Wounds of Veins.—*Ruptures, lacerations, bruises,* and *erosions* of veins are of common occurrence. They take place much more frequently in the case of small veins than in the case of the arteries. Aside from direct trauma, an increase of intravenous pressure plays the most important part in the rupture of veins. Hemorrhage from the conjunctival veins may result from spasmodic coughing and vomiting. The same cause may lead to rupture of small veins in the mucosa of the larynx and trachea. The veins of a collateral circulation frequently burst, particularly in the case of œsophageal varices in hepatic cirrhosis. The pressure of a tumor or aneurism upon the great cervical veins may lead to a rupture of the meningeal veins. Ruptures of the veins of the lower extremities are of frequent occurrence. Increase of pressure, fatty degeneration of the vein wall, etc., are causal factors. Rupture of varices and hemorrhoids occurs very frequently. Intense cramps of the muscles of the calf may cause the rupture of small veins. In a case reported by Frerichs fatty degeneration of the wall of the portal vein led to the rupture of this vessel. Rupture of the superior and inferior venæ cavæ may be caused by falls, crushing of the body, etc. A fall upon the head may cause a rupture of a cerebral sinus even without external signs of injury. Bullet wounds of all kinds may occur in the veins in any part of the body. The wound may be caused by the direct action of the projectile or by pieces of bone. Cases have been reported in which the bullet remained embedded in the vein wall.

The sequelæ of such injuries are hemorrhage into the tissues or body cavities (hæmothorax, hæmatopericardium, etc.), hæmatoma formation, thrombosis at the point of injury, healing, and the formation of a collateral circulation. If the artery is injured at the same time with the vein an *anastomosis arterio-venosa* may be formed. Embolism of air or of portions of the thrombus may also follow. The aspiration of air into the veins is particularly likely to happen in injuries of the veins of the neck, but is seen more rarely in the case of injuries of the veins of the extremities, uterus, head, and possibly through the veins of the stomach in the case of erosions, ulcerations, etc. Repeated small hemorrhages from hemorrhoids or eroded varices may give rise to the severest grades of anæmia. In the case of the loss of large quantities of blood on a single occasion, death may follow immediately or later, as the result of fatty degeneration of the heart and other organs. In the case of injury of the large veins death usually follows at once from the large amount of blood lost, but even in these cases spontaneous recovery may take place. Surgical interference reduces greatly the mortality in these cases. Ligation of the jugular, innominate, axillary, and femoral veins, as well as of the inferior vena cava, has been successfully performed, and a collateral circulation established. Successful healing has also followed the suturing and resection of the femoral and the vena cava.

The treatment of rupture of veins is chiefly surgical. Compression, clamping, ligaturing, and suturing are resorted to according to the conditions. Even in the case of hemorrhage into the body cavities (pericardium, etc.) the bleeding veins may be found and successfully ligatured. In some cases ligature of the corresponding artery may be necessary to subdue the hemorrhage. The occurrence of after-hemorrhage should always be anticipated. General principles of absolute rest, proper position, etc., should also be applied according to the indications.

Perforation of the veins occurs in the case of aneurism, abscess, tuberculosis, actinomycosis, and new growths. Abscesses of the liver may rupture into the inferior vena cava. Erosions are of importance particularly in the case of the stomach veins; in cirrhosis of the liver, splenic anæmia, stenosis of the portal vein, etc., fatal hemorrhage may follow the erosion of the greatly dilated branches of the coronary or œsophageal veins.

Thrombosis.—Thrombosis of veins is of very common occurrence. In general the causes are: Changes in the vessel wall (phlebitis, intoxications, etc.); changes in the lumen (phlebectasias, varices, obstruction, etc.); changes in the composition of the blood (intoxications, anæmia, cachexia, hydræmia, presence of agents causing coagulation, etc.); changes in the blood current (stagnation, retrograde currents, etc.).

According to their location venous thrombi may be divided into *parietal* and *valvular*. A thrombus completely obstructing the vein is called an *obturating* thrombus. Parietal and valvular thrombi are found particularly in the veins of the extremities; obturating thrombi in the uterine, vesical, and hemorrhoidal veins, in the pampiniform plexus and in the dural sinuses. An *autochthonous* thrombus is one arising at the place in which it is found. A thrombus may, however, originate in the saphenous or the femoral, and growing upward by continued accretions finally reach the right heart. This process is spoken of as a *progressive* or *continued* thrombosis.

According to their structure venous thrombi may be divided into *white, red, mixed,* and *laminated*. The red ones are composed chiefly of red cells, and are formed as the result of the stasis of the venous flow. The white or gray thrombi are composed of blood plates, fibrin, or leucocytes, or contain these elements in varying proportions. They are formed during the circulation of the venous blood, and represent substances which are thrown out of the blood. In the case of alternating rates of current the thrombus may be formed of alternate layers of white and red clot (*laminated thrombus*), or the white and red portions may be mixed in varying proportions.

Venous thrombi may undergo simple, purulent, or gangrenous softening, organization, and calcification, or the elements of the thrombus may be loosened from their attachment to the vein wall and be carried away as emboli. Simple softening without embolism, organization, and calcification are the most favorable terminations of thrombosis; purulent and gangrenous softening the most unfavorable. The last two are due to infection, either hæmatogenous or extravascular. Embolism of portions of the thrombus is of importance according to the size of the transported material, and its origin from a bland or infected thrombus. Bland emboli may, if large enough, cause immediate death through obstruction of the pulmonary artery; when smaller they may obstruct the smaller branches of the pulmonary arteries and cause disturbances of the pulmonary circulation (*hæmorrhagic infarction*). The embolism may, however, be without effect if the collateral circulation is adequate, as is usually the case in the normal lung. The production of a hemorrhagic infarct of the lung is therefore dependent upon the size of the embolus and the condition of the pulmonary circulation. If the emboli are infective, metastatic abscesses and gangrenous processes are set up at the point of lodgment. The occurrence of multiple infected emboli gives rise to the picture of pyæmia. The larger infected emboli arising from venous thrombi find lodgment in the pulmonary circulation, but the bacteria arising from such thrombi may pass through the lungs and give rise to pyæmic abscesses in the spleen, kidneys, heart, etc. Fortunately, pyæmia is not to-day such a common condition as it was a generation ago. Thrombophlebitis of the uterine veins after abortion or childbirth is the most common source of pulmonary emboli. The diagnosis, prognosis, and treatment is the same as that discussed under phlebitis. (See also *Thrombosis.*)

Phleboliths.—As the result of the calcification of venous thrombi there arise the so-called *vein stones* or *phleboliths*. They usually lie free in saccular dilatations or in varices. Through rotation the calcified mass becomes rounded and smooth. They are found most frequently in the splenic veins, in the hemorrhoidal, vesical, and pampiniform plexuses. Adherent and partly organized thrombi may undergo calcification and appear as calcified masses in the vein wall.

Air Embolism.—This subject is discussed elsewhere. It is only necessary in this connection to mention general facts. Aspiration of air into the veins occurs particularly in the case of wounds of the veins of the neck and upper extremities, but may take place also through the

veins of the head, uterus, and stomach. Small amounts entering the venous blood are absorbed, but in the case of the aspiration of larger amounts, the air collects in the right ventricle and pulmonary artery, forming a foamy mass which the contractions of the ventricle are unable to drive onward, and death ensues from stoppage of the circulation. Opinions differ as to the amount of air which must be aspirated in order to bring about a fatal termination. It is probable that the amount varies; the fatal termination depending in part at least upon other factors.

Phlebosclerosis.—Sclerosis of the veins is a common process, but does not occur to the same extent or with the same frequency as arteriosclerosis. It may involve the entire venous system, or be confined to certain veins or even to portions of these. We may therefore distinguish a *diffuse, local,* and *nodal* phlebosclerosis. The changes are rarely so marked as to be distinguishable with the naked eye as is the case in the arteries. Microscopically the process is characterized by a subendothelial formation of connective tissue, which assumes a hyaline character, but undergoes fatty degeneration and calcification much more rarely than is the case in the arteries. In extreme cases a partial or complete obstruction of the vessel lumen may result. These cases are usually secondary to a chronic obliterating phlebitis. Many writers speak of a *primary* and a *secondary phlebosclerosis,* but such a classification is hardly practical, as it is not possible to differentiate between a sclerosis following a phlebitis and one arising primarily.

In the great majority of cases phlebosclerosis is the result of a previous phlebitis, either acute or chronic. In a certain proportion of cases, however, the hyaline thickening of the vein wall may be of a compensatory nature, the new formation of connective tissue and elastic fibres in the intima and media serving to compensate for a weakening of the vessel through loss of its elasticity. Such a weakening of the wall may be congenital, or acquired as the result of a chronic venous stasis following cardiac and pulmonary affections, chronic intoxications, cachectic conditions, etc. Statistics show that the veins of the lower extremities most frequently show a condition of sclerosis, those of the upper extremities next, the venæ cavæ following next in order. The portal, mesenteric, and splenic veins occasionally present a marked degree of sclerosis, which is sometimes associated with extensive calcification and fatty change. The changes may be so marked as to be plainly visible to the naked eye; the smaller branches of these veins may appear like sclerotic arteries. The condition is probably secondary to an old thrombopylephlebitis, but may be caused by an increase of portal pressure due to hepatic disease or portal stenosis. Chronic intoxication may also play a causal rôle. Such sclerosis of these vessels occurs in splenic anæmia and hepatic cirrhosis. Phlebosclerosis usually bears no relation to arteriosclerosis; the latter may exist in an extreme degree without the occurrence of changes in the veins. On the other hand, extensive phlebosclerosis may be seen involving a large portion of the venous system without corresponding changes in the arteries. Phlebosclerosis presents no recognizable clinical symptoms by which it can be diagnosed with certainty. In the case of the superficial veins the condition may be revealed by the presence of thicker and more rigid vessels. In such cases it is necessary to determine whether these appearances are transitory (phlebitis) or are permanent.

Stenosis and Obstruction of Veins.—The lumen of veins may be narrowed or wholly obstructed by compression from without, by diseased conditions of the vein wall, by anomalies of the vein contents, or by combinations of these factors. On account of the thinness and relatively slight elasticity of the walls of veins, particularly of the large veins, the lumen is easily influenced by pressure from without. Such pressure may be due to tumors, aneurisms, abscesses, inflammatory infiltrations, tuberculous processes, gummata, actinomycotic nodules, etc. The contraction of scar tissue in the neighborhood of a vein may likewise affect its lumen. In exudative phlebitis the thickening of the wall due to the inflammatory infiltration may be so great as partially or completely to obstruct the vessel. Sclerosis, calcification, and the thickening of the wall due to chronic phlebitis, may also lead to stenosis or to obliteration of the lumen. Thrombosis is the most common cause of venous stenosis and obstruction. Parietal thrombi lead to stenosis; obturating thrombi to complete obliteration of the lumen. As the result of the organization of thrombi the lumen of veins may be partly obstructed by diffuse or local thickenings of the wall, by connective-tissue bridges, etc. Phleboliths also cause a more or less complete obstruction of that portion of the vein in which they are found. The invasion of the lumen by sarcoma or carcinoma may also lead to the blocking of a vein by tumor masses growing within the vessel. Combinations of these different factors may also occur.

As the result of such obstruction or stenosis the region tributary to the affected vein becomes hyperæmic or cyanotic if the collateral circulation is not perfectly established. If the obstructed vein is a chief vein and the collateral anastomoses are poorly developed, œdema and effusions into the body cavities result. Hypertrophies, chronic inflammations, hemorrhages, and thrombosis also occur in the area of stagnation. The lowered nutrition and diminished resistance of the tissues in such an area predisposes to the occurrence of gangrene.

Stenosis of the superior vena cava may be caused by aneurisms, tumors of the mediastinum, lungs, thyroid, thymus, cervical glands, tuberculosis and actinomycosis of the bronchial glands, gummata, and scars of old tuberculous, syphilitic, and actinomycotic processes. The condition is revealed by the intense cyanosis and œdema of the head and upper extremities, and by the ectasia of the superficial veins. The innominate, vena azygos, inferior vena cava, common iliac, and femoral veins may also become stenosed or obstructed. In all these cases the diagnosis is dependent upon the location of œdema, cyanosis, hemorrhage, and other symptoms due to venous stagnation. The prognosis is more unfavorable in the case of obstruction of the superior vena cava than of the inferior. Cerebral symptoms soon appear in the former case, and dangerous effusions into the pleural and pericardial sacs may occur. In general the prognosis depends upon the nature of the accompanying condition, the rapidity with which the obstruction takes place, and the possibility of the formation of a collateral circulation. The working capacity of the heart is also an important factor. The treatment must be directed to the causal factor as well as to the condition itself. Tumors, aneurism, etc., must be treated according to the indications. In all cases the strength of the heart must be maintained. Venesection may give temporary relief from cerebral symptoms in obstruction of the superior cava. The treatment in the case of obstruction of the inferior cava is more favorable; rest, proper position of the extremities, bandaging, etc., may add much to the patient's comfort. In the case of portal vein obstruction Talma's operation may be attempted. Through new blood-vessels formed in the adhesions set up between the diaphragm, abdominal wall and the liver, spleen, and omentum, the portal stagnation may be relieved to such an extent that ascites is no longer produced.

Phlebectasias and Varices.—Diffuse dilatations of the veins are known as phlebectasias; local or partial dilatations as varices. Phlebectasias are due to a thinning of the vein wall caused by stretching; inflammatory changes in the wall follow secondarily. Varices are due to primary changes in the wall, usually of the nature of a phlebitis which results in a local weakening of the wall. Phlebectasias are cylindrical, serpentine, or cirsoid; varices are saccular, nodular, or bell-shaped. The different forms may pass into each other. The nodular varices may reach the size of a hen's egg. The walls of ectatic veins are at first thin and atrophic, but after a time there develops a productive phlebitis which leads to a connective-tissue hyperplasia and sclerotic changes

with atrophy of the muscle and elastic fibres. Occasionally the latter become increased. The vasa vasorum are dilated and show greatly thickened walls. The lumen of the vein may finally become partly or wholly obliter-

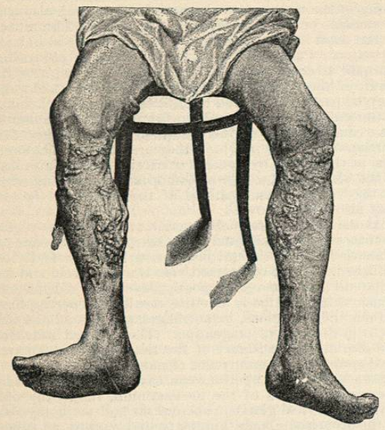

FIG. 5013.—Varices of Veins of Legs in a Man Aged Fifty-four Years, Otherwise Healthy. (After Schrötter.)

ated. In the walls of old phlebectasias there is not infrequently a deposit of lime salts. Large amounts of blood pigment may also be found in the wall as the result of hemorrhages. Thrombosis is of common occurrence in the saccular varices; through the calcification of the thrombi there arise phleboliths. The pressure of varicose portions of a vein when lying close together may lead to a pressure atrophy and confluence of the neighboring parts (*varix anastomoticus*). In this way a tumor-like formation composed of communicating cavernous blood spaces may be formed. In the true varix an increase in the length of the affected veins may sometimes occur. The saccular dilatations offer conditions favorable to thrombosis, and the impregnation of the thrombi with lime salts results in the formation of phleboliths.

The chief etiological factor in the case of both phlebectasias and varices is to be found in a general or local disturbance of the circulation leading to an increase of venous pressure and hindering the return flow of the venous blood. A general venous stasis may be caused by cardiac weakness, valvular lesions, diseases of the lungs leading to an obstruction of the pulmonary circulation, loss of the aid to the venous circulation through the lessening of the negative pressure in the thorax, effects of gravity, etc. A local venous stasis may result from the pressure of an aneurism, tumor, gravid uterus, enlarged lymph glands, or from the obstruction of the vein by a ligature, thrombosis, obliterating phlebitis, intravascular growth of tumor cells, constricting bands of scar tissue, tight belts, garters, bandages, etc.; or through inherent weakness of the vein wall (congenital varix), excessive muscular activity, loss of tension in the tissues surrounding the vein, influence of gravity, etc. Stasis in the portal vein is usually secondary to diseases of the liver. The overloaded rectum in cases of chronic constipation may likewise cause an obstruction to the venous flow in its neighborhood. The influence of gravity is shown particularly in the case of varices of the lower extremities. In individuals who habitually stand upright without adequate muscular movements of the lower limbs, especially in long-limbed individuals, varices of the veins of the leg are of very common occurrence. The saphena magna and its branches are most frequently affected. The aid given to the venous circulation by the intermittent pressure exerted upon the veins by the contractions of the muscles is in such cases but slight; through the influence of gravity the veins become dilated, their valves become relatively insufficient, and the vein walls, unable to support the long column of blood in the vena cava, become stretched. Veins which have been dilated for some time do not regain their old calibre. The valves retain their original size, though an increase in the tissues of the walls is necessary to the enlargement of the lumen. Heredity is a factor of some importance in the etiology of phlebectasias and varices. In just what lies the weakness of the vessel wall is not yet known. The state of the tension of the surrounding tissues is also an important factor.

Phlebectasias are most common in the veins of the hemorrhoidal plexus, the spermatic veins, pampiniform and pudendal plexuses, the utero-vaginal and vesical veins, the tributaries of the portal vein, the veins of the lower extremities, etc. Dilatations are of less frequent occurrence in the veins of the œsophagus, pharynx, meninges, brain, and uterus wall. They are of rare occurrence in the veins of the upper extremities. *Hemorrhoids* are the most frequent form of phlebectasias. The knots and convolutions of dilated veins may lie above the anus (internal hemorrhoids) or project from it (external hemorrhoids). Aside from the general etiological factors mentioned above, hemorrhoids may be caused by catarrhal inflammations of the mucosa of the rectum due to constipation. The extension of the inflammation to the vein walls leads to a dilatation of the lumen and a thinning of the wall. The collateral anastomoses of the hemorrhoidal veins make them subject to the influence of stasis in either the portal system or that of the vena cava. Hepatic cirrhosis is therefore associated with more or less extensive formation of hemorrhoids. The dilatations of the œsophageal veins in cases of portal stasis are also styled hemorrhoids (*œsophageal hemorrhoids*). The dilatations of the veins along the spermatic cord are known as varicocele; those of the veins of the abdominal wall in the neighborhood of the umbilicus in the collateral anastomosis of portal stasis are designated the "*caput Medusæ*." In the development of such a collateral circulation the veins of the œsophagus, the hypogastric, spermatic, inferior epigastric, internal mammary, and intercostal veins, the veins of the kidney capsule, the azygos veins, and the small branches in the suspensory ligament of the liver may become ectatic.

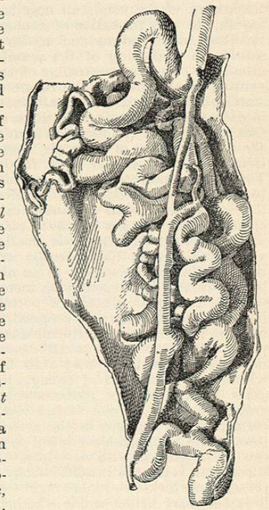

FIG. 5014.—Varices from Leg. (After Ziegler.)

The sequelæ of varix formation are of great importance. Rupture of the dilated veins is of common occurrence. This occurs most often in the case of hemorrhoids. Fatal hemorrhage may follow, or as the result of repeated small hemorrhages the most severe types of anæmia may

be produced. Varices of the œsophagus, stomach, and spleen may give rise to fatal hemorrhage or to severe anæmia. Death may follow the rupture of a varix in the brain or meninges. Next to rupture and hemorrhage thrombosis is the most important result of venous dilatation. Embolism from such thrombi is most common in the case of hemorrhoids (after operation) and in the veins of the lower extremities. Thrombophlebitis and lymphangitis are not infrequent complications of varices, and may result fatally. Further, as the result of phlebectasias and the stagnation of the venous flow, the tissues of the region tributary to the affected veins present a condition of chronic œdema. In the case of the skin there

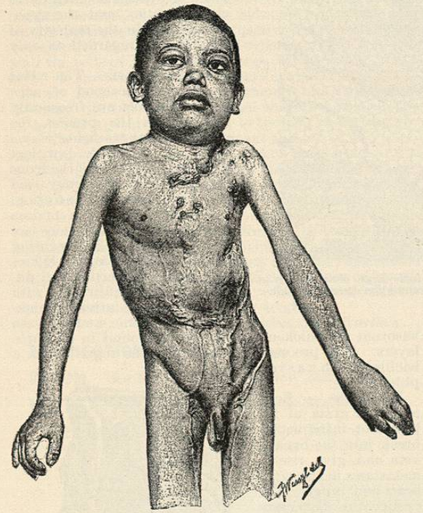

FIG. 5015.—Phlebectasia of Superficial Veins in Case of Obstruction of Venæ Cavæ due to Actinomycosis of Mediastinum. (After Schrötter.)

may be an increased secretion of sweat; in mucous membranes a condition of chronic catarrh results. Hemorrhage by diapedesis from the smaller capillaries is of frequent occurrence. The skin over varices often shows a marked atrophy, pigmentation, desquamation, or a condition of chronic eczema (eczema varicosum). As a result of the chronic venous congestion and chronic œdema the skin is often the seat of a chronic inflammation which leads to a fibroid thickening of the dermis and subcutaneous tissues. Over the legs these thickenings may form irregular folds or nodules (elephantiasis phlebectatica). Ichthyotic thickenings of the stratum corneum may also be produced, the epidermis presenting the appearances of warty excrescences. The neighboring periosteum may likewise be involved in the inflammatory process and a new formation of bone may take place (periostitis ossificans). As the result of slight injuries or wounds torpid ulcers very often form (*ulcera cruris, varicose ulcers*). These run a very chronic course, and may involve a very large part of the skin of the leg. Abscesses of the skin are frequently associated with these. Thrombophlebitis, pyæmia, and amyloid disease may follow such ulcerations.

The diagnosis of varices offers no difficulties. The prognosis is dependent upon the extent of the varices, their etiology, and the occurrence of such complications as thrombophlebitis, embolism, hemorrhage, chronic ulcerations, amyloid disease, etc. The treatment of varices is chiefly palliative: elastic stockings, bandages, etc., ligatures, cauterization, electropuncture, excision, injec-

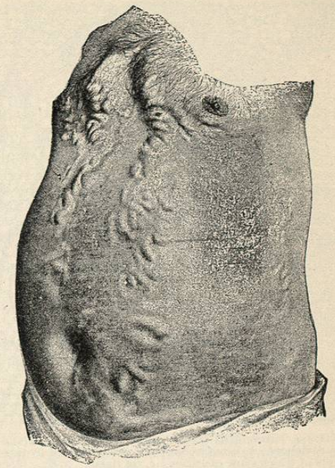

FIG. 5016.—Formation of Collateral Anastomoses Between the Superior and Inferior Epigastric Veins in Obstruction of Inferior Vena Cava. (After Schrötter.)

tions of ergotin, chloral, etc. The injection methods are at present but little used, and are in themselves dangerous. Trendelenburg's double ligature is the operation

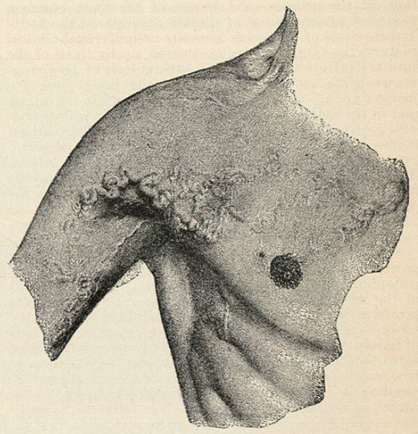

FIG. 5017.—Development of Collateral Circulation in Thrombosis of Subclavian Vein. (After Schrötter.)

most frequently carried out. (See *Anus and Rectum. (Surgical.)*; *Varicose Veins*; and *Sexual Organs, Male, Diseases of.*)

TUBERCULOSIS.—Tuberculosis of the veins occurs most frequently in the veins of tuberculous organs, but also

not rarely in the larger veins in the neighborhood of tuberculous lymph glands, etc. In the latter case the infection of the vein wall takes place through direct extension or through the lymphatics. In rare cases primary tuberculosis of the intima of veins may take place through the presence of tubercle bacilli in the circulation. Involvement of the veins from neighboring tuberculous processes is much more frequent in the case of the veins than of the arteries, and is one of the most common modes of dissemination of disease throughout the body. Tuberculosis of the veins presents no individual clinical picture, the symptoms being covered up by those of the primary infection. In miliary tuberculosis of hæmatogenous origin the veins throughout the body show numerous miliary tubercles in their walls, both in the intima and in the media, appearing as small gray or yellow nodules projecting into the lumen of the vessel. The earliest stages of these may appear as small collections of leucocytes or as agglutination thrombi resting upon the intima. Epithelioid proliferation of the cells of the intima occurs later, and finally typical tubercles are formed. In the case of extension from neighboring tuberculous foci the veins come to present the picture of a caseating tuberculous phlebitis.

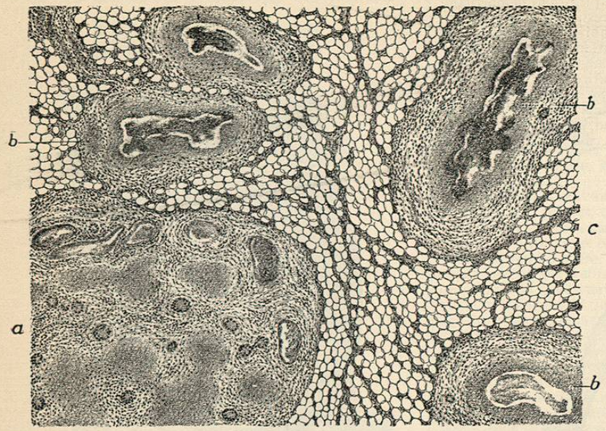

FIG. 5018.—*a*, Tuberculous lymph gland with giant cells and caseous foci—large blood-vessels at the periphery; *b*, veins whose walls are thickened by tuberculous granulation tissue, the inner layers of which show caseation; *c*, fat tissue. × 28. (Ziegler.)

SYPHILIS.—The most frequent changes in the veins ascribed to syphilis are of the nature of an obliterating endophlebitis or a chronic periphlebitis. The small veins of the meninges, brain, liver, and other internal organs are most often affected. In the neighborhood of the affected veins there is usually found a gumma or focus of induration. In the great veins, and particularly in the subcutaneous veins, symptoms of an acute phlebitis occur frequently in the early stages of syphilis, as shown by swelling, redness, and tenderness corresponding to the course of the affected vessel. In the later stages gummatous infiltrations of the vein walls and the neighboring tissues also take place. The proliferation and formation of granulation tissue may begin in the intima, media, or adventitia. Thrombosis may be associated with any of these processes. Sclerotic changes, calcification, and fatty change may follow. The portal vein may show marked changes of this character. Chiari has described a proliferating endophlebitis of the hepatic veins which he regarded as of syphilitic origin. Similar changes have also been found in the veins of the intestinal wall and mesentery associated with multiple strictures of syphilitic nature. In congenital syphilis the veins of the umbilical cord may show a thickening of the intima, due to a cellular proliferation or new formation of connective tissue. A peripylephlebitis syphilitica neonatorum has also been described. With the exception of the typical gummata which occur but rarely in the vein walls, the venous changes ascribed to syphilis present no distinctive characteristics, and the syphilitic origin in the majority of cases can be regarded as only probable.

ACTINOMYCOSIS.—The veins in the neighborhood of actinomycotic foci are frequently involved in the process, the resulting changes being of the nature of a chronic purulent phlebitis. Rupture of the focus through the intima may lead to a metastasis of the infection.

LEPROSY.—In lepra nodosa and anæsthetica the veins are frequently affected, presenting nodular thickenings. Microscopically the media and adventitia are infiltrated with round cells, the intima is thickened and hyaline, and the vasa vasorum are thickened. Bacilli are found in all three layers. The process is therefore to be regarded as a bacillary invasion phlebitis.

PARASITES. — Echinococcus cysts of the liver not infrequently break into the hepatic vein and give rise to metastases in the right heart and lungs. The cysticercus, filaria sanguinis hominis, and distoma hæmatobium are also found in the veins.

FOREIGN BODIES.—Under very rare conditions foreign bodies which have entered the veins may be found attached to the wall of the vessel. One case is reported of the finding of a small pistol bullet in the internal jugular.

NEW GROWTHS. — Hæmangiomata, angiosarcomata, and endotheliomata may be primary in veins, particularly the smallest veins. Primary myomata of veins have also been described. Many of the cases reported as primary tumors of veins are of doubtful nature, it being very probable that the vein walls were secondarily involved. In so far as the metastasis of tumors is concerned, the veins play a much more important part than the arteries. The secondary involvement of veins by primary carcinoma and sarcoma of other organs and tissues is of very common occurrence. In the extension of the tumor into the vessel wall rupture of the tumor cells into the lumen is prevented for a long time by the endothelium, which is apparently very resistant to the advancing tumor cells. Large nodules may project into the lumen of the vein, but on microscopical examination the endothelium will be found intact over the growth. Carcinomata break through the walls of large veins more rarely than do sarcomata, because of the plastic influence which the carcinoma cells exert upon the connective-tissue cells of the vessel wall. As the carcinoma cells approach the vein wall the cells of the latter undergo proliferation, and a layer of new tissue is formed between the vein and the tumor. In the case of sarcoma there is but little plastic influence exerted upon the vessel wall, the cells of the tumor being genetically related to those of the vein wall.

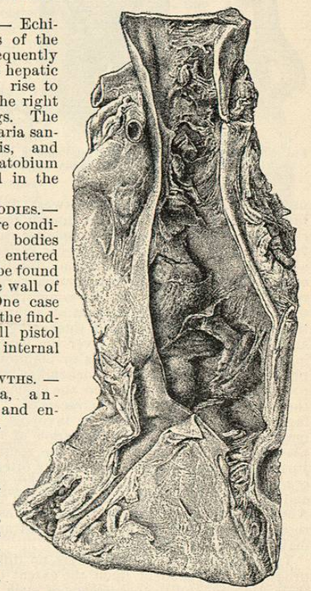

FIG. 5019.—Changes in Inferior Vena Cava due to Syphilis. (After Schrötter.)

In the case of primary malignant tumors of the adrenals, kidneys, testis, etc., large tumor masses not infrequently grow into the veins and may extend into the vena cava. Portions of these may break loose and lodge in the heart or pulmonary arteries. Free tumor emboli may occasionally be found in the right ventricle. Secondary carcinomata of the liver frequently break into the hepatic veins and give rise to metastases in the lungs. In the case of tricuspid insufficiency or violent spasmodic coughing a retrograde transportation of tumor cells may take place (retrograde metastasis). In very vascular tumors the veins are often markedly ectatic.

Aldred Scott Warthin.

VENESECTION. See *Blood-letting.*

VENOM. See *Poisonous Reptiles*, and *Toxins, etc.*

VENTILATION. See *Air*, and *House Sanitation.*

VENTNOR, ISLE OF WIGHT.—The Isle of Wight possesses certain favorable climatic features, both on account of its geographical position off the south coast of England, and also on account of its peculiar geological formation. It is at Ventnor, however, on the south side of the island, that the conditions are the most favorable for a winter resort, and Ventnor rivals Bournemouth in its reputation for the climatic treatment of pulmonary tuberculosis, both resorts having a royal national hospital or sanatorium for cases of consumption.

In general, the climate of the Isle of Wight is a marine, moist, mild one, cool in summer and mild in winter, with the prevailing winds from the southwest, *i.e.*, from the ocean. At every season of the year one can obtain here a mild tonic sea air.

In the southern portion of the island a great range of chalk downs stretches from east to west, and in the southeastern portion these downs form high, precipitous cliffs constituting the coast line, broken here and there by "chines" or deep indentations. Between the sea and these high cliffs is a narrow strip of coast-land, called the "Undercliff," consisting of irregular terraces of chalk and sandstone, the results of landslips from the cliffs above. This undercliff is from 100 to 150 feet above sea level, and extends for a distance of about six or seven miles. The aspect of the undercliff is, in general, due south. In the rear, to the north, rise the cliffs, the precipitous face of the downs, which are from 400 to 800 feet high, thus affording complete protection from the northerly winds.

Upon the undercliff, and upon a series of rocky terraces formed by the landslides, Ventnor is built, a town of about 6,000 inhabitants, stretching from the beach up the cliff to a height of nearly 500 feet; consequently the streets are very steep, and among the picturesque features of the place are this precipitous steepness and the arrangement of the streets one above another. Indeed, there are but few level stretches, and those are down by the sea.

The soil consists of soft sandstone and chalk, and is fairly absorbent and dry, but it forms readily, in dry weather, a light irritating dust. The vegetation of the undercliff is exceedingly luxuriant and varied, a proof of the mildness of the climate and the protected situation. Spring opens early, and many plants and shrubs blossom

FIG. 5020.—View of Ventnor, Looking Westward.