions of Pathological Society of London*, vol. xl., p. 75.
[3] *Ibid.*, vol. xliii., p. 64.

ulcer of the stomach. There was much swelling of the arm, shoulder, and neck, and enlargement of the surface veins over the chest and upper arm.

Dr. Wilberforce Smith has described[1] an instance of thrombus, organized and adherent in the innominate and subclavian veins, in a case of pulmonary phthisis. There was a cavity in the apex of the lung, over which the pleura was thickened, and the adjacent part of the subclavian vein was thickened and narrowed. Four weeks before death œdema and lividity of the arm appeared.

Besides these examples of thrombosis occurring in conditions which are of themselves of grave import, it is easily provoked in persons who are debilitated by over-fatigue, anxiety, starvation, or other depressing influences. Herein, although the debility is the predisposing cause, the immediate cause is most often some severe muscular effort, and the thrombus starts in the strained or over-used muscle. Such is the origin of most of the cases of non-infective thrombosis of the upper limbs, a condition not very often met with.

## Pulmonary Embolism.

Pulmonary embolism may occur in any case of phlebitis or thrombosis, in the course of slight as well as severe attacks. The second and third weeks are the periods most liable to this danger, which is not often met with after the sixth week. Nevertheless, fatal embolism may be produced by violence, such as a blow or severe pressure applied to a blocked vein, at much later periods.

Dr. Playfair, in a paper in the *Transactions of the Pathological Society*,[2] collected twenty-five cases of thrombosis and embolism of the pulmonary artery occurring in women

[1] *Transactions of Pathological Society of London*, vol. xxxii., p. 70.

[2] *Ibid.*, vol., xviii., p. 68, and the *Lancet*, 1867, vol. ii., pp. 66, 93, 153.

after delivery, and pointed out that thrombosis occurs before the fourteenth day, often on the second or third day (fifteen cases); but that embolism does not occur until after the nineteenth day (seven cases).

The detachment of a large venous thrombus, and its lodgment in the main trunk or in one of the chief divisions of the pulmonary artery, may cause almost immediate death. This detachment of clot usually ensues upon some movement of the limb or of the body, some sudden change of posture, or some pressure upon a blocked vein. Thus, sitting up in bed, which involves flexion at the groin, stooping, kneeling, or the movements concerned in leaving or returning to bed, have often been the immediate cause of this disaster.

Sudden and intense dyspnœa occurs, with great pain in the chest, cyanosis, and feeble, irregular pulse, followed directly, or in a few minutes, by death.

When smaller branches of the pulmonary artery are blocked the symptoms are less severe, and may either gradually increase and lead to a fatal end, or diminish and be followed by recovery. In other cases a localized pneumonia, with hæmorrhagic expectoration, may ensue. I have known this sequence of symptoms to recur several times, with eventual recovery; but the condition is, of course, one of grave danger, demanding the most absolute quiet on the part of the patient.

## Pulmonary Thrombosis.

Obstruction of the pulmonary artery may also occur from thrombosis. It is probable that in some of the cases recorded as instances of pulmonary embolism the obstructing plug may have been formed *in situ*, and have been really due to thrombosis. Dr. Newton Pitt[1] has collected

[1] *Transactions of Pathological Society of London*, vol. xliv., p. 48.

orty cases of thrombosis of the pulmonary artery out of 3,218 autopsies at Guy's Hospital, and gives reasons for thinking that the condition is of much more frequent occurrence than is usually supposed.

In many cases the clot forms gradually, and is situated in the smaller branches, so that there are no sufficiently characteristic symptoms to enable a diagnosis to be made during life.[1] Sometimes, however, a thrombus may form in a large branch, giving rise to symptoms similar to those of embolism—severe and distressing dyspnœa, faintness, cyanosis, and great circulatory disturbance. These symptoms may subside, and recur at varying intervals with each addition to the clot, until at last the complete occlusion of the vessel brings about a fatal result. More rarely, thrombosis of the main trunk or its primary divisions is a cause of sudden death.

Pulmonary thrombosis occurs under similar conditions to thrombosis of other arteries and veins, especially in feeble and cachectic states with lowered vitality and weak circulation; and, although degeneration of the coats of the vessel is much less common in the pulmonary than in the other large arteries, yet such disease may be the starting-point of a thrombus.

I have collected from the post-mortem records of St. George's Hospital for the last ten years, which include 2,903 necropsies, the following cases of venous thrombosis. The list shows the relative frequency with which the veins were affected in fatal cases.

[1] *Cf.* Paget, *Transactions of Royal Medical and Chirurgical Society*, vol. xxvii., p. 162, and vol. xxviii., p. 353, who shows 'that a large and quickly increasing part of the pulmonary circulation may be arrested without immediate danger to life, or any striking indication of what has happened.'

## TABLE II.

FATAL CASES OF VENOUS THROMBOSIS, SHOWING THE VEINS AFFECTED.

| | |
|---|---|
| Iliac veins ... ... ... ... | 31 cases. |
| Inferior vena cava ... ... ... | 15 ,, |
| Lateral sinus ... ... ... ... | 15 ,, |
| Femoral ... ... ... ... ... | 12 ,, |
| Internal jugular... ... ... ... | 7 ,, |
| Saphenous ... ... ... ... | 7 ,, |
| Hepatic ... ... ... ... ... | 5 ,, |
| Cerebral cortex ... ... ... ... | 4 ,, |
| Portal ... ... ... ... ... | 3 ,, |
| Superior mesenteric ... ... ... | 3 ,, |
| Innominate ... ... ... ... | 2 ,, |
| Subclavian ... ... ... ... | 2 ,, |
| Splenic ... ... ... ... ... | 2 ,, |
| Renal ... ... ... ... ... | 2 ,, |
| Ovarian ... ... ... ... ... | 2 ,, |
| Inferior mesenteric ... ... ... | 1 case. |
| Pelvic ... ... ... ... ... | 1 ,, |
| Uterine ... ... ... ... ... | 1 ,, |

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Dr. Newton Pitt gives the following list of venous thromboses found in 3,128 autopsies during seven years at Guy's Hospital (*Transactions of Pathological Society*, vol. xliv., p. 48).

| | |
|---|---|
| Iliac veins ... ... ... ... | 34 cases. |
| Femoral veins ... ... ... ... | 33 ,, |
| Prostatic veins ... ... ... ... | 27 ,, |
| Internal jugular vein ... ... ... | 17 ,, |
| Lateral sinus ... ... ... ... | 15 ,, |
| Uterine veins ... ... ... ... | 12 ,, |
| Inferior vena cava ... ... ... ... | 12 ,, |
| Innominate veins ... ... ... ... | 9 ,, |
| Renal vein ... ... ... ... | 8 ,, |
| Pelvic veins ... ... ... ... | 8 ,, |
| Portal veins ... ... ... ... | 7 ,, |
| Broad ligament veins ... ... ... | 6 ,, |
| Hepatic veins ... ... ... ... | 4 ,, |
| Superior mesenteric veins ... ... ... | 4 ,, |
| Longitudinal sinus ... ... ... | 4 ,, |
| Popliteal vein ... ... ... ... | 3 ,, |
| Subclavian vein ... ... ... ... | 3 ,, |
| Cerebral veins ... ... ... ... | 3 ,, |

Besides these there were forty cases of thrombosis of the pulmonary artery, and twenty-six cases entered as pulmonary embolism, of which Dr. Pitt thinks some were more probably instances of thrombosis.

Table III. shows the diseases in connection with which the thrombosis occurred.

## TABLE III.

FATAL VENOUS THROMBOSIS, GIVING THE DISEASES OF ORIGIN.

| | |
|---|---|
| Middle-ear disease... ... ... ... | 14 cases. |
| Cancer (various organs) ... ... ... | 12 ,, |
| Inflamed varix ... ... ... ... | 6 ,, |
| Appendicitis ... ... ... ... | 4 ,, |
| Gastric ulcer ... ... ... ... | 3 ,, |
| Ovariotomy... ... ... ... ... | |
| Abscess of liver ... ... ... ... | |
| Peritonitis ... ... ... ... ... | 2 ,, |
| Puerperal septicæmia ... ... ... | |
| Inflammation of uterus ... ... ... | |
| Ulceration of bowel ... ... ... | |
| Heart disease ... ... ... ... | |
| Pleurisy ... ... ... ... ... | |
| Cystitis ... ... ... ... ... | 1 case. |
| Stricture and prostatic abscess[1] ... ... | |
| Pyosalpinx ... ... ... ... ... | |
| Anæmia ... ... ... ... ... | |
| Phthisis ... ... ... ... ... | |
| Suppurating ovarian cyst... ... ... | |
| Necrosis of femur ... ... ... ... | |
| Ulceration of leg ... ... ... .. | |
| Impacted tooth-plate in œsophagus ... | |
| Gonorrhœa... ... ... ... ... | |
| Lardaceous disease ... ... ... | |
| Gall-stones ... ... ... ... ... | |
| Renal calculus ... ... ... ... | |
| Cirrhosis of liver ... ... ... ... | |
| Pneumonia ... ... ... ... ... | |
| Pancreatitis... ... ... ... ... | |
| Sclerosis of spinal cord ... ... ... | |
| Suppuration of hip-joint ... ... ... | |
| Fractured leg ... ... ... ... | |
| Actinomycosis ... ... ... ... | |
| Uterine hæmorrhage ... ... ... | |
| Operation for radical cure of hydrocele... | |
| Empyema ... ... ... ... ... | |

[1] This was the only case in which gangrene occurred, and there was thrombosis of the external iliac and femoral arteries, as well as the femoral and profunda veins.

Thrombosis of the pulmonary artery occurred in eight cases in connection with the conditions shown in Table IV.

## TABLE IV.

### Diseases in connection with which Thrombosis of Pulmonary Artery occurred.

| | Sex. | Age. |
|---|---|---|
| Cirrhosis of liver ... ... ... | Female ... | 41 |
| Radical cure of hydrocele ... ... | Male ... | 47 |
| Pleurisy ... ... ... ... | ,, ... | 18 |
| Carcinoma of stomach ... ... | ,, ... | 66 |
| Gastric ulcer ... ... ... ... | ,, ... | 49 |
| Appendicitis ... ... ... ... | ,, ... | 58 |
| Tropical abscess of liver (exhaustion) | ,, ... | 32 |
| Disease of heart ... ... ... | ,, ... | 4 |

Fatal pulmonary embolism occurred in fifteen cases in connection with the conditions shown in Table V., of which it will be observed that eleven had reference to abdominal disease.

## TABLE V.

THE CONDITIONS IN CONNECTION WITH WHICH FATAL PULMONARY EMBOLISM OCCURRED IN FIFTEEN CASES.

| | Sex. | Age. |
|---|---|---|
| Thrombosis of varicose saphenous vein ... | Male | 36 |
| ,, ,, ,, ,, ... | Female | 40 |
| Ovariotomy: Thrombosis of both internal iliac veins and inferior vena cava ... | ,, | 54 |
| Ovariotomy: Thrombosis of saphenous vein ... ... ... ... ... | ,, | 41 |
| Removal of cyst of broad ligament: Thrombosis of femoral and iliac veins ... ... | ,, | 42 |
| Pyosalpinx: Thrombosis of ovarian and iliac veins ... ... ... ... ... | ,, | 29 |
| Appendicitis: Operation ... ... ... | ,, | 21 |
| Gastric ulcer: Thrombosis of internal iliac vein ... ... ... ... ... ... | ,, | 23 |
| Empyema: Thrombosis of internal iliac vein ... ... ... ... ... | ,, | 27 |
| Renal calculus: Thrombosis of femoral vein... ... ... ... ... ... | Male | 44 |
| Ulceration of rectum, pelvic abscess: Thrombosis of renal vein ... ... | Female | 43 |
| Abscess of liver: Thrombosis of hepatic veins | Male | 34 |
| Tubercular peritonitis: Thrombosis of iliac veins ... ... ... ... ... | ,, | 51 |
| Thrombosis of uterine veins ... ... | Female | 53 |
| Fractured leg: Thrombosis of veins of leg | Male | 31 |

## TABLE VI.

THE VEINS IN WHICH THROMBOSIS OCCURRED IN CONNECTION WITH FATAL PULMONARY EMBOLISM.

| | |
|---|---|
| Iliac veins ... ... ... | 6 cases. |
| Saphenous veins ... ... | 3 ,, |
| Femoral veins ... ... ... | 2 ,, |
| Inferior vena cava ... ... | 1 case |
| Renal vein ... ... ... | 1 ,, |
| Hepatic vein ... ... ... | 1 ,, |
| Uterine vein ... ... ... | 1 ,, |
| Ovarian vein ... ... ... | 1 ,, |
| Veins of leg ... ... ... | 1 ,, |

The symptoms of simple non-infective phlebitis are chiefly those of a localized thrombosis; the constitutional disturbance if the case is uncomplicated is not usually severe. There may be an initial rigor, followed by a rise of temperature and pulse, but more often the first noticeable symptom is pain, felt most commonly in the left calf. This pain may at first be intermittent, subsiding for a few hours and then recurring with increased severity; if the deep veins are affected, it is of cramp-like character. These symptoms are coincident with the formation of a thrombus and the commencement of phlebitis. If the phlebitis spreads the temperature rises, perhaps to 101° or 102° F., and there is sometimes sweating; pain increases, especially if the large veins are concerned, and there is local tenderness. The amount of œdema is variable, for the occurrence of œdema in cases of venous obstruction depends upon a variety of conditions, and is by no means the simple matter which it has been sometimes represented. It results from 'a disturbance of the normal equilibrium which exists between blood, bloodvessels,

tissues, and lymphatics.'[1] Much will depend upon the rapidity, extent, and position of the thrombosis, the condition of the venous coats, the possibility of collateral circulation, the force of the blood-stream, the precedent degree of venous and arterial pressure, and the composition of the blood. Variations in these factors account for the remarkable differences observed as to the occurrence of œdema in venous obstruction ; thrombosis of the femoral vein or inferior vena cava, for instance, may be attended by extreme œdema, by very little, or by none.

Doubtless the production of œdema depends largely, as Cohnheim taught, upon increased intravascular pressure, increased permeability of the vessel wall, or both of these conditions. The obstruction to the return of venous blood caused by the thrombus will give rise to increased intravascular pressure behind the obstruction, and the interference with the nutrition of the capillary endothelium will lead to increased permeability of the capillary walls, and therefore to the easier transudation of serum. This will also be influenced by the hydrostatic pressure, as seen in the effect of position, and the greater frequency of œdema in connection with thrombosis of the lower than of the upper limbs.

But this is not sufficient to account for all the phenomena of œdema ; for if the lymphatics can carry off the effused fluid there will be no œdema. And the facility of absorption depends not only upon the quantity, but also upon the quality of the fluid to be absorbed. The larger the amount of proteid contained in the fluid, the slower and more difficult is absorption, and watery solutions of crystalloids are absorbed with a rapidity proportionate to their dilution. The presence or absence of inflammation is therefore of importance in the production of œdema,

[1] Lazarus-Barlow, 'Manual of Pathology,' 1904, p. 218.

for if inflammation is present the fluid which escapes into the tissues contains more proteid and is of higher specific gravity than that which escapes as the result of mere venous congestion.

The influence of the nervous system must also be taken into account, for, as shown by Cornil and Ranvier, if in an animal a vein is tied and the vaso-motor nerves divided, the arteries dilate, more blood is carried to the part, and the tension in the capillaries leads to exudation of fluid and to œdema. Ranvier had previously shown that if the inferior vena cava is ligatured and the sciatic nerve is divided in one limb, œdema only occurs in the limb in which section of the nerve has been made.

Dr. Lazarus-Barlow, who has done important work in this relation, has called attention to the part played by the tissues in the production of œdema. He says:[1] 'It is astonishing how in all discussions concerning lymph and œdema formation the tissues have been left out of consideration, when we remember that every condition which affects the small bloodvessels, and especially the capillaries, must at the same time affect the tissues also. In some cases even in which œdema occurs the tissues are affected first and to the greatest extent. It is a fault in the mechanical explanation both of lymph and of œdema formation that it places the tissues absolutely at the mercy of the vascular system. The amount of lymph which the tissues receive, according to that explanation, does not depend upon the needs of the tissues, but upon the condition of the bloodvessels. And yet the whole *raison d'être* of the circulating system is the existence of the tissues. Normal lymph formation and œdema formation must be the ultimate result of at least two processes, one in which the tissue cells are paramount, the other in which the bloodvessels are paramount.'

[1] 'Manual of Pathology,' p. 202 note.

During hæmostasis the tissues are affected in two ways: they are deprived of nutriment, and the waste products of their own metabolism are not removed. This leads to an active arterial congestion and an increased flow of lymph. Part of this lymph is carried away by the lymphatics, but if there is more than can be so disposed of œdema results.

It is evident that in venous thrombosis both these conditions are present: there is diminished nutrition of the tissues and accumulation within them of the products of their metabolism. Upon this follows an increased flow of lymph, and upon the capacity of the lymphatics to carry this away depends the occurrence or not of œdema. It has already been pointed out that the degree in which absorption of effused fluid takes place depends upon the nature of the fluid, and this again will depend upon the composition of the blood, the condition of the vessels, and the presence or absence of inflammation.

Enough has been said, I think, to show that the production of œdema in venous thrombosis is the result of a variety of complex conditions. Moreover, it will depend somewhat on the size and position of the affected veins.

If a superficial vein is inflamed, the skin over it shows a dull red line wider than the vein, along which is an area of tenderness; the vein may be felt solid with clot; there is little or no œdema. If the intramuscular veins are implicated there will be more pain, and some deep swelling, the limb feeling tight and heavy, but showing little superficial œdema. If a main trunk is obstructed, as the femoral, there will be more general œdema, and the limb may become tensely swollen. There may still be but little constitutional disturbance, and the chief danger to be apprehended is the detachment of clot. If the patient is kept at rest these symptoms may gradually subside, the circulation through the veins involved may become

re-established, and the limb after a time completely regain its normal condition. But in many cases some of the affected veins are permanently obliterated, and when large trunks are involved they are often left with the walls thickened and lumen diminished by the adhesion and shrinking of organized clot (Fig. 4). I have found some of the smaller veins of the calf blocked by firm adherent clot in cases where the limb appeared to have completely recovered from attacks of phlebitis. The veins of the lower extremity are much more often attacked than those of the upper limb, and those of the left more often than those of the right limb. The disease begins very commonly in the deep veins of the left calf. In this class of cases there is not so great a liability to recurrence as in those of gouty origin. I have had the opportunity of observing several patients for varying periods up to ten years, in whom there has been no recurrence after a severe first attack.

A satisfactory subsidence and recovery is, however, not always the rule. The thrombus may extend in the direction of the blood-current, and so to the larger trunks, causing increasing embarrassment to the circulation. Thus from the femoral vein coagulation may spread through the iliacs to the inferior vena cava, or from the veins of the neck and upper extremity to the superior cava, and thus to the heart. Professor Humphry described such cases with characteristic accuracy in a thesis on coagulation of the blood in the veins, published in 1859.[1] I have seen a case in which the thrombus extended as high as the renal vein, and in which recovery took place, but with permanent obstruction of part of the

[1] 'On Coagulation of the Blood in the Venous System during Life,' by George Murray Humphry, M.D., F.R.S.: Macmillan, Cambridge, 1859.

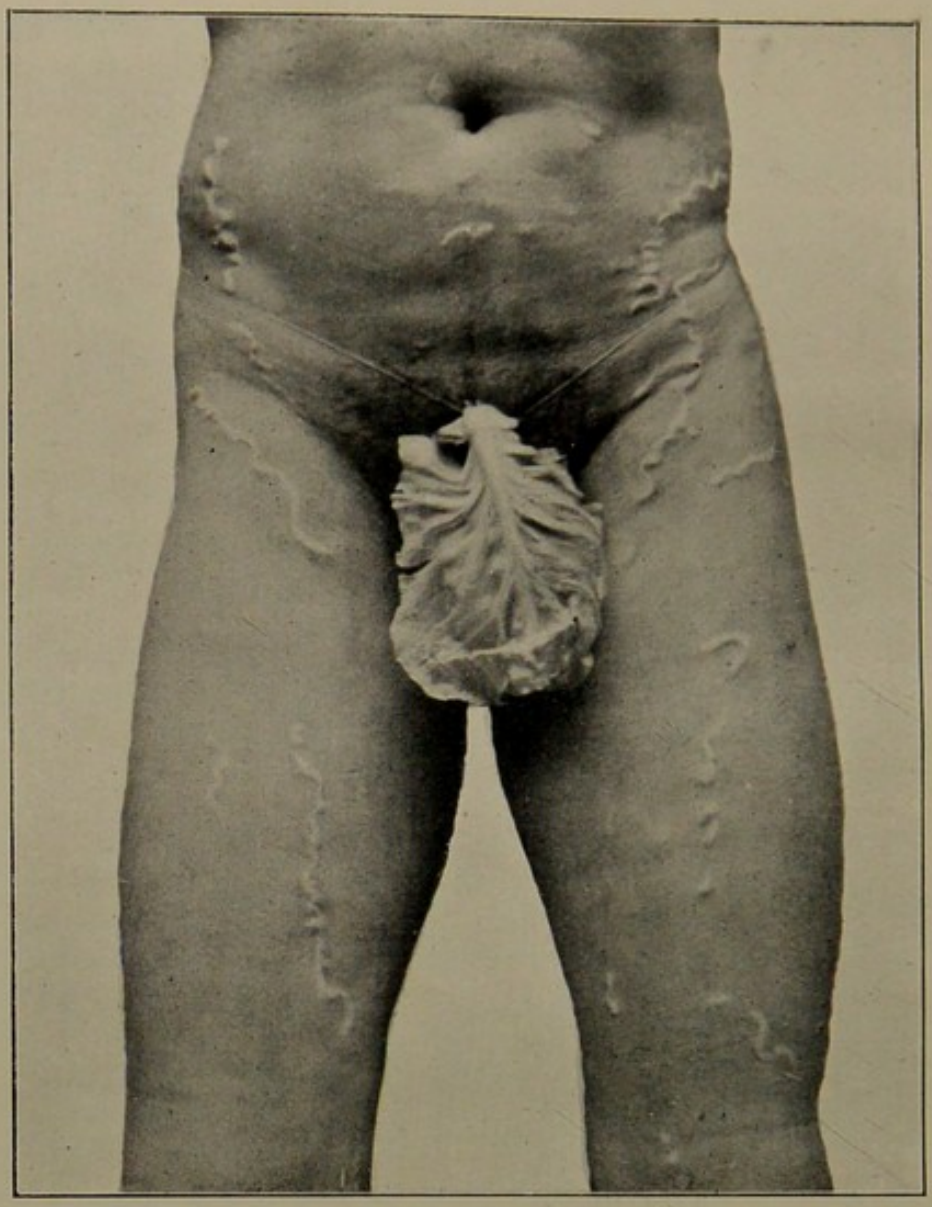

FIG. 6.—PHOTOGRAPH SHOWING THE DEVELOPMENT OF THE SUPERFICIAL VEINS TWENTY-ONE YEARS AFTER OBLITERATION OF PART OF THE INFERIOR VENA CAVA.

[*To precede Fig. 7.*

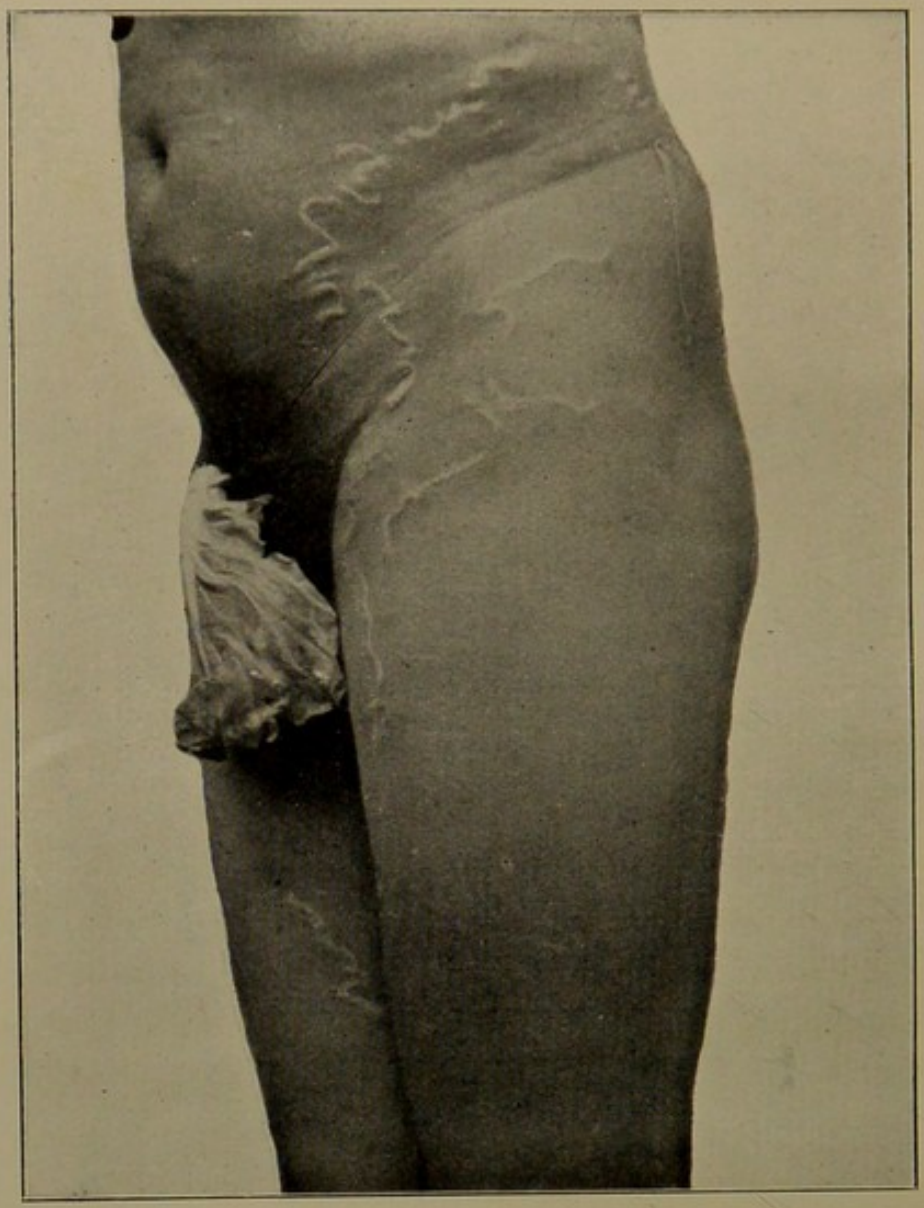

FIG. 7.—PHOTOGRAPH SHOWING THE DEVELOPMENT OF THE SUPERFICIAL VEINS TWENTY-ONE YEARS AFTER OBLITERATION OF PART OF THE INFERIOR VENA CAVA.

*To face p.* 59.

vena cava. When this occurs an enormous development of the superficial veins of the groin and abdominal wall usually takes place for carrying on the collateral circulation. A similar condition has been observed in connection with obstruction of the superior vena cava, though this event is rare except in consequence of the pressure of intrathoracic tumours.

Figs. 6 and 7 are from photographs, showing the development of the superficial veins twenty-one years after obliteration of part of the inferior vena cava. In this patient it took about ten years for these veins to reach a troublesome degree of enlargement, and it is only lately that the circulation has become completely compensated, so that now there is no difference in the size of the limbs at night and in the morning, and the outline of the muscles is easily perceptible.

Sir Thomas Watson,[1] in his 'Lectures on the Principles and Practice of Physic,' has described in his inimitable manner two cases illustrating the effects of obliteration of the venæ cavæ, and has given diagrams which show the collateral circulation.

One of these was the case of a man who exhibited in a remarkable degree the results of obliteration of the superior vena cava by an aneurism of the innominate artery. The whole surface of the thorax in front, with that of the shoulders and of part of the abdomen, was thickly overspread with a network of prominent veins, 'whereby the blood descending from the head found its way at length, through many circuitous channels, to the heart.'

The second case related by Sir Thomas Watson was that of a woman in whom 3 inches of the inferior

[1] 'Lectures on the Principles and Practice of Physic,' 1857, vol. ii., p. 350.

vena cava was obliterated by the pressure of a tumour of the liver, a great development of the superficial veins of the thorax and abdomen contributing to the collateral circulation. Sir Thomas Watson draws attention to the tortuosity of the veins in which the direction of the current is retrograde.

Mr. C. Mansell Moullin[1] has recorded a case of thrombosis of the inferior vena cava following a fall on the back and over-extension of the spine. The vein was obliterated from a point immediately below the entrance of the renal vein, and the left common and external iliacs and the femoral vein were blocked. The right leg was scarcely œdematous; the left was 6 inches more in circumference, hard, brawny, and severely ulcerated. The contrast between the two limbs was striking and showed that even when the inferior cava has been obliterated, if the other veins are not interfered with, a collateral circulation may be established quite sufficient for all ordinary purposes.

But complete and permanent obstruction of the inferior vena cava may occur without any marked development of the superficial veins, as in a case examined at St. George's Hospital, where the collateral circulation seemed to have been carried on almost entirely by the greatly-enlarged azygos veins. The patient, a woman of fifty years, died of pneumonia; and post-mortem it was discovered that the inferior vena cava, from just above the entrance of the right renal vein to immediately below the entrance of the hepatic vein, had been converted into an impervious fibrous cord. The azygos veins were greatly dilated and tortuous, but the veins of the surface of the body were not dilated nor prominent.[2]

Dissections of similar conditions in cases of obliterа-

[1] *Transactions of Clinical Society of London*, vol. xvii., p. 115.

[2] 'Post-mortem and Case Book,' 1896, No. 145.

tion of the vena cava were described many years ago by Dr. Matthew Baillie[1] and by Mr. Wilson.[2]

Uncomplicated peripheral venous thrombosis does not cause gangrene. When this does occur it is in consequence of arterial as well as venous obstruction, or else of the addition of inflammatory disturbance.

[1] *Transactions of Society for the Improvement of Medical and Chirurgical Knowledge*, vol. i., p. 127, plate v.

[2] *Ibid.*, vol. iii., p. 65. See also Dr. Peacock in *Transactions of Royal Medical and Chirurgical Society*, vol. xxviii., p. 1, 'On Thrombosis of the Vena Cava Superior ;' with References to other Published Cases.'

# LECTURE III

Thrombosis of—(*a*) cerebral sinuses; (*b*) mesenteric veins; (*c*) gastric veins; (*d*) portal veins; (*e*) hæmorrhoidal veins (ischiorectal abscess); (*f*) renal veins; (*g*) splenic veins; (*h*) prostatic veins; (*i*) corpus cavernosum.

Treatment of thrombosis and phlebitis.

Remote effects of thrombosis: their treatment. — Importance of developing deep collateral veins.—Cause of enlargement of the limb.

Effects of venous obstruction on the heart.

General management of cases of blocked veins.

SIMPLE non-infective *thrombosis of the cerebral sinuses* is met with chiefly in overworked chlorotic young women, in children who have suffered with long-continued diarrhœa, and in the subjects of exhaustion such as ensues towards the end of fevers. It may also be the result of injury.[1] It commences most often in the longitudinal sinus, and gives rise to much cerebral congestion, with minute hæmorrhagic extravasations into the brain substance and fluid distension of the ventricles. There may also be optic neuritis. The symptoms are severe headache, drowsiness, vomiting, convulsions, and paralysis. These may subside and end in complete recovery, or they may go on to coma and death.[2]

[1] Mr. Artbuthnot Lane has recorded a case of thrombosis of the longitudinal sinus after fracture of the parietal bone, there being no discoverable injury of the sinus (*Transactions of Clinical Society*, vol. xxiii., p. 219).

[2] Professor Humphry, in the thesis alluded to, gives an admirable description of such a case, with the post-mortem examination (*op. cit.*,

I have seen an instance in a boy recovering from scarlet fever, in whom, besides convulsions and coma, there was protrusion of the globe of the eye, chemosis, and almost complete interference with ocular movement, presumably due to thrombosis of the cavernous and petrosal sinuses. In spite of these serious symptoms the boy recovered.

In infective cerebral thrombosis there will be added to the symptoms described the rigors, sweating, and irregular temperature significant of pyæmia.

Dr. Newton Pitt, in the Gulstonian Lectures delivered before the Royal College of Physicians in 1890,[1] gives a most valuable analysis of forty-four cases of sinus thrombosis.

| | Primary. | Secondary. | | |
|---|---|---|---|---|
| | | Ear Disease. | Other Causes. | Total. |
| Longitudinal sinus ... | 5 | 4 | 3 | 12 |
| Lateral sinus ... | 5 | 22 | 9 | 36 |
| Cerebral veins ... | 5 | 1 | 1 | 7 |
| Cavernous sinus ... | — | 1 | 3 | 4 |
| Circular sinus ... | — | 1 | 1 | 2 |
| Inferior petrosal ... | — | 1 | 1 | 2 |
| Superior petrosal ... | — | 2 | — | 2 |

Primary thrombosis occurs, Dr. Pitt points out, in cases 'with a feeble cerebral circulation, associated with exhausting diseases.' 'In this form, owing to the stagnation of the circulation, clotting tends to take place in the sinuses; the clot becomes adherent, but seldom infects the blood or gives rise to emboli.'

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p. 32). Dr. J. W. Ogle recorded a case of thrombosis of the cerebral veins and sinuses in a woman who died exhausted by rectal disease, in whom the symptoms were only those of exhaustion until a short time before death, when there was loss of the power of speech, the mind remaining unaffected (*Transactions of Pathological Society of London*, vol. vi., p. 31).

[1] *Brit. Med. Journ.*, 1890, vol. i., p. 774.

Secondary thrombosis occurs in connection with adjacent diseases, chiefly of the ear. Of thirty-six cases, twenty-two were due to ear disease, and in eleven of these the clotting spread to the jugular vein. In the majority of the cases the symptoms were mainly those of pyæmia, three-quarters of them dying from pulmonary infection. 'Of the remaining cases, seven were traumatic, three had spread from malignant pustules or carbuncles on the face, two from an adjacent pachymeningitis, one from compression by a growth, and only one from a distant source —namely, from pyæmia set up by a carbuncle on the back.'

Dr. A. Brayton Ball[1] has published an interesting contribution to this subject, in which he makes a similar division of cases of intracranial venous thrombosis into two classes presenting marked differences in their etiology and symptomatology. He attributes secondary thrombosis either to the propagation of a phlebitis, or to the direct prolongation of a thrombus having origin in otitis media, in disease of the cranial bones, in septic wounds, and in carbuncular or erysipelatous inflammation of the face and head. Primary thrombosis he connects chiefly with debilitating illness and enfeebled circulation, and he draws especial attention to the cases occurring in anæmic and chlorotic girls without any preceding symptoms. Dr. Ball quotes a number of illustrative cases, which exhibit the variety and mobility of the symptoms; and he points out that, 'apart from the fact that these symptoms often show a mobility that is uncommon in most cerebral affections, there is nothing distinctive in their character. The diagnosis in this class of cases, when possible at all,

[1] *Transactions of the Association of American Physicians*, vol. iv., p. 52., 'Thrombosis of Cerebral Sinuses and Veins,' by A. Brayton Ball, M.D., with a bibliography of the subject.

must be made from the association of anomalous cerebral symptoms with the anæmic state, from evidence in external veins of backward pressure from the intracranial venous circulation, and from the occurrence of thrombosis in other parts of the body, particularly the internal jugular or the veins of the upper or lower limbs.'

*Thrombosis of the mesenteric veins* is most often associated with intestinal ulceration. When it occurs in connection with peripheral thrombosis the onset is usually sudden and the symptoms severe. Vomiting, rapid intestinal distension, griping pain, and intestinal paralysis, are the chief symptoms, leading often to rapid death.

I have seen a case in which, during the course of a femoral phlebitis, there were symptoms indicative of thrombosis of a *gastric vein*. The patient was suddenly attacked with vomiting, followed by a great gastric and intestinal distension, dyspnœa, and severe pain in the upper part of the abdomen. The distension gradually diminished, but from the moment of the attack there was complete anorexia and an absolute cessation of salivary secretion. The digestive function was for a time in almost complete abeyance: only very small quantities of peptonized fluid food could be taken; and if the fluid entered the stomach when the patient was lying on the left side, acute pain was felt, which at once subsided if the patient was turned on to the right side. The suppression of salivary secretion and consequent dryness of the mouth led to the collection and decomposition of pharyngeal mucus, which, in spite of the frequent use of antiseptic washes, caused great distress. After passing through a period of great exhaustion the patient ultimately recovered.

Dr. Rolleston has recorded in the *Transactions of the*

*Pathological Society*[1] a case in which there was thrombosis of the superior and inferior mesenteric veins, of the left internal and external iliac veins, and of the splenic vein, of a man who died after profuse diarrhœa and hæmatemesis, and in whom there was ulceration of the vermiform appendix.

Dr. Hilton Fagge[2] described a case of acute thrombosis of the superior mesenteric and portal veins, with rapidly fatal collapse. A woman, aged thirty-four, had thrombosis of both femoral veins sixteen days after confinement. On the thirty-sixth day she was attacked with violent pain in the abdomen and vomiting. Collapse rapidly succeeded: 'the eyes were sunken, pulse almost imperceptible, pain coming on in paroxysms, but never completely intermitting, severe retching, and frequent vomiting of a rather viscid blood-stained liquid in small quantity.' She was perfectly conscious till death, eleven hours from the onset of the attack.

*Thrombosis of the portal vein* may arise by extension from a mesenteric vein or from gastric or intestinal disease. It may be due to pressure by tumours or by the interstitial growth of cirrhosis or syphilis; and it may depend upon disease, degeneration or injury of the coats of the vein. The symptoms will vary with the rate at which the thrombus is formed. When the thrombosis is acute, ascites is rapidly developed (the fluid quickly reaccumulating after tapping), and there may be hæmatemesis, intestinal hæmorrhage, and other evidences of portal obstruction. If the patient survives, a notable development and dilatation of the superficial veins of the abdominal wall becomes apparent. If the process is gradual there may be few or no characteristic symptoms.

[1] *Transactions of Pathological Society*, vol. xliii., p. 49.

[2] *Ibid.*, vol. xxvii., p. 124.

Professor Osler[1] has described the case of a man, aged sixty-two, who died two days after admission to hospital, suffering from ascites and hæmatemesis. Post-mortem the liver was found to be in a state of advanced cirrhosis, and contained a large infarct. 'The portal vein presented a soft brown thrombus, occupying the upper part of the trunk, but not completely obliterating it; the branches passing to the right lobe had closely-adhering light brown thrombi; that passing to the antero-lateral region, where the infarct was situated, was filled with a firm, solid, partially laminated clot, evidently formed some time before death.'

In this case Professor Osler points out that the rare condition of infarction of the liver probably depended upon the cirrhosis and the consequent obliteration of many of the branches of the hepatic artery.

In contrast with this, Professor Osler has related[2] the case of a man, aged twenty-eight, in whom there had been for years complete obliteration of the portal vein. The collateral circulation was, however, so fully compensatory that there was no material interference with the functions of the organs. There were extensive communications between the gastric and œsophageal veins, and through the latter with the azygos and lower intercostal veins and those of the diaphragm, all of which were greatly enlarged.

Professor Welch[3] has recorded a case in which there seemed good reason to attribute the formation of a portal thrombus to a blow on the abdomen. The case is one to be borne in mind in regard to abdominal injuries. 'A lad who had received a severe blow on the abdomen was

[1] *Transactions of Association of American Physicians*, 1887, p. 137.
[2] *Journal of Anatomy and Physiology*, London, 1882, p. 208.
[3] Allbutt's 'System of Medicine,' vol. vi., p. 220.

admitted into Belle Vue Hospital with extreme ascites, which had come on within two weeks after the injury. He was repeatedly tapped, the clear fluid reaccumulating at first with great rapidity after each tapping, afterward more slowly, until, in the course of months, there was complete recovery. In the meantime enlarged veins made their appearance over the upper part of the abdomen.'

The dilated veins constituting *venous hæmorrhoids* are very prone to become thrombosed, especially when protruded through the sphincter. The clot herein is liable to the invasion of micro-organisms, and the extension of this septic clot frequently leads to suppuration, and thus to the formation of ischiorectal abscess. I have several times traced the thrombosed vein from an inflamed hæmorrhoid into the ischiorectal cellular tissue to the spot where suppuration has occurred, and I am sure that this is no uncommon mode of origin of ischiorectal abcess.

*Thrombosis of the renal veins* may be primary and marantic, or may be the consequence of extension from the vena cava; it may also depend upon disease of the kidney and upon the pressure of neighbouring tumours. When of gradual occurrence the symptoms may be few and indistinguishable, and the collateral circulation may be sufficiently developed for complete recovery. When of sudden onset it is manifested by the appearance of blood or albumin in the urine, and may be rapidly fatal.

Dr. W. W. Ord[1] has recorded the case of a boy, aged one year, who was recovering from scarlet fever, and who was suddenly seized with abdominal pain and vomiting, rapidly succeeded by collapse and death in four hours.

[1] *Transactions of Pathological Society of London*, vol. xlvi., p. 39.

Post-mortem, the left kidney was swollen and of dark purple colour, and under the microscope showed great engorgement of its vessels, with numerous small extravasations; but there were no signs of interstitial nephritis. There was thrombosis of the renal vein extending into, but not occluding, the vena cava.

Dr. Theodore Fisher[1] has published the case of a girl, aged thirteen, who had been the subject of old hip disease, and who was admitted into the Bristol Royal Infirmary on account of general œdema of three weeks' duration. There was no history of scarlet fever. The urine contained 3 per cent. of albumin, some blood, and granular casts. The quantity of urine passed varied between 15 and 46 ounces in the twenty-four hours during the first week. It then diminished rapidly to only 12 ounces the day before death, which occurred eleven days after admission. Post-mortem, the kidneys were enlarged and the cortex swollen, pale yellow, and opaque. There was thrombosis of both renal veins and of the vena cava, and also of the pulmonary arteries. The clot in the renal veins was becoming organized, and that in the pulmonary artery was channelled.

Beckman[2] states that thrombosis of the renal veins is no uncommon occurrence in infants dying with profuse diarrhœa and atrophy. He has examined ten cases carefully. The left renal vein was the one most often affected; in a few cases the thrombus extended into and obstructed the inferior cava. The coagula were mostly dark red, and loosely adherent to the walls of the vessels. No blood in the urine, or other symptoms, seem to have been observed during life.

[1] *Transactions of Pathological Society of London*, vol. xlvii., p. 113.

[2] 'Year-Book of Medicine and Surgery,' 1860, p. 203 (New Sydenham Society).

*Thrombosis of the splenic vein* is usually due to extension from the vena cava. It may be associated with thrombosis of other abdominal veins, as in Dr. Rolleston's case already alluded to;[1] it may be the consequence of suppuration or cancerous disease of the pancreas; and it may be caused by degeneration of the walls of the splenic vein. As the process is seldom confined to the splenic vein, there are no characteristic symptoms, excepting perhaps, a rapid swelling of the spleen.

*Thrombosis of the prostatic veins* would seem, from post-mortem evidence, to be not very rare, and may perhaps be the result in some cases of a past prostatitis. It is probably also in some cases the first step in the causation of prostatic abscess.

*Thrombosis in the corpus cavernosum* of the penis is occasionally met with, but is not very common. It is usually associated with the gouty constitution or with syphilis, but I have seen a case in which no such connection could be traced.

Sir Prescott Hewett[2] recorded two cases of thrombosis of the corpus cavernosum in men of sixty-five and fifty-eight years, both gouty. In one case there was a single, hard, painless nodule; in the second case there were four nodules, three on the left and one on the right side, 'varying in size from a pea to that of a French bean. They were perfectly circumscribed, hard to the touch, knot-like, and painless when handled.' The nodules all gradually diminished, and in two years two of them had disappeared, no treatment having been adopted.

Sir Dyce Duckworth[3] has recorded a case of painful priapism, due to thrombosis of the corpus cavernosum,

[1] *Transactions of Pathological Society of London*, vol. xliii., p. 49.
[2] *Transactions of Clinical Society of London*, vol. vi., p. xl.
[3] *Ibid.*, vol. xxv., p. 97.

which occurred in a man of forty-two years, during a distinct attack of acute gout. The condition persisted for three weeks, and gradually disappeared.

Sir Dyce Duckworth[1] also quotes two similar cases—one in a very gouty man of forty-five years, in whom the condition lasted three weeks; and a second in a man of sixty years, also gouty, in whom a second attack occurred after a year's interval.

The *treatment* of a case of simple, non-infective phlebitis or thrombosis consists chiefly in the enforcement of complete rest. The point to be kept in mind is the importance of taking every possible precaution against the detachment of clot. This is the chief danger, especially when the larger venous trunks are involved, and it belongs to the slight as well as to the severe cases. The patient must be at once and rigidly confined to bed, and must avoid all exertion; especially must he be warned against any straining effort, as, for instance, in defæcation. The bowels, therefore, must be kept acting easily by gentle laxatives, and, of course, the bed-pan must be used. The neglect of this precaution has led to many disastrous results. If there be troublesome cough, laryngeal sedatives must be used and easy expectoration promoted, so as to avoid the disturbance and effort which cough involves.

In gouty phlebitis the amount of solid food should be diminished, but distilled water should be taken freely (2 or 3 pints in the twenty-four hours). This should be mixed with a carminative (as ginger), as care should be taken to avoid flatulence, for any distension of the abdominal viscera is a serious embarrassment to the circulation. An occasional dose of calomel is usually beneficial.

[1] *Transactions of Clinical Society of London*, vol. xxv., p. 100.

I have already alluded to Professor Wright's[1] observations, showing that the administration of citric acid is followed by a decalcification of the blood and a corresponding diminution of its coagulability.

Mr. C. Nepean Longridge has kindly furnished me with some observations upon lying-in women to the same effect. He also found that by taking citric acid the coagulation time of his own blood was increased from two minutes forty-five seconds to three minutes thirty-five seconds, without any change of diet. I think, therefore, that citric acid should certainly be given for the treatment of thrombosis, as well as for its prevention in conditions in which that danger is likely to occur. Having in mind also the difficulty of certainly excluding the possibility of an infective origin, and of separating clinically the infective and non-infective cases, I hold it wise to give quinine in moderate doses. It also seems to me that Professor Wright's[2] suggestion, as to decalcifying the milk by the addition of citrate of soda, should be adopted wherever large quantities of milk are given to patients prone to thrombosis. The same author has also proved experimentally[3] that the coagulability of the blood is diminished by ingestion of alcohol, by drinking large quantities of fluid, and by reducing the supply of food. Water, therefore, should be freely taken, and freshly-made lemonade is a useful beverage. The diet should be light and easily digestible, and may perhaps with advantage include some

[1] *Brit. Med. Journ.*, July 14, 1894, p. 57, and the *Lancet*, October 14, 1905, p. 1096.

[2] *Transactions of the Royal Medical and Chirurgical Society*, vol. lxxxvi., p, 1. See also the *Lancet*, July 22, 1893, p. 194, Dr. A. E. Wright on 'The Advantages of Decalcified Milk in the Feeding of Infants and Invalids'; and Dr. F. J. Poynton on the same in the *Lancet*, 1904, vol. ii., p. 433.

[3] *Transactions of Pathological Society of London*, vol. li., 1900, p. 298.

alcohol. Tobacco-smoking would seem to be harmless, or perhaps even beneficial.

Dr. B. W. Richardson[1] made a great number of experiments on the blood of a gentleman who was addicted to constant smoking. He observed that in the morning after rising, and before smoking, the blood coagulated in two minutes with moderate firmness, and the corpuscles were generally natural in shape. 'When two or three pipes had been smoked, a distinct change occurred in the blood: it retained its brightness, but flowed freely and coagulated slowly and feebly. After a long day of smoking—from fifteen to twenty-five pipes having been taken—the blood, though still retaining its bright colour, flowed much more readily, and would sometimes refuse to coagulate altogether. Left for a time in the cup of a microscope-glass, it would thicken from a sort of drying process, but would not healthily coagulate.' Under the microscope the red corpuscles showed some irregularity of shape and did not form rouleaux. After a night's rest the blood resumed its normal condition.

Dr. Richardson also observed that the blood of jaundiced persons coagulated slowly and feebly. He also found that the administration of ammonia caused similar changes in the corpuscles, and a decrease in the coagulability of the blood.

It has been observed that

*The Coagulability of the Blood is increased by—*

Carbonic acid.
Lime salts (calcium chloride, calcium lactate).
Milk.
Magnesium carbonate.
Restriction of fluid.

[1] 'The Cause of the Coagulation of the Blood,' by B. W. Richardson, M.D., 1858, p. 101.

*The Coagulability of the Blood is diminished by—*

Oxygen.
Alcohol.
Ammonia.
Restriction of food.
Diminution of lime salts.
Large quantities of fluid.
Citric acid.
Rhubarb.
Acid fruit-juices.
Acid wines.
Tobacco-smoking.

The treatment must, however, be modified to meet the special conditions of those in whom the phlebitis is a complication of other serious diseases, as, for instance, in chlorosis, influenza, and enteric fever. I do not think that in any case depletion is desirable. The local measures should be directed to relieving pain and insuring rest to the affected limb. For the relief of pain nothing is so effectual as the application of heat; and, as it is most important to disturb the limb as little as possible, it is best to use dry heat. Thin flannel bags, filled with bran and heated in an oven, are convenient for this purpose; they are light and retain heat for a considerable time. When only a small portion of vein is involved, a hot lead and opium lotion, or a fomentation of boracic lint covered by jaconet, is comfortable; but the extensive use of wet applications is inconvenient and involves frequent disturbance. When pain is not severe, the most comfortable dressing is a layer of Gamgee tissue secured round the limb by a many-tailed bandage, the skin having been first dusted over with powdered boracic acid. If the inflamed vein is superficial and tender, it is well to paint along the line of redness a thick lotion of oxide of zinc, glycerine, and carbolic acid in water. I have never seen the least advantage from the application of belladonna

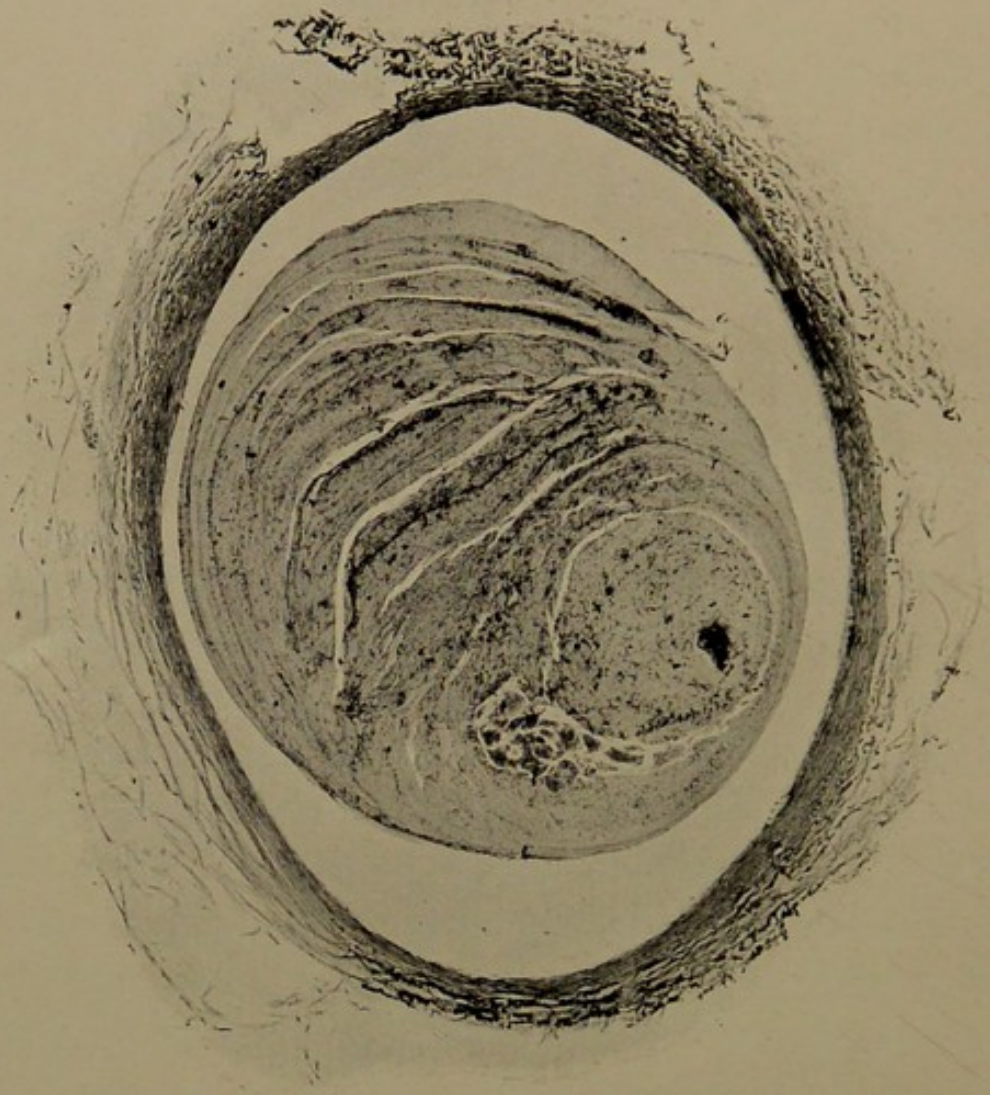

FIG. 8.—THROMBOSIS OF FEMORAL VEIN, BUT NO THICKENING OF VENOUS COATS.

[*To face p.* 75.

which, though often recommended, seems to me to be disagreeable and useless. When the veins of the upper limb are affected, the arm should be extended upon a pillow raised slightly above the level of the body and away from the side. It is best secured by wrapping a second pillow partially round the limb and fixing it by two or three separate strips of wide bandage. In this way sudden movements in sleep or otherwise are prevented. A similar arrangement can be adapted to the leg, or a well-padded splint may be applied and the limb slung from a cradle.

I wish now to consider some of the more remote effects of phlebitis, for it seems to me that too little attention has been paid to conditions upon the management of which will often depend much of the future comfort and activity of the patient.

After a severe attack of phlebitis, especially if the large venous trunks have been involved, some of the affected veins will probably be left more or less obstructed. The lumen of the vein may be completely filled by blood-clot, which, becoming gradually organized, obliterates the canal, and converts the vein into a solid cord; or the clot may be tunnelled and a channel for the blood remain, either through the thrombus or between it and the wall of the vein. In non-infective phlebitis or thrombosis the outer coat of the vein is not materially thickened, and the diminished lumen of the vessel depends upon the organization and contraction of the adherent clot (Fig. 8). But in phlebitis originating in an infecting focus or wound the outer coat is always much infiltrated, and may be left permanently thickened and indurated (Fig. 4). In this way, and by subsequent contraction, the lumen of the vessel is encroached upon and much diminished, and this may be still further narrowed by the addition of organized clot from within.

When a main venous trunk, such as the femoral or iliac, is permanently blocked, certain changes result in the limb concerned. I will take as an example a case in which the femoral and external iliac veins are obliterated. At first, whenever the limb is dependent or the upright posture is assumed, the veins, both superficial and deep, become full, the limb becomes painful, tense, and somewhat swollen, and a sense of constriction is felt at the groin and in the popliteal space. The skin presents a dusky, congested appearance; the muscular power is diminished; and, after walking a short distance, severe aching pain occurs, which makes a rest imperative. If the patient is now placed recumbent and the limb elevated, the veins gradually become less full, the skin recovers its normal colour, and the pain subsides; but the limb remains somewhat larger and firmer than its healthy fellow, and the contraction of the superficial muscles is less easily seen. The collateral venous circulation begins to develop almost immediately upon the blocking of the main trunk, and in the course of a month or six weeks some enlargement of the superficial veins at the groin and lower part of the abdominal wall becomes manifest (superficial epigastric, superficial circumflex iliac, external pudic, etc.). This goes on gradually increasing until the veins attain an enormous size, and a number of tortuous veins $\frac{1}{2}$ inch or more in diameter extend over the groin and abdominal surface (Figs. 6 and 7, p. 59).

At the same time the deep communicating veins are enlarging, and as the collateral circulation becomes developed, the upright posture causes less congestion and swelling, walking is less painful, and the limb recovers more quickly when rested. But the enlarged superficial veins, although assisting the return of the venous blood, are themselves the cause of much local discomfort; their

distension gives rise to painful aching, and their prominence and lack of support render them liable to injury. It is, therefore, desirable in all cases in which a main venous trunk is blocked, to favour as far as possible the development of the deep collateral vessels, and to prevent the excessive enlargement of the superficial veins. It will be remembered that the return of the blood through the deep veins is assisted by muscular contraction, which has but little effect upon the veins of the surface; and that the deep veins, having the support and protection of the tissues among which they are placed, do not become easily over-distended or varicose and are not liable to injury. Evidently, therefore, it is to be desired that the collateral circulation should depend as much as possible upon the deep veins, and as little as possible upon those of the surface. This is, I think, a matter of considerable importance in the management of such cases, and a good deal can be done in the direction indicated. In the first place, it is necessary to decide at what period the use of the limb may safely be attempted. This requires careful consideration, for while, on the one hand, there is the danger of displacement of clot if the limb is used too soon; on the other hand, the functional activity of the muscles is the best aid towards the development of the deep collateral veins.

Pulmonary embolism is not common after the sixth week from the formation of a thrombus, although instances have been recorded of the detachment of a clot by a blow or pressure at later periods. There are great variations in the time required for the organization of thrombi. It must be borne in mind that this process is really the invasion of the thrombus by active cells and new vessels, and the gradual replacement of the blood-clot by connective tissue. This change needs for its favourable progress the functional

activity of the venous wall, and is inevitably delayed by the presence of septic organisms. In simple non-infective cases evidence of commencing organization may be found in a thrombus at the end of a week, whereas in veins the walls of which have undergone degeneration very little change may have occurred at the end of a month.

In an uncomplicated case of phlebitis it will probably be safe at the end of six weeks from the last extension of the thrombus to commence some movement of the limb, but this should not be permitted as long as there is any tenderness over the vein, for tenderness implies the possibility of further extension of thrombus; moreover, the first movements should, of course, be made with great gentleness and caution. In cases of septic origin, and also in cases in which there is disease or degeneration of the veins, a longer time should be allowed before movement.

The organization of thrombus in varicose veins is usually very slow, and when thrombosis occurs in cysts and tortuous dilatations, especially in veins near the knee or in the long saphenous in the thigh, the safest course is to ligature the vein and excise the thrombosed portion.

When in an uncomplicated case of phlebitis the clot has undergone early and rapid disintegration, and the blood-current through the vein can be felt to be re-established, movement may be allowed at an earlier period, and may be cautiously commenced when the vein is entirely free from pain and tenderness.

When a large trunk, such as the femoral or iliac vein, becomes permanently blocked, many small superficial veins become developed, which not infrequently become inflamed in consequence of some local irritation. A little tenderness is felt on some part of the skin, and on careful

inspection a narrow branching line of slight redness is seen, under which is felt the soft cord of a minute blocked vein. If the limb is kept quiet for a few days, and the tender vein covered with a warm lotion of boric acid or lead, or painted over with oxide of zinc lotion, the little vein usually soon becomes again pervious, the tenderness and redness subside, and the trouble is at an end; but if the condition is neglected, the phlebitis may spread to larger veins, or other small branches may become involved, and a troublesome area of inflamed skin is left, causing much irritation and annoyance. When the patient first begins to walk, a soft elastic bandage should be gently applied from the foot to the groin, and carried in a figure-of-eight round the iliac region of the abdomen, the skin having been first dusted with powdered boracic acid. The upright posture should be maintained only for a very short time at first; a walk of about ten minutes will cause the limb to become painfully congested and necessitate a rest in the recumbent posture, with the limb well raised. Standing should be avoided as much as possible, but the exercise of the muscles should be gradually increased. If all goes well, and if the blocked vein is quite free from tenderness, massage may be commenced after about three months. This should consist of gentle rubbing in an upward direction, the actual course of the blocked vein being avoided, and the limb dusted freely with boracic acid. It should be done in the evening, and no exercise should be taken afterwards. The foot of the bed should be raised on blocks 6 or 8 inches in height. As more use of the limb becomes possible, it will be best to substitute for the bandage a well-fitting elastic stocking. If the femoral vein is blocked the stocking should be in two pieces, the lower reaching from the foot to just below the knee, the thigh-piece from below the knee to the fold of the

groin, with a peak ending in a strap for attachment to braces.

If the obstruction extends above Poupart's ligament to the iliac veins or the vena cava, a combined thigh-piece and abdominal belt should be worn; and this should be adopted from the beginning, so as to prevent as far as possible the enormous dilatation of the superficial collateral veins at the groin and iliac region which will otherwise take place (Figs. 6 and 7). Probably no care will prevent considerable enlargement of these veins, and a graduated pad should be introduced into the belt when the enlargement becomes apparent. But whichever part of the venous trunk is obstructed, it is a mistake to delay the application of the elastic support until the superficial veins begin to dilate; every effort should be made from the first to encourage the return of the blood by the deeper branches.

In most cases the thigh-piece can be discarded after four or five years, but in some instances the great development of the collateral veins of the iliac region necessitates continued support and protection. Great care should be taken to avoid all constriction of the limb. Elastic stockings are often made with a tight band at the upper limit, which acts like a garter. This is harmful and unnecessary, for if the stocking is made to measure and fits well, it will keep in place without any such addition. The patient should be instructed to take every opportunity of keeping the limb raised, especially towards the end of the day. Exercise should be taken in the early part of the day. Hard seats, especially chairs with a hard rim at the front of the seat, should be avoided, and a footstool used, so as slightly to raise the front of the thigh from the chair. It is astonishing how small an amount of pressure will obstruct the venous circulation, especially where it de-

pends largely upon the superficial veins: the weight of the arm resting upon the thigh, the weight of the leg if crossed over the opposite limb, is sufficient seriously to impede the venous return.

Towards the end of the day, especially if there has been much standing, the limb will become somewhat swollen, heavy and congested, and this is often accompanied by a good deal of aching, itching, and discomfort. The best remedy for this is a thoroughly hot bath, followed by gentle massage with the limb raised.

Among the remoter troubles resulting from blocked crural veins is the development of a plexus of small tortuous veins between the inner ankle and the heel, which leads to a condition of irritation and malnutrition of the skin in that situation, which may even go on to ulceration. When these veins begin to appear, a thin pad of lint or felt may with advantage be introduced under the stocking, behind the inner malleolus; and when signs of irritation of the skin are observed, a very useful application is an ointment of hazeline and lanoline gently rubbed over the part at night.

Groups of small, slightly raised, bright red spots on the skin, somewhat resembling psoriasis, are also met with, chiefly about the inner side of the knee and the outer side of the thigh. These spots appear usually after long standing, and most commonly in cold weather; they cause much itching and burning, for which the lanoline and hazeline ointment is a suitable application, and they rapidly disappear with rest. Sometimes after much standing or prolonged walking bright red patches appear on the legs, attended by a sense of heat, but not of itching. The redness disappears on slight pressure, and fades gradually after a day or two of rest. These conditions are much benefited by the hot bath.

There is seldom any subcutaneous œdema such as gives

rise to pitting on pressure, but there is usually some permanent enlargement of the affected limb. I do not think that this increase in the size of the limb is due, as was suggested by Sir James Paget,[1] to muscular growth, but I believe it depends upon an increase in the intermuscular and subcutaneous cellular tissue: for the outline of the muscles and their visible contraction is obscured, and the muscular power of the limb is diminished, not increased. The morning and evening measurements of the limb will be found to differ considerably, and this is certainly due chiefly to fulness of the vessels, though it may partly depend upon intermuscular œdema.

Moreover, in two of the cases related by Sir James Paget[2] the enlargement of the limbs disappeared under the use of friction and the hot douche, which would hardly have been the case if it had been due to muscular growth.

The measurements which I had the opportunity of taking in a case nine years after blocking of the femoral and external iliac veins were:

| | | Sound limb. |
|---|---|---|
| Morning circumference of thigh... | = 20½ inches | 20 inches. |
| Evening „ „ ... | = 21½ „ | |
| Morning „ calf ... | = 13½ „ | 13 „ |
| Evening „ „ ... | = 14 „ | |

Sir Prescott Hewett[3] speaking of the after-history of cases of phlebitis, says: 'When seen in after-years, the condition of the limb in these cases of blocked veins has varied very much. In a few the limb recovered its usual appearance, save perhaps a very slight increase in its size, in no way interfering with the freest action; for I have known one gentleman who, after an attack of this kind

[1] 'Clinical Lectures and Essays,' p. 307. *Medical Times and Gazette*, March, 1858, p. 261. Here Sir James Paget says that he has not dissected a case, and 'can only guess that there is a real overgrowth of muscles.'

[2] 'Clinical Lectures and Essays,' p. 305.

[3] *Transactions of Clinical Society of London*, vol. vi., p. xxxvii.

which confined him to his couch for months, returned to deer-stalking with as much zest and activity as before. In other cases the limb remained more or less swollen, and consequently with its action more or less interfered with, the circulation being carried on by anastomosing veins, largely increased in size and tortuous.'

The bones of the affected limb may appear to be enlarged, but examination by the Röntgen rays shows that this is due to increase in the subperiosteal connective tissue, not to osteal growth.

After some years, as the obstructed venous return is compensated by the development of the collateral veins, the enlargement of the limb may disappear or greatly lessen, its shape becoming more natural and the outline of the muscles more apparent. This improvement will be aided by hot baths, followed by gentle rubbing in the direction of the venous current, by moderate use of the limb, and by elastic support to the veins.

The heart, of course, has much extra work thrown upon it by venous obstruction, and when exercise is first resumed in a case of blocked vein dyspnœa is easily provoked; care must therefore be taken to put no needless strain upon the heart, as by rapid walking or ascending hills. It should be remembered that if the resistance in the vessels is such as the heart can overcome, hypertrophy of the heart takes place to meet the increased resistance; but that if the resistance is greater than the heart can overcome, dilatation of the heart results.

But gentle exercise is good, and sunlight and pure air are very beneficial; for the better the oxygenation of the blood, the more easily it circulates, and the less is the fatigue of muscular action. Moreover, the more thoroughly the blood is oxygenated the less prone is it to thrombosis. It has been proved that the inhalation of

oxygen gas,[1] or even the increased intake of oxygen by rapid respiration,[2] diminishes the coagulability of the blood.

The sufferer from blocked veins who wishes to reduce the inconvenience to a minimum must live temperately on light diet; be careful to avoid constipation or fæcal accumulation; take moderate exercise early in the day, and rest in the evening; avoid hurried, severe, or prolonged exertion; clothe warmly but lightly; keep out of hot and ill-ventilated rooms; breathe pure air, and seek the sun.

The necessary limits of the time allotted to these lectures has, of course, obliged me to omit much that might have been said upon this most interesting subject—the coagulation of the blood within the living vessels; but one thing which will probably have become apparent is the small amount of our certain knowledge concerning the blood—that wonderful living fluid which carries life and nourishment to every tissue, giving to the body its power of action and of feeling, and to the brain its power of thought and memory; which is so delicately constituted that by the invasion of a minute organism and the raising of its temperature two or three degrees it may be so profoundly altered that the whole body at once becomes disturbed, action impeded, and thought confused; and which, if it should undergo clotting within the vessels, is changed from a source of healthy action to a cause of mortal danger, or even of sudden death.

I say how little we really know of the vital chemistry and physiology of the blood. Here is a wide and fertile field for our advancing physiological chemists. I have only endeavoured in these lectures to make some contribution to our clinical knowledge, and to give some hints which I hope may be useful in the treatment of what is often a troublesome and sometimes a dangerous disease.

[1] Wright, *Brit. Med. Journ.*, July 14, 1894, p. 57.

[2] Hasebroek, *Zeitschrift f. Biologie*, 1882, p. 41.

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