Morgagni reports a case of the hepatic veins joining the vena cava inferior after it had pierced the diaphragm (quoted by W. Krause). Hyrtl has seen the hepatic veins empty by a common trunk into the right auricle

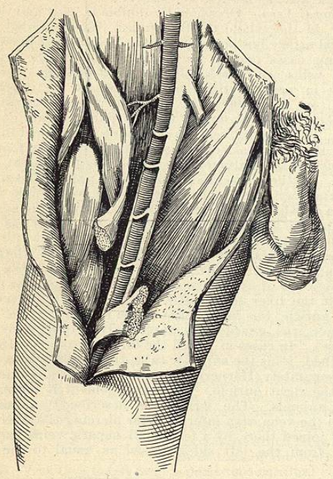

FIG. 5008.—Small Looped Veins passing Over the Femoral Artery, forming a Double Femoral Vein. (R. Quain.)

to the inner side and separately from the inferior cava; this, as mentioned above, is the normal course when the vena cava is absent or its place is taken by a persistent cardinal vein.

Umbilical Vein.—This vein has occasionally been found patent for a variable distance below the liver. It may communicate with the epigastric, and thus establish a collateral circulation; this is much more evident when a diseased condition of the liver obstructs the venous circulation.

J. A. Russel[23] reports two cases of persistent communication between the umbilical and portal veins in the

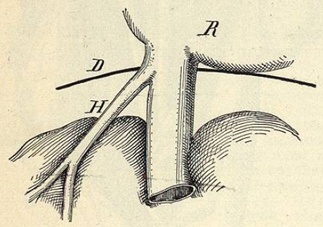

FIG. 5009.—Hepatic Veins from Right Lobe of the Liver Opening by a Common Trunk near the Entrance of the Inferior Vena Cava through the Diaphragm. *R*, Right auricle; *D*, line of diaphragm; *L*, liver; *V*, vena cava; *H*, abnormal hepatic veins. (Shepherd.)

human subject. F. Champneys[24] describes a communication between the external iliac and portal veins through the epigastric and umbilical veins.

This was due, probably, to fusion of Luschka's *par umbilicalis* (which, according to him, always exists normally as a communication between the portal and epigastric veins), and the channel was afterward increased in size by obstruction, due to enlarged liver. A communication of large size between the umbilical and epigastric veins is the normal arrangement in many of the lower animals, as the rorqual, seal, sheep, pig, etc., and in man is an early fœtal condition. Numerous examples have been recorded of communication between the veins of the abdominal parietes, as the phrenic (azygos, etc.) and the portal vein. The writer has already described, in connection with the renal vein, a case in which a large branch of communication existed between the left renal vein and the splenic.

Menière has described a case in which a large vein, as thick as a finger, went from the portal vein to the right iliac. And Brigidi[25] has reported a case of free communication between the umbilical vein, which was patent, and the right iliac by means of a branch of large size in a case of cirrhosis of the liver.

W. Krause[26] mentions a number of cases of communication between the portal vein and the iliac veins by means of a patent umbilical, connected directly by a branch or through an epigastric vein.* These communications are all due to persistent fœtal conditions, and are much more apparent when there is any obstruction to the portal circulation. *Francis J. Shepherd.*

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VEINS, PATHOLOGY OF.—In general, the same pathological processes are found in the veins as in the arteries, though varying in degree and importance in the two sets of vessels, some conditions being more pronounced in the arteries, others in the veins. Thus, for example, inflammatory changes are of much more frequent occurrence in the veins; sclerosis and calcification in the arteries. Thrombosis of veins and thrombophlebitis are among the most common and important of pathological conditions; while, on the other hand, from the nature of things, arterial embolism holds a like important place in pathology. In addition to the pathological processes common to both sets of vessels there is a number of morbid conditions peculiar to the veins, among the most important of which may be mentioned varices and phlebectasias.

ANOMALIES.—*Congenital anomalies* in the number, branching, course, and distribution of veins are very common. They are rarely of clinical importance, though at times causing temporary embarrassment during surgical operations. Congenital dilatations or varices are also of very frequent occurrence. They may vary in size from a slight local increase in the prominence of the veins (*venous nævus*) to extensive dilatations of the superficial veins involving an entire extremity, or the greater portion of the trunk or body. Heredity is an important factor. As a rule the condition does not become especially noticeable until about the age of puberty when there is usually a rapid increase in the degree of the abnormality. The superficial veins alone may be affected, or anomalous communications may exist between these and the deep venous trunks, or the entire venous system of an extremity may be over-developed without a corresponding development of the artery. (See *Veins, Anomalies of.*)

RETROGRADE CHANGES.—*Atrophy.*—A thinning of the wall of a vein may result from long-continued overdistention, or from a lessened function, as in the case of an amputation stump. As the result of such thinning of the vessel wall there may follow a dilatation of the lumen, formation of varices, insufficiency or rupture of the valves of the veins, or even rupture of the vessel itself. The atrophy may be confined to one venous system or may be universal. The latter occurs particularly in the case of extreme general venous congestion, as in chronic valvular lesions of the heart. A secondary atrophy of the walls of veins appears also as a frequent accompaniment or sequela of a variety of primary conditions affecting the veins, such as phlebitis, etc. Hypertrophy of the vein wall is usually followed by a secondary thinning, which may be associated with retrograde changes, such as fatty degeneration, hyaline change, calcification, etc. A secondary thinning of the vein wall is also of common occurrence in varices.

Fatty degeneration occurs in the intima of veins under the same conditions as in that of arteries (anæmia, intoxications, infections, etc.). In the intima of the affected vein there are seen whitish or yellowish spots or patches which are usually slightly elevated above the general surface of the intima. The degeneration is usually confined to the endothelium, and the affected cells are more easily scraped from their basement membrane than are the normal cells. In long-continued and severe intoxications, as in sepsis, chronic nephritis, icterus, pernicious anæmia, etc., extensive fatty degeneration may be found in the intima of the veins. Similar marked changes may also be found in very severe cases of certain of the acute infections, such as diphtheria and pneumonia, also in severe anæmia due to hemorrhage, and in poisoning with phosphorus, arsenic, etc.

Hyaline degeneration of the connective tissue of the walls of veins, while not so common as in the walls of arteries, is nevertheless of relatively frequent occurrence. The radicles of the portal vein are most often affected, but the process is common enough in the femoral veins and in the veins of the lower extremities. The spermatic and ovarian veins, and the veins of the broad ligament not infrequently show this change. It may be primary or follow secondarily upon a chronic phlebitis. The change may be diffused or localized. In the majority of cases it is the sequela of a fibroid thickening of the intima, the new connective tissue formed beneath the endothelium soon becoming hyaline in character (phlebosclerosis). Calcification and fatty degeneration may follow, but are much less common than in the case of the arteries. The portal radicles, hepatic veins, and the veins of the lower extremities are most frequently affected. When occurring primarily and not as a sequela of phlebitis, hyaline degeneration of the wall of veins is usually dependent upon a chronic intoxication, as in the case of syphilis. The media and adventitia are rarely involved. After thrombosis of a vein and subsequent organization of the thrombus the newly formed connective tissue replacing the thrombus may become hyaline in character. In extreme chronic passive congestion of the lungs (mitral stenosis) the pulmonary veins as well as the arteries may become greatly thickened and present a hyaline change of greater or less degree.

Calcification.—This is on the whole relatively rare in veins, and is usually localized to small areas. Only rarely are veins calcified throughout any considerable portion of their wall. The deposit of lime salts most frequently follows hyaline or fatty changes, or is a sequela of thrombophlebitis. In cases of chronic phlebitis true bone may sometimes be formed in the walls of veins. This event occurs particularly in the femoral and its branches and in the radicles of the portal vein. The writer has observed a number of cases of calcification of the femoral and saphenous veins following thrombophlebitis of these vessels. In two cases osteoid and true osseous tissues were found in the vein walls and in the adjacent tissues. Calcification of the portal vein is of not infrequent occurrence. Virchow, Spiegelberg, and others have described such cases. The lime salts are deposited in the form of thin laminæ, usually in the media of the vein, but at times also in the intima and adventitia. Evidences of a chronic pylephlebitis are usually present in the form of sclerotic changes, irregular thickenings of the wall, bridges of connective tissue crossing the lumen of the vein, etc. The mesenteric and splenic veins may show similar changes. In two cases of Banti's disease studied by the writer there was a marked stenosis of the portal vein with extensive sclerosis and calcification, involving chiefly the middle coat of the vessel. The irregular character of the vein wall, the roughened intima, and the strands of connective tissue crossing the lumen in one of the cases made it very probable that the stenosis and the accompanying sclerosis and calcification were the sequelæ of an old thrombopylephlebitis. In both cases the splenic and mesenteric veins and their radicles were greatly dilated and showed marked sclerotic changes. Deposits of lime salts were also present in the sclerotic walls of many of the smaller branches of the mesenteric veins. Several similar cases have been reported under various heads in the older literature. In connection with these cases the question arises if the symptom complex now known as splenic anæmia may not be secondary to portal obstruction due to various causes. Sclerosis and deposits of lime salts are not rare in the portal vein and its branches in the late stages of cases of hepatic cirrhosis. Calcification of the vein walls in other parts of the body is of rare occurrence, except in the case of local deposits at the site of calcified thrombi.

Amyloid change is much rarer in the veins than in the arteries, and is usually found in but slight degree. It is extensive only in the case of extreme general amyloidosis.

PROGRESSIVE CHANGES.—*Hypertrophy* of all three coats of the vein wall with dilatation of the lumen occurs in athletes and in individuals accustomed to heavy labor. The degree of hypertrophy bears no constant relation to excessive muscular activity, and such an enlargement of the veins may be entirely absent in individuals in whom such conditions are present. It must, therefore, be assumed that some inherent peculiarity in the structure of the vein walls lies at the foundation of such hypertrophy. This view is further supported by the fact that individuals not given to marked muscular activity may also show such a venous hypertrophy. In the latter case the hypertrophy is usually congenital and often inherited, particularly from the paternal side. It is probable that such congenital hypertrophy of the veins is due to a primary weakening of the walls or to a lowered tension in the tissues surrounding the vessels. The loss of the aid given to the venous circulation by muscular activity may also play a part in the production of the condition.

A hypertrophic condition of the veins occurs also in the vessels taking part in the formation of a collateral circulation. In general it may be said that stagnation of the blood-flow in veins leads to a thickening of their walls. A general hypertrophy of the venous system may therefore be the result of a general venous stasis, as in the case of heart lesions without proper compensation. In mitral stenosis there is almost always a marked thickening of the walls of the pulmonary veins, and, in case the right ventricle is not compensating fully for the lesion, a similar thickening of the systemic veins also re-

* Sappey is of opinion that all recorded cases of free communication between the umbilical and epigastric veins rest on errors of observation; he holds that the dilated vein is not the umbilical vein, but one of the accessory portal veins. Professor Giacomini is also of the same opinion, saying that the umbilical vein, in its course from the umbilicus to the portal vein, neither gives nor receives any branch normally. Professors Bordoni and Ronitti, however, have investigated the subject afresh, and find that the umbilical vein anastomoses with the epigastric, not only in infants a few days old, but in those five and six months of age. In bodies of infants several days or months old they never failed to demonstrate the anastomoses between the branches of the epigastric and umbilical veins by means of Richardson's injecting medium (see London Medical Recorder, July, 1888, p. 274).

sults. A local venous stagnation may give rise to a local venous hypertrophy. Such enlargement of the veins and thickening of their walls are frequently seen in the neighborhood of tumors. A secondary atrophy follows sooner or later in all cases.

The increase of thickness may involve all three coats, but often the muscularis is alone hypertrophic. At other times the intima may show the most thickening, and in other cases still the adventitia. In the case of collateral anastomoses, particularly in arterio-venous anastomoses and behind obstructions, the vein wall may show a very marked degree of thickening. In the majority of cases such hypertrophies are in reality connective-tissue hyperplasias due to chronic hyperplastic phlebitis. A true muscular hypertrophy and hyperplasia do occur, however. The new connective tissue formed in the vein wall is usually of the character of scar tissue. Secondary atrophy, contraction and thinning, in association with sclerotic changes, fatty degeneration, calcification, etc., follow the hypertrophy. The hyperplasia of the muscularis in the case of collateral anastomoses is to be regarded as purely compensatory in nature, though often associated with inflammatory changes. In all organs in which there has been for some time a condition of marked chronic congestion the number of large veins appears to be greatly increased. It is due to the fact that the walls of the smaller veins become thickened in proportion to the dilatation of the vessel lumen. In these cases the muscle does not become increased in amount, the thickening of the wall being due to a new formation of connective tissue which later becomes more or less hyaline in character.

Inflammation. Phlebitis.—Inflammation is a much more common occurrence in the veins than in the arteries. Though analogous in structure to the latter, the walls of the veins are thinner and weaker, and their coats less sharply differentiated. For this reason it is hardly possible to separate inflammatory processes of the vein walls into endo-, meso-, and periphlebitis. In the great majority of cases all three coats are coincidently affected, the process extending quickly from the intima to the adventitia, or in the opposite direction, so that the entire vein wall becomes equally involved. A further and more important difference between the arteries and veins is the part played by the latter in inflammations of the tissues. In such conditions the arteries present no recognizable anatomical changes; the veins, on the other hand, present the phenomena of a marginal disposition of the leucocytes and diapedesis of the same. In purulent inflammations the smallest veins may become so involved that the elements of their walls suppurate or show signs of proliferation. Such changes should be included under phlebitis in a broad sense, but the term is usually restricted in its application to inflammatory processes involving the large veins.

According to Köster, phlebitis is to be regarded as a *lymphangitis* of the vein wall, the inflammatory process extending along the lymph spaces and lymph vessels with which the wall is richly supplied. The extension upward along the vein (*ascending phlebitis*) is due to an ascending lymphangitis. The vasa vasorum likewise play an important part in the extension of the process. The connection between peripheral lymphangitis and phlebitis is very close. In the case of infected wounds of the extremities the signs of an ascending lymphangitis precede those of phlebitis, the infection of the vein wall following that of the lymphatics.

In all cases of phlebitis in which the intima of the vein is damaged thrombosis results. The combination of thrombosis with phlebitis is known as *phlebothrombosis*, and is to be regarded as one of the most important features of the process. In the case of hæmatogenous infection (intravascular) the formation of a thrombus may constitute the first step of the process, the inflammation of the vein wall following upon this. In the case of periphlebitis (extravascular infection) the thrombosis follows the extension of the process to the intima. By some writers the term *thrombophlebitis* is applied to the first condition, *phlebothrombosis* to the second. This distinction is not commonly observed, however, and the term thrombophlebitis is used to designate both processes. Thrombosis may be associated with either acute or chronic phlebitis.

Etiology.—The infection of the vein may be either intra- or extravascular. In the first case the infection is either of metastatic or of cryptogenic origin. More frequently the vein is involved by an extension (lymphangitis) from neighboring suppurative processes. Infected wounds (trauma, operation, childbirth, etc.) form the most common causes of phlebitis. The primary infection may be of slight degree, or the infection may take place through uninjured tissues (hair follicle). Phlebitis due to infection following abortion or childbirth is of frequent occurrence. In the majority of the infectious diseases phlebitis occurs often, being due either to the direct action of the specific infectious agent, the intoxication, or to a secondary infection. In smallpox, typhoid fever, influenza, erysipelas, diphtheria, dysentery, scarlatina, pneumonia, and gonorrhœa phlebitis not infrequently occurs. In the milder infections, such as measles, it is a more rare complication. Chronic intoxications, syphilis, gout, alcoholism, etc., are regarded by some writers as causal agents in chronic phlebitis. Obliterating endophlebitis in particular has been ascribed to syphilis. By other writers the existence of a specific rheumatic or gouty (alcoholic) phlebitis is denied. Of the micro-organisms causing phlebitis, the streptococcus, staphylococcus, pneumococcus, gonococcus, typhoid bacillus, and the colon bacillus play the chief rôles.

Varieties.—As mentioned above, it is rarely possible in the acute forms of phlebitis to differentiate endo-, meso-, and periphlebitis. In chronic phlebitis such a classification is often possible, the chronic forms being more often confined to the inner and middle coats. According to the chief characteristics of the inflammatory process, there may be distinguished the following varieties of phlebitis:

Phlebitis.	Acuta (hæmatogenous or extravascular).	1. Exudativa. (*a*) Purulenta. (*b*) Suppurativa.	Phlebothrombosis or thrombophlebitis.
		2. Dissecans.	
		3. Productiva, s. adhæsiva, s. organisatoria.	
		4. Gangrænosa.	
	Chronica (hæmatogenous or extravascular).	1. Hyperplastica.	
		2. Atrophica.	
		3. Obliterans.	

Various other terms have been applied by different authors to designate especial features of given cases: *ascending phlebitis*, *diffuse phlebitis*, *universal phlebitis*, *panphlebitis*, etc. *Phlebitis circumscripta* is also applied to a localized phlebitis, more particularly to areas of localized inflammation in the walls of the cerebral sinuses.

Acute Phlebitis.—The vein wall may be infected from the blood (*hæmatogenous*) or from the tissues (*extravascular*). The latter occurrence is probably the more common.

(*a*) *Extravascular Origin.*—In this case the adventitia is first affected, and the early stages of the process appear as a purulent periphlebitis. The lymphatics and the small veins of the vessel wall become filled with leucocytes, and these may collect in such numbers as to give the process the character of a purulent infiltration of the vein wall (*phlebitis exudativa* or *purulenta*). Œdema and liquefaction of the elements of the wall may also take place as the process increases in severity (*phlebitis suppurativa*). Inasmuch as the connective-tissue sheath of the vein wall is very resistant the exudate collects beneath it and extends along the vessel for some distance, giving rise to other abscesses. In this way the vein may become surrounded by a mantle of pus, which may be so great as to separate the vessel from its neighboring tissues and lead to the complete necrosis of the elements of its wall. The media and intima are usually quickly involved by direct extension and also through the lymphatics and vasa vasorum. Ultimately the entire vessel wall may present a dense purulent infiltration; in some cases only traces of the original tissues of the wall can be made out. The thickness of the wall may become greatly increased,

ten to twelve times as great as normal; and the lumen of the vein may be obstructed or wholly obliterated as the result of the swelling alone. In large veins or in the case of a lesser degree of infiltration the intima may show circumscribed areas of bulging corresponding to the points of greatest infiltration. In addition to the swelling caused by the œdema and cellular exudate, hemorrhages of varying size may occur.

The elastic tissue of the media plays an important part in the extension of the infiltration toward the intima. The advancing exudate pushes the elastic fibres toward the intima, and finally breaks through them at the points of least resistance. The elastica interna remains preserved for the longest time, but finally gives way, and the exudate presses through the point of rupture. Finally, the elastic fibres become involved in the complete dissolution of the vein wall.

The damage to the intima results constantly in thrombosis. The thrombus thus formed may be obturating or parietal; in both cases it serves to prevent further entrance of the exudate into the lumen of the vein. In the formation of the thrombus the elements of the blood circulating through the vein may take part, or, as affirmed by Köster and others, the *intravasation* of red and white cells and fibrinogen-containing exudate from the affected wall into the vessel lumen may aid in the formation of the thrombus, or be alone concerned in its production. The inflammation of the vessel wall is, therefore, the primary process, the thrombosis the secondary, the entire process being regarded as analogous to the formation of exudates upon serous membranes, the exudate containing red blood cells, blood plates, leucocytes, fibrinogen, etc., these being poured out from the vasa vasorum over the intima of the affected portion of the vessel. With the formation of the thrombus upon the intima the process may be appropriately designated phlebothrombosis (*phlebothrombosis purulenta*). By Hunter it was termed *phlebitis pseudomembranacea*. By the majority of writers no distinction is made between thrombophlebitis and phlebothrombosis, but by others the distinction as given above is favored. Inasmuch as the thrombosis is in one case the primary process and in the other a secondary, it would seem well to retain the distinction.

The œdema and infiltration of the coats of the affected vein may lead to their separation from each other (*phlebitis dissecans*). In the case of an involvement of a vein in a gangrenous process the phlebitis may also assume a

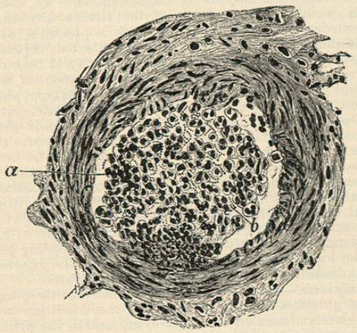

Fig. 5010.—Secondary Vasculitis of a Small Vein of the Leg in Phlegmon due to Streptococcus pyogenes. *a*, Leucocytes; *b*, Streptococci. (After Weichselbaum.)

gangrenous character (*phlebitis gangrænosa*). With the advent of thrombosis and the partial or complete obturation of the vessel on the one hand and the cessation of the inflammation on the other, organization of the thrombus may take place (*phlebitis productiva, s. adhæsiva, s. organisatoria*) and healing may result. The sequelæ of such healing may be thickening of the vessel wall, obliteration of the lumen, formation of connective-tissue bridges, calcification, etc. During the healing process or subsequently thereto a regeneration of elastic fibres may take place, and newly formed fibres may also be found in the organized thrombus.

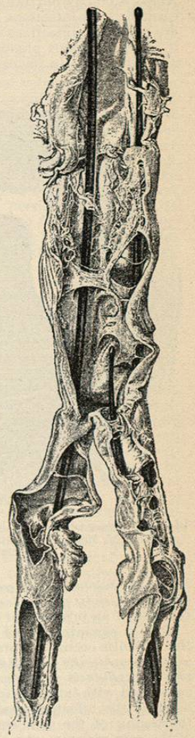

Fig. 5011.—Changes Produced in the Inferior Vena Cava and Iliac Veins by the Organization and Contraction of a Thrombus Resulting from the Involvement of the Vessels by a Retroperitoneal Abscess. The irregularities of the intima and the connective-tissue bridges crossing the lumen cause the vessel to resemble the superior longitudinal sinus. (After Schrötter.)

(*b*) *Hæmatogenous Origin.*—The phlebitis caused by intravascular excitants of inflammation runs a different course. There is usually a primary thrombosis followed by a secondary inflammation. As a result of changes produced in the intima of the vein by toxins, or by the direct action of bacteria, or by the direct action of heat, freezing, trauma, etc., there results a thrombosis. If micro-organisms capable of exciting suppuration are not present in the thrombus (so-called benign thrombus), the only effect upon the vein wall is the slight reactive inflammation caused by the presence of a foreign body. Organization of the thrombus takes place, and it is replaced by newly formed connective tissue and blood-vessels (*phlebitis productiva, s. adhæsiva, s. organisatoria*). The sequelæ of calcification, obliteration of the lumen by connective tissue, thickening of the vessel wall, formation of connective-tissue bridges, etc., likewise occur in this connection. Irregular dilatations and stenoses of the affected vein result; the newly formed connective tissue in its lumen may become channelled by new vessels, and the circulation partly or wholly re-established. As the result of such changes the vein may come to have a structure suggesting that of the longitudinal sinus of the dura mater. (Fig. 5011.)

Much more unfavorable is the course if the original thrombus contains pyogenic micro-organisms capable of multiplication. These exert their characteristic effects upon the intima and upon the elements of the thrombus; necrosis, liquefaction, and infiltration of the vein wall follow, the process extending outward toward the adventitia (*thrombophlebitis purulenta s. suppurativa*). The infection of the lymphatics and blood-vessels of the vein wall leads to a further extension of the process, and the surrounding tissues become secondarily involved. The thrombus and the entire vein wall at the seat of infection may be completely destroyed by the suppurative process. Healing and organization may take place; but in the majority of cases the softening of the thrombus leads to embolism of infected material, and the suppurative process repeats itself at the site of lodgment of the emboli.

The presence of pyogenic organisms in the circulation leads to a general pyæmia which is frequently fatal. In the pre-aseptic days this form of phlebitis was much more common than the secondary phlebothrombosis, but it is now relatively more rare. Childbirth, abortion, infected wounds from trauma or operation, malignant endocarditis, acute infections, and secondary infections following the acute infectious diseases are most often followed by hæmatogenous phlebitis. Cryptogenic infections are also common. In the case of infection by certain organ-

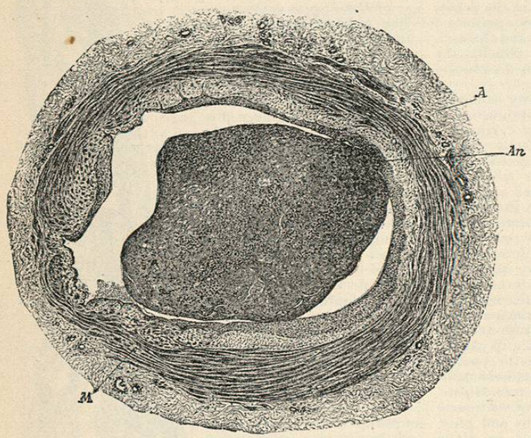

FIG. 5012.—Chronic Phlebitis, with Beginning Organization of the Thrombus. *A*, Adventitia; *M*, Media; *An*, thickened intima at point of attachment of organizing thrombus. (After Schrötter.)

isms the phlebitis may assume a gangrenous character (*thrombophlebitis gangrænosa*). This is more rare than in the case of an infection of the vein from the tissues.

Should organization of the thrombus and healing of the phlebitis occur the process assumes the character of a *thrombophlebitis organisatoria*. An *acute productive phlebitis* (*phlebitis prolifera*) without thrombosis may be associated with inflammatory processes in the neighborhood of the affected vessel, as shown by a fibroblastic thickening of the vein wall, dilatation, and thickening of the vasa vasorum, etc. It is also possible that similar changes may be of hæmatogenous origin.

Chronic Phlebitis.—The processes of organization, repair, etc., following an acute phlebitis are often regarded as of the nature of a chronic phlebitis. They are more properly sequelæ. A true chronic phlebitis, however, occurs as the result of chronic inflammations in the neighborhood of veins, and possibly also as a result of chronic intoxications. In hepatic cirrhosis the branches of the hepatic veins usually show chronic inflammatory changes. The veins in the neighborhood of chronic ulcers or abscesses, chronic tuberculous or actinomycotic foci usually show marked changes of the nature of a chronic inflammation. These are characterized by a connective-tissue thickening of the vein wall (*phlebitis hyperplastica*), enlargement of the vasa vasorum, and thickening of their walls, and either a dilatation or stenosis of the vessel lumen. The thickening of the vessel wall may depend to some extent upon a chronic infiltration. Secondary thinning of the wall frequently follows (*phlebitis atrophica*). By many writers varices are regarded as a sequela of chronic phlebitis. Sclerotic changes or calcification may also occur. Thrombosis may or may not be associated with the process. The organization of the thrombus or the proliferation of the intima may lead to an obliteration of the vein at the affected point (*phlebitis obliterans*). (Fig. 5012.)

An *endophlebitis obliterans* (*endophlebitis prolifera*) occurs coincidently with or under the same conditions as endarteritis obliterans. As a result of the proliferation of the intima the lumen is partially or completely filled with a newly formed connective tissue, often resembling myxomatous tissue, and containing numerous new blood-vessels, giving it a certain amount of vascularization. In the majority of cases the condition may be ascribed to syphilis, but similar changes may follow the ligation of veins, thrombosis, and possibly various chronic intoxications. The veins of the lower extremities, the portal radicles, and the branches of the hepatic veins appear to be the most frequently affected. In three cases described by Chiari the proliferation of the intima of the hepatic veins led to a marked stenosis of their lumen, in some vessels even to a complete obliteration, as a result of which secondary changes occurred in the liver, accompanied by overdistention of the portal vein and ascites. Death took place from thrombosis of the obstructed veins. Chiari regarded syphilis as the most probable causal factor in these cases.

Symptoms.—Phlebitis may occur without any individual signs or symptoms; or these may be obscured by the general symptoms of the primary disease. Particularly in the case of the deeply lying veins may symptoms be absent, and the condition may become evident only through the occurrence of thrombosis or of other sequelæ. In acute phlebitis there is usually an irregular fever, which at times may reach a high degree. The course of the vein, if superficial, may be revealed by the presence of a dark blue, or reddish, thick cord, which is very tender on pressure. In the event of thrombosis the cord becomes more firm and hard. If the surrounding tissues are markedly œdematous and infiltrated it may not be possible to feel the vein. Pressure over deep-seated veins is also painful. The pain is also spontaneous, not only along the course of the vein, but also in its neighborhood. An œdematous swelling of the vein or of the tissues surrounding a vein does not necessarily indicate a phlebitis, as the condition may result from a non-infected thrombus. The distention of the superficial veins may in both cases be regarded as evidence of the establishment of a collateral circulation. The symptoms of chronic phlebitis are in general similar to those of the acute, but are less severe. Acute exacerbations are common in the infected cases, the symptoms being those of an acute phlebitis. In the case of superficial vessels the thickening remains permanent, while the redness and tenderness disappear. The development of the collateral circulation is usually more pronounced, and the distention of the superficial vessels lasting in the case of chronic phlebitis.

It must be noted, however, that the symptoms of phlebitis may vary greatly in different cases, according to the cause, location, sequelæ, and the nature of the general or local disease with which it is usually associated.

Diagnosis.—From the above it is clear that the diagnosis of phlebitis is often not possible, the condition being completely concealed under the symptoms of the primary or accompanying disease, the two forming a symptom complex usually designated as *cryptogenic septicopyæmia*. The diagnosis is, however, easy in many cases, particularly when superficial veins are involved. The differentiation between phlebitis with secondary thrombosis (phlebothrombosis) and thrombosis with secondary phlebitis (thrombophlebitis) is in the majority of cases impossible. Neither the nature of the primary affection, the character of the fever or pain, nor the late appearance

of the symptoms in the course of another disease can be used as differential points. Both thrombosis and phlebitis may occur late in an infectious disease, even during convalescence. The persistence in the body of virulent bacteria may lead to phlebitis and thrombosis long after the original attack of the disease.

Occurrence.—The veins of the lower extremities are most frequently affected. The reasons for this are largely anatomical, these veins being more likely to suffer from stasis and consequent dilatations. Trauma with infection is also of frequent occurrence in this region. Varicose ulcers of the legs very often lead to phlebitis of the *saphenous veins*. The *venæ spermaticæ* and *uterinæ* may become infected after abortion or childbirth. Retention of the placenta leads frequently to an infection of the *uterine veins*. The thrombophlebitis beginning in these vessels may extend to the *vena cava* or the *femoral vein* (phlegmasia alba dolens). The *femoral vein* itself is very often affected primarily in the acute infections and in marasmic conditions. Phlebitis of the *hæmorrhoidal* and *vesical veins* follows cystitis, prostatitis, gonorrhœal infections, rectal and perirectal abscesses. Infection through hemorrhoids themselves is also common. In all of these cases the colon bacillus doubtless plays an important rôle. Phlebitis of the *mesenteric veins* and *pylephlebitis* occur in connection withdy sentery, perityphlitis, and other inflammatory conditions of the intestines. In infections of the umbilical cord in the new-born the *umbilical vein* usually presents the picture of a thrombophlebitis purulenta which may extend into the vessels of the liver and into the portal veins. The *veins of the upper extremities* are less frequently the seat of a phlebitis, but show the condition often enough. Infected wounds of the hands and boils are the most common sources of infection. Cryptogenic infection through an uninjured hair follicle may take place. Venesection was formerly the most common cause of phlebitis of the upper extremities. The *meningeal veins*, *sinuses*, and the *veins of the diploë* are very frequently the seat of phlebitis. Infected wounds of the head, mastoid or middle-ear disease, caries of the petrous bone, etc., give rise to thrombophlebitis of these vessels and further to meningitis and encephalitis. The *internal jugular* may ultimately be affected. The *sinus sigmoideus* plays an important rôle in the extension of infections from the ear. Phlebitis of the meningeal veins and sinus thrombosis may also follow infections through the orbital or nasal cavities. In rare cases carious teeth may be the primary lesion from which sinus thrombosis ultimately results. Phlebitis of the *pulmonary veins* may result from septic infarcts, abscesses, gangrene, and other inflammatory conditions of the lungs. In osteomyelitis the *veins of the bone marrow* present the picture of thrombophlebitis.

Sequelæ.—The most favorable sequela of thrombophlebitis or phlebothrombosis is the organization of the thrombus and the partial or complete obliteration of the affected vessel with the development of an adequate collateral circulation. Embolism of infective material from the primary focus in purulent phlebitis is the most unfavorable sequela. Such an embolism may be direct, retrograde, or paradoxical. In general, it may be assumed that from every phlebitic focus portions of material are set free into the venous circulation. If these contain pyogenic bacteria the emboli given off may cause an endocarditis of the right side of the heart, septic infarcts and abscesses of the lungs; or they may pass through the pulmonary capillaries and give rise to a mitral or an aortic endocarditis, or to pyæmic abscesses throughout the arterial system. Phlebitis in the regions tributary to the superior vena cava may give rise to liver infarcts and abscesses through retrograde embolism. Phlebitis of the pulmonary veins in particular leads to the production of embolic infarcts and abscesses throughout the general circulation, especially of the brain, spleen, and kidneys. Pylephlebitis may lead to infarcts and abscesses of the liver and lungs. In general the sequelæ of thrombophlebitis depend upon the location of the vein, the character and extent of the thrombosis, the presence of bacteria and the variety of the same, the possibilities of embolism, and the vital importance of the affected organs.

Prognosis.—This depends upon the size and location of the affected vein, the bland or infectious nature of the process, the occurrence of embolism, and the importance of the affected organ. In the less severe cases of phlebitis of the lower extremities the prognosis is relatively favorable, even in the case of pulmonary embolism, since the emboli are likely to be of small size. The more severe cases with marked œdema of the extremity (*phlegmasia alba dolens*), in which the principal veins are affected, are very dangerous because of the possibility of emboli of large size or the growth of the thrombus into the vena cava. Sudden death from pulmonary embolism may occur. The etiological factors in phebitis of the larger veins are in themselves of grave significance. The prognosis in the septic cases is always grave because of the possibility of embolism and the occurrence of general pyæmia and septicæmia. The persistence of fever is the most important indication to be followed. As long as there is a daily rise of temperature the danger of fresh thrombosis and embolism or of an extension of the phlebitis is present.

Treatment.—The old methods of treatment with mercurial ointments, iodine, etc., have been entirely abandoned. The local massage employed in their administration made their application a source of danger.

Prophylaxis is of prime importance. Asepsis or antisepsis at all times is the chief means by which the complication of phlebitis and its attendant thrombosis may be avoided. A small boil or even the slightest infected scratch may lead to a dangerous phlebitis. The pernicious habit of "paring corns" with domestic instruments is also a source of infection, which may sometimes run a dangerous and fatal course. The puerperium is obviously a time when modern aseptic precautions should be carefully observed. Abortion with retention of placental fragments is likewise of the greatest danger. Infection through carious teeth should be avoided by prompt attention to the condition. Mastoid and middle-ear disease should receive proper operative treatment. As a result of modern prophylactic measures the dictum of Cruveilhier that "la phlébite domine toute la pathologie" no longer applies. With the almost universal adoption of aseptic and antiseptic measures, pyæmia and puerperal sepsis have become much less frequent, and thrombophlebitis in consequence is a much more rare complication.

In the case of an established thrombophlebitis the most important indication is the avoidance of embolism by means of absolute rest until no doubt exists as to the subsidence of the process and the complete organization of the thrombus, and the establishment of the collateral circulation. This is particularly the case in thrombophlebitis of the extremities, especially the lower ones. Elevation and fixation of the affected limb may be of service in promoting the return flow from the obstructed area. In the event of severe pain the application of cold, etc., may be useful. The dangers of embolism and the results of moving the limb should be thoroughly impressed upon the patient.

Many cases of phlebitis are now treated surgically. The opening and drainage of periphlebitic abscesses should be carried out as soon as possible. Ligation of the efferent veins, exposure of the affected vein with direct antiseptic treatment of the same, or resection of the diseased portion, etc., are recent operations performed with great success. In the case of mastoid and middle-ear disease with extension to the meninges and sinus thrombosis the jugular may be ligated. As the result of these operations the duration of the process is shortened, the dangers of embolism are removed, and recurrences rendered impossible. The more severe the case the greater the indications for such operative interference.

The local and general conditions should receive appropriate treatment also. Septic and pyæmic conditions should be treated according to the conditions.