any more difficult for a witness to swear falsely in regard to blood stains than in regard to any other subject. But it would be quite possible on cross examination to ask the expert whether, in measuring the corpuscles obtained from a stain, he had measured all the well-defined corpuscles in the field of the microscope, or whether he had selected the large corpuscles and rejected others which were smaller.

In treating blood stains with solvents of fibrin, to liberate the corpuscles for measurement, it has been found in many cases that the corpuscles were smaller than in fresh blood unless maceration was long continued, old stains requiring several weeks before the corpuscles were in a condition to be measured. When a biconcave blood corpuscle is placed in water, the coloring matter (hæmoglobin) is dissolved out, the corpuscle swells up, thickens at the edges, becomes transparent and spherical. In this condition the diameter of the corpuscle becomes less than normal. It thus happens in examining blood stains that all corpuscles which have lost their color or have become spherical are by some experts rejected and not measured. Some corpuscles in fresh blood have much more coloring matter than others, and these corpuscles retain their color and form (as we think) much longer than paler corpuscles. We are not aware that any fluid used or likely to be used for softening blood stains will cause the corpuscles to become larger than normal. In this statement the most noted authors agree.

If the corpuscles obtained from a stain do not recover their normal dimensions it is almost absolutely certain that their average measurement will be less than normal and *never* greater than normal. Thus, in the language of Professor Wormley, we may confidently say: "*Thus, then, while the blood of man might on account of contractions in diameter of the blood corpuscles be confounded with that of some animal having smaller corpuscles, the reverse could never occur.*"

From this discussion I claim that it has been proved beyond any reasonable question:

1. That in favorable cases blood stains can be so treated that reliable measurements and credible diagnosis of their origin can be given, as shown in the tables given and in others which might be referred to.

2. That if error occurs on account of imperfect restoration of the form and diameter of the corpuscles obtained from a stain, proved (by (*a*) the guaiacum test; by (*b*) the spectroscope; by (*c*) the production of hæmin crystals) to be blood, the error, if any, will be to make human blood appear like that of one of the inferior animals, and never to mistake the blood of the ox, pig, horse, sheep, or goat for human blood.

3. In general, when a stain has been proved to be blood by the above tests, it may be decided certainly whether it is or is not mammalian blood. So, also, a stain from the blood of the ox, pig, horse, sheep, and goat may be distinguished from human blood, thus confirming the claim of an accused person in many cases that his clothes are *not* stained with human blood. This negative testimony is certainly quite as important in many cases as testimony inculpating a prisoner.

Lastly, the expert can say, when the average of a suitable number of corpuscles from a blood stain corresponds with the average of fresh human corpuscles, that the stain is surely not from the blood of the ox, pig, sheep, or goat.

Such testimony by a skilled microscopist is of untold importance in saving the lives of the innocent, and often in overthrowing the plea of those who are guilty. Such testimony is quite as reliable and important to the welfare of society as that of the chemist who testifies to the presence or absence of poison that might have some resemblance to the many recently discovered ptomaïns.

The testimony of the expert might take the following form, as recommended by C. H. Vibbert, "Précis de médecine légale":

"This stain is *not* composed of the blood of such an animal [ox, sheep, horse, pig, or goat] as the accused claims. It is like the blood of man, or some animal having corpuscles very nearly the same size as those of man, as the dog or rabbit."

Or the declaration may take the reverse form, thus: "This stain is *not* composed of human blood; it might be the blood of a horse, ox, pig, sheep, or goat, as claimed by the accused."

Such declarations are justified, then, and then only, when the examination has been conducted with great care and the measurements have been made with reliable instruments.

Moses C. White.

BLOOD-VESSELS, HISTOLOGY OF.—The Arteries.—An artery consists of three coats, which, named from their relative position, are the inner, the middle, and the outer. The structure and relative thickness of these coats vary in vessels of large, medium, and small calibre. A medium-sized artery, such as the radial, shown in the accompanying illustration (Fig. 621), has been taken as the type and first described in detail, the structural peculiarities of the larger and smaller vessels being subsequently noticed.

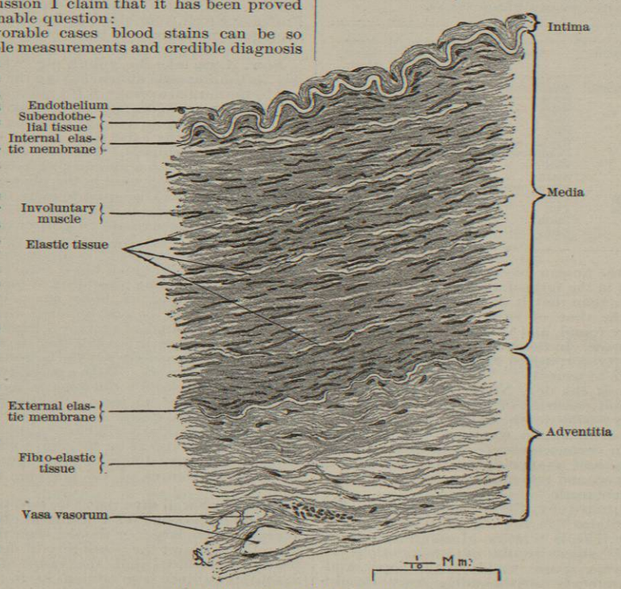

Fig. 621.—Section of Human Radial Artery.

The *inner coat*, or *tunica intima*, is the thinnest coat of the artery and consists of three distinct structures: (*a*) an endothelial lining; (*b*) a layer of subendothelial connective tissue, and (*c*) an internal elastic membrane. The endothelial lining consists of a single layer of flat endothelial cells, each containing a centrally situated nucleus of round or oval form. When examined in a transverse section of the vessel, in which the cells are seen in profile, the endothelial plates are inconspicuous, the nucleus often being the only part of the cell readily made out. Examined from the surface, after staining with silver nitrate, the boundaries of the individual cells are clearly defined by the darkly tinted cement substance which unites the endothelial plates. In such preparations the lining cells appear spindle-shaped or lanceolate in form, their long axes corresponding with that of the blood-vessel. Careful examination of the outlines of the cell shows these to be serrated or sinuous, contrasting with the more regular lines of apposition in epithelial tissue. The lining cells of blood-vessels were first described by Kölliker and others as epithelium; later, His applied to them the name endothelium as more appropriate for elements derived from mesoderm and closely related to serous surfaces. In principle, endothelial cells are modified connective-tissue elements.

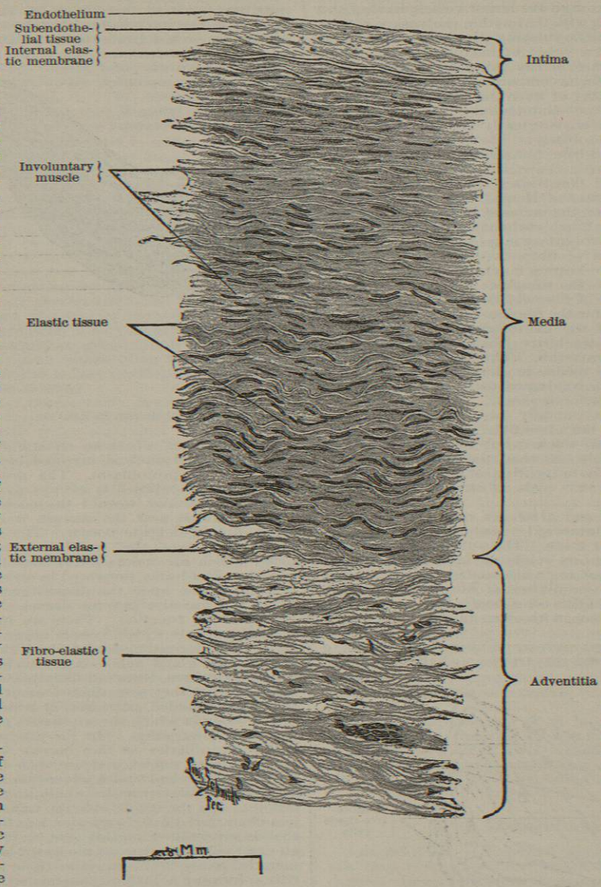

Fig. 622.—Section of Human Aorta.

The *subendothelial layer* consists of bundles of white fibrous connective tissue interwoven with a delicate network of elastic fibres, and meagrely distributed branched connective-tissue corpuscles lying within the lymph spaces of the tissue.

The *internal elastic* membrane appears in arteries of medium size as a structureless, glistening, corrugated band that stands out as the most conspicuous structure of the intima. It constitutes the most external layer of the latter and forms a sharp line of demarcation between the narrow and faintly stained intima and the broad and more deeply tinted media. While apparently a homogeneous membrane in the smaller arteries, in vessels of large size the internal elastic membrane is represented by a number of delicate lamellæ of elastic tissue, which are pierced by apertures of varying size. The entire structure in these cases has been appropriately named the *fenestrated membrane of Henle*, in recognition of the anatomist who called attention to its peculiar arrangement. The majority of the elastic fibres forming this reticulated network run longitudinally, but are intermingled with some oblique fibres as well as a limited number of branched connective-tissue corpuscles.

The *middle coat*, or *tunica media*, is the thickest coat of the artery. It consists of circularly disposed lamellæ of involuntary muscle intermingled with connective tissue in which elastic fibres are conspicuous. The individual muscle cells are irregularly spindle-shaped, often with ragged outlines, and possess the characteristic rod-shaped nuclei. The muscle cells of the media are shorter and thicker than the slender and more elongated corresponding elements in other localities. The individual cells, held together by interstitial cement substance, are grouped into illy defined bundles, which are closely associated with small spiral bundles of white fibrous and elastic connective tissue, the whole forming the most compact coat of the artery. The elastic fibres are very numerous, and, in ordinary preparations being almost unstained, stand out in marked contrast among the more deeply stained masses of involuntary muscle.

The *external coat*, or *tunica adventitia*, is composed of closely felted bundles of white fibrous and elastic tissue arranged in fine wavy masses. Many of these bundles have a longitudinal direction, while others pursue an oblique course. Connective-tissue cells are present in considerable quantity. While looser in texture and apparently of less strength, the adventitia is nevertheless more resistant than either of the other coats, and is the tunic upon which the integrity of a ligature chiefly depends. The walls of the arteries are nourished by a

special system of minute vessels, the *vasa vasorum*, which enter the adventitia and penetrate the media. The vasa vasorum do not spring from the vessels which they supply, but are usually derived from neighboring arteries, the blood flowing through the larger vessels having as a rule no direct relation with that within the vasa vasorum.

The differences distinguishing the coats of a large artery from those of a vessel of medium size consist, in general, of an increase in the thickness of the various tunics, as shown in the accompanying Fig. 622.

The immediate lining of even the largest arteries consists of a single layer of endothelial cells; the subendothelial tissue, however, is greatly thickened not only relatively but also absolutely. The subendothelial bundles of white fibrous tissue are coarser, and the elastic tissue is present in greater amount and constitutes the fenestrated elastic membrane of Henle. The media likewise is thicker, the increase, however, being due not so much to the greater amount of involuntary muscle as to the addition of fibro-elastic tissue, which here bears a much greater proportion to the muscular tissue. This increase of fibro-elastic tissue is responsible for the diminution of elasticity and the increased stiffness which characterize the walls of the larger arteries. The elastic tissue of the media in the latter vessels is often condensed to form an *external elastic membrane*, which defines the boundary between the media and the adventitia.

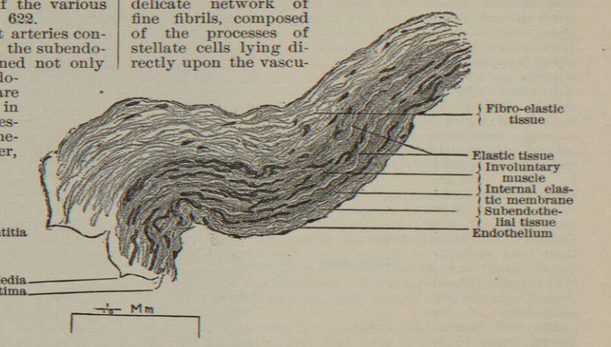

FIG. 624.—Section of Human Radial Vein.

The adventitia is also thicker, but the increase in this tunic is proportionately less than that affecting the intima and media. The adventitia of small arteries, in relation to the other two coats, is thicker than that of the large arteries.

The small arteries and arterioles, on the other hand, differ from the medium-sized vessels by a reduction in the thickness of their coats. The subendothelial tissue almost entirely disappears, while the involuntary muscle of the media is reduced to an attenuated layer of muscle cells, with little or no admixture of fibro-elastic connective tissue. The adventitia consists of a few longitudinal bundles of fibrous tissue, with often an absence of elastic fibres.

The capillaries are microscopic vessels establishing communication between the arteries and the veins. In

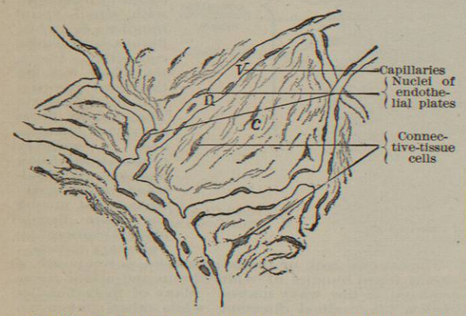

FIG. 623.—Capillary Blood-Vessels from Mesentery of Dog. × 240.

the tips of the fingers, the tips of the toes, the tip of the nose, the splenic pulp, and the erectile or cavernous tissue of the genital organs the arteries communicate directly with the veins. A capillary consists of a single layer of nucleated endothelial cells united by a small amount of intervening cement substance. By reason of the character of their thin walls, they are virtually protoplasmic tubes admirably adapted to facilitate the distribution of nutritive fluids to the tissues or, as in the pulmonary alveoli, to the interchange of gases. As the capillaries become larger a delicate tunica adventitia is superadded, which is formed of a delicate network of fine fibrils, composed of the processes of stellate cells lying directly upon the vascular walls. Each of these stellate cells consists of a large elongated nucleus, invested by an extremely delicate layer of protoplasm. The protoplasm of which the cells are composed is always more abundantly and distinctly granular toward the centre and around the nucleus, whilst near the margin it is quite clear, and thins off to a delicate border. The capillaries by their union constitute the capillary plexuses, *retia capillaria*, which are more or less constant in the different organs. The forms presented in these plexuses depend to some extent upon the disposition of the elementary parts, and are also in some degree dependent upon the energy of the function. There are in many organs certain tissues into which vessels never penetrate,—as the transversely striped muscular fibres, the nerve fibres, cells of all kinds, gland acini,—and which, therefore, according to their form, trace out definite courses for the capillaries, so that they sometimes present elongated meshes, sometimes rounded, narrower, or wider reticulations.

The mode in which the capillaries pass into the larger vessels is interesting. On the arterial side it is found that the capillaries, as they become wider, present more closely placed nuclei, and are then invested externally with a structureless tunica adventitia and solitary muscle cells, the whole structure exhibiting the aspect of arterioles. On the venous side the transitionary vessels are less characteristic for a greater length. The first thing that is superadded, on this side, to the capillary wall is an external, homogeneous nucleated layer, which may be regarded as a sort of connective tissue—the tunica adventitia,—forming a distinctly laminated structure, the veniol. Besides the finest capillaries, which, however, always admit the passage of very flexible blood corpuscles, some observers have admitted the existence of still finer vessels, the so-called *vasa serosa*, which no longer allow of the passage of blood, but only of its plasma. Hyrtl thinks that it is necessary to admit of the existence of vessels of this kind in the cornea.

Among the *structural peculiarities* of certain arteries may be mentioned the slight development, or even complete absence, of the subendothelial connective tissue in the cœliac, the external iliac, the mesenteric, the renal, and the uterine arteries of young individuals. The aorta possesses a marked development of the subendothelial

connective tissue, as well as the presence of longitudinal muscle cells within the intima. Longitudinally disposed muscle is present in the media of the subclavian artery and in the adventitia of the iliac, the superior mesenteric, the splenic, the renal, and the dorsalis penis, as well as in the umbilical arteries of the fœtus. Some arteries, notably the vessels within the cranial cavity and the vertebral canal, possess coats which are very thin in proportion to their calibre. In these vessels, however, it is the media and the adventitia that suffer reduction, the intima remaining almost unaffected.

A typical vein, as shown in the accompanying drawing, (Fig. 624) has thinner walls and a larger lumen than the corresponding artery; to this rule, however, the pulmonary veins are exceptions, since they are of the same capacity as, or of less capacity than, the pulmonary arteries. The constitution of the coats of a vein is less constant than it is in an artery, variations in the arrangement, proportion, and amount of the component tissues being very frequently encountered.

The *inner coat*, or *tunica intima*, consists of (*a*) endothelial cells, (*b*) subendothelial connective tissue, and (*c*) elastic fibres. The endothelium is composed of a single layer of cells held together by interstitial cement substance. The cells are shorter and wider than the corresponding lanceolated elements of the artery. The subendothelial connective tissue is very scanty in the majority of veins and rudimentary or even entirely absent in the smaller ones. The elastic tissue often represented by a few longitudinal bundles is less developed and does not form a fenestrated membrane.

The *middle coat*, or *tunica media*, is much thinner and looser than in the artery, chiefly on account of the admixture of connective tissue associated with the circularly disposed involuntary muscle cells. The elastic fibres, so conspicuous in the media of the artery, are very few in the vein.

The external coat, or tunica adventitia, is the thickest coat of the vein, reaching often twice or even three times the breadth of the media. It consists of wavy bundles of white fibrous tissue, usually following a circular direction, among which a few longitudinal fibres may be found; the greater number of the latter fibres are of the elastic variety. Connective-tissue cells are present in considerable number. The walls of the vein are nourished by vasa vasorum similar to those supplying the arteries.

Many veins, especially those of the lower extremities, are provided with valves; these are crescentic folds of the intima, which are brought into apposition by the dilatation of the pockets formed between the attached portion of the valve and the wall of the vessel. The valve consists of an extension and thickening of the fibro-elastic tissue of the intima, covered on each side by endothelial cells. The free edge of the valve is thicker than the attached border.

The variations in the structure of veins depend largely upon modifications of the muscular tissue. The involuntary muscle is best developed in the media of veins of the inferior extremities; in the veins of the gravid uterus involuntary muscle occurs in the adventitia as well as in the intima. Additional longitudinally disposed bundles of muscle are present in the inner part of the media in the mesenteric, the iliac, the femoral, and the umbilical veins. Involuntary muscle is sometimes encountered in the adventitia, in the abdominal cava, the axillary, the hepatic, the external iliac, the superior mesenteric, the renal, the splenic, the spermatic, and the azygos major veins. The presence of striated muscle in the pulmonary veins and cardiac ends of the venæ cavæ is to be regarded as an extension of the striped muscular tissue of the auricular wall.

Robert Formad.

BLOOD-VESSELS, PATHOLOGICAL ANATOMY OF.—THE CAPILLARIES.—The capillaries are less independent structures than the arteries and veins, and the pathological changes in the capillary wall are generally intimately connected with lesions in the surrounding tissues. The important rôle played by the capillaries in the different forms of inflammation is discussed in the article on *Inflammation*.

All tissue proliferation, whether inflammatory or non-inflammatory, is almost without exception associated with the new formation of capillary vessels by processes of budding of pre-existing capillary endothelium, and these changes are described in the article on *Regeneration*.

Capillary new formation lies at the bottom of tumors composed of capillaries, the capillary angiomata and teliangiectasias of congenital or acquired origin; they form mostly flat tumors of the skin and subcutaneous tissue, of a bright red color and a soft feel; the capillaries in them have thick cellular walls, and there is abundant evidence that new capillaries have been formed. Similar changes occur in capillaries in other tumors (see *Tumors*).

Long-continued congestion will result in dilatation of the capillaries, capillary ectasis or capillary aneurism; this is shown well in passive congestion of the lungs.

Hyaline degeneration of the capillary walls is frequently observed in the brain, especially in paralytic dementia, but also in the kidney, the conjunctiva, the lymph glands, and in various tumors. Hyaline degeneration of the glomeruli is an important alteration in chronic nephritis, especially the interstitial form. Hyaline degeneration is a rather vague term and the exact origin of hyaline material is not understood. In certain tumors mucoid degeneration of the capillary walls sometimes takes place.

Mallory in his study of colloid or hyaline changes in the brain shows that the material is always deposited in the vessels; in the larger vessels the middle coat is earliest and most affected. The hyaline material has a marked tendency to calcification, and in some cases the capillary network of the central cortex, the dentate nucleus, and the granular layer of the cerebellum undergoes hyaline change with calcification; this causes atrophy of the included nervous tissue and leads to the formation of sand-like deposits and of stone-like concretions.

Fatty changes in the capillary epithelium* occur frequently, especially in the nervous system and in the various toxic and infectious states. The lesions in the capillaries that may underlie the tendency to spontaneous hemorrhage in the hereditary and acquired hemorrhagic diathesis have not yet been cleared up; in these conditions it is difficult to determine whether the hemorrhages result from diapedesis or from rhexis. The relations of the capillaries to thrombosis and embolism are described in the articles dealing with those subjects.

Calcareous infiltration of the capillary walls occurs in the brain of old people; generally calcification is preceded by hyaline degeneration. Calcification of the capillaries also takes place in certain tumors, especially psammoma.

Amyloid degeneration is an important change in the capillary walls, observed always in the capillaries of the spleen, kidneys, liver, etc., in general amyloidosis. The amyloid material appears first in the delicate capillary sheath outside of the epithelium, which is soon crowded to one side and the lumen closed.

THE ARTERIES AND VEINS.

ABNORMITIES.—Congenital abnormities of blood-vessels may concern width, thickness of wall, origin, and course. Deviations from the normal in these respects on the part of the smaller and medium-sized vessels are considered to best advantage in connection with the normal anatomy of blood-vessels.

The congenital defects and anomalies of the *pulmonary artery* and *aorta* are closely associated with defects and anomalies of the heart. Among the important irregularities of the primary vessels is transposition of the aorta and the pulmonary artery. This may occur in hearts that are not otherwise defective or in association with other developmental anomalies of the heart. Transposi-

* The term "epithelium" is used in place of endothelium in this article.—L. H.

tion of the venæ cavæ and the pulmonary vein is also described. In nearly all cases of this kind the foramen ovale is open to a greater or less extent and generally the ductus arteriosus is pervious. In some cases the only communication between the two circulatory systems was through the widely open foramen ovale. Death occurs in early infancy.

The septum between the aorta and pulmonary artery may be defective. In an instance of this kind that I examined there was an oval defect about 1.5 cm. in its greatest diameter between the pulmonary artery and the aorta, so that the beginning of the aorta and pulmonary artery had a common trunk from which emerged, in the usual places, the right and left pulmonary arteries and the branches of the arch of the aorta. The ductus arteriosus was patent and large, the aorta distal to the duct wider than that proximal to the duct. The foramen ovale was widely open. The large veins and the heart were normal.

Instances of aortas with double arches are described by Hommel, Curnow, Malacarne, Zagorski, Welch, and Lee Shaw. According to Lee Shaw, Hommel, in 1737, was the first to record this anomaly. In his case the two divisions united after encircling the trachea, and passed downward on the left side of the vertebral column. Malacarne's report, as described by Meckel and Quain, differs from all the others. Five valves guarded the aortic opening of the left ventricle and the arterial trunk immediately divided into two branches, which before uniting embraced the pulmonary artery, trachea, and œsophagus; from each division was given off in succession a subclavian, an external, and an internal carotid artery. In Welch's case, the posterior branch passed between the trachea and œsophagus, and from each division arose a common carotid and a subclavian artery. On the posterior branch was found an aneurism from which the right common carotid took origin. Lee Shaw's specimen differs from all of those described except Malacarne's, in that the two divisions unite behind the œsophagus. It differs from all in that the right branch is the larger, the junction of the two branches is on the right side, and the descending aorta passes downward on the right side, to the upper border of the eighth dorsal vertebra. This arrangement not only resembles the vascular arrangement in the reptile, but also more closely follows the distribution in the bird than it does that in the mammal.

Such anomalies of the aorta are probably due to the persistence and enlargement of fetal vessels which normally become obliterated. From the embryonal aortic bulb two vessels arch backward, one on either side of the foregut, forming the first pair of vascular arches, and descend along the sides of the notochord as the primitive aortæ. As the heart gradually moves away from the head, four more pairs of vessels develop, which connect the bulb with the descending trunks; making in all five pairs of arches, one for each branchial plate. Zimmerman describes an additional pair between the fourth and fifth.

"The bulb, primarily a single cavity from which, through two common trunks, one on each side, blood is sent to all the arches, is divided by a septum parallel to its long axis into two compartments: one becoming the pulmonary artery, situated anteriorly, and continuous with the fifth pair of arches, the other forming the systemic aorta, placed posteriorly, and communicating with the fourth pair, through which all the arches above receive their blood supply. Notwithstanding this separation near the heart, both pairs of arches, *i.e.*, the fourth and fifth, ultimately empty into the descending aorta which has been formed by the coalescence of the primitive aortæ.

"This symmetrical arrangement of the vascular system is soon destroyed by the obliteration of certain vessels. Even before the last arch is perfectly formed the connections between the first pair and the descending aortæ may be destroyed. However, the fourth and fifth arches, lying nearest the heart and soon exceeding the others in size, are the chief factors in this transformation. The vascular arches on the left side continue to increase in size and in greater part become permanent, while those on the right gradually become obliterated, except where they furnish the supply to permanent arteries.

"On the left side the fourth arch becomes the arch of the aorta and, in the fœtus, is constructed between the origin of the left subclavian and the junction of the ductus arteriosus, forming the isthmus, which soon after the closure of the duct attains the diameter of the adjacent trunk. The fifth, on this side, forms a part of the

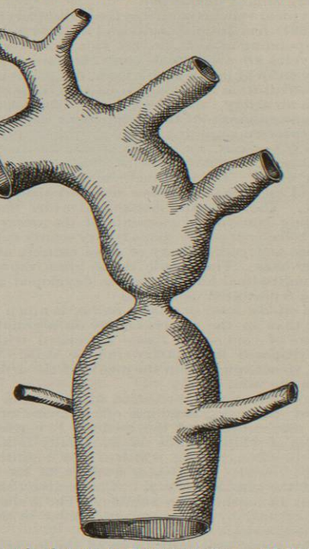

Fig. 625.—Stenosis of Aorta at Ductus Arteriosus. (Modified from Cruveilhier.)

left pulmonary artery and the ductus arteriosus, which is occluded soon after birth but remains as a round cord. On the right side the fourth arch is permanent for a short distance as the innominate and right subclavian arteries, as far as the origin of the vertebral, beyond which it diminishes in size and finally disappears. Only a small portion of the fifth persists on this side as the root of the right pulmonary artery; the distal portion becomes obliterated."

In Lee Shaw's case, the fourth arch on the right side not only remained patulous, but exceeded the left in size. The fourth arch of the left side retained some of its fetal characteristics, because it was doubtless unnecessary for the isthmus to enlarge after occlusion of the ductus arteriosus, as the blood current could easily pass through the right arch.

Premature obliteration of the ductus arteriosus has been described as causing an abnormal smallness of the pulmonary artery. The pulmonary artery may divide at a point lower than usual, or arise by two distinct roots, instead of continuing into the aorta as the ductus arteriosus; it may form the left subclavian artery. Supplementary or vicarious branches distributed to the lungs may arise from the aorta as far down as the cœliac axis.

In narrowing or obliteration of the aorta after having given off the vessels to the head and upper extremities, the inferior parts of the body are supplied with blood from the pulmonary artery through the ductus arteriosus; the descending aorta is said to be given off from the pulmonary artery. Now if the degree of constriction of the aorta be but slight at the time of birth, the ductus arteriosus may become closed. Later in life great narrowing and even complete obliteration of the aorta may take place (Fig. 625).

The theory usually offered in explanation of the pathogenesis of this condition was first broached by Skoda in 1855. The narrowing is made dependent upon the presence in the walls of the aorta of embryonal connective tissue connected with the ductus arteriosus; the postnatal involutional changes then lead to varying degrees of stenosis of the lumen of the aorta. Aberrant vestiges of duct tissue may occur in the aortic walls. Thrombosis of the aorta beginning in the ductus arteriosus may end in complete aortic obliteration (Rauchfuss).

Of 113 cases of narrowing and closure of the aorta at or near the opening of the ductus arteriosus studied by Wadstein, atresia was present in 20. In 16.4 per cent. the maximum narrowing was above the duct, in 33.8 per cent. on a level with the duct, and in 44.8 per cent. below it. The narrowing is sometimes double. In about a third of the cases the aorta was sclerotic, and generally more so on the proximal side of the narrowing. The most important secondary changes are hypertrophy of the left ventricle and the formation of collateral circulation, the blood reaching the lower portion of the body through the intercostals and the abdominal aorta, through the superficial epigastric and long thoracic arteries and the deep epigastric, and through the anastomoses of the intercostals with the ilio-lumbar and circumflex iliac arteries.

The ductus arteriosus is as a rule completely obliterated within from one to two weeks post partum. The obliteration is accomplished by contraction and endarterial proliferation. The resulting scar in the wall of the aorta often becomes calcified. Incomplete involution of the ductus arteriosus may become the occasion for thrombosis; the thrombus may project into the aorta and into the pulmonary artery and give rise to embolism (Klob).

Transposition of the *large veins* occurs in connection with cardiac abnormities. There may be two ascending or two descending venæ cavæ, in the first case due to failure of union of the canals of Cuvier; less than or more than four pulmonary veins, which may be inserted into the superior vena cava, into the left innominate vein, or into the ventricles. Either of the venæ cavæ may be absent. An apparent reduplication of the inferior vena cava results from the union of the hepatic veins in one stem, which may empty into the vena cava above or below the diaphragm, or directly into the right auricle. The union of the iliac veins may take place higher than usual, the right and the left iliac vein continuing upward on each side of the aorta, sometimes as far up as the liver.

Coronary Arteries.—Abnormities of the coronary arteries have been described by Hepburn, Turner, Brooks, and others. The most frequent deviation from the normal is an increase in number and irregularities in branching. Brooks describes the origin of a coronary artery in the right anterior sinus of Valsalva of the pulmonary artery; this abnormal coronary anastomosed with the right coronary and acted as a vein owing to the greater pressure in the aorta. In another, more complicated case, Brooks found an artery originating in the beginning of the pulmonary artery, anastomosing with the aortic coronaries and the left subclavian artery; it entered into intimate relations with a cirsoid mass at the base of the pulmonary artery. This vessel also acted as a vein, emptying its blood into the pulmonary artery.

Aplasia or hypoplasia of an organ or part of the body is associated with absent or diminished development of the corresponding blood-vessels. Abnormal origin and course of arteries and veins are of greater surgical than clinical interest, because it is exceedingly rare to observe that such abnormities cause symptoms. The origin of the right subclavian artery to the left of the left subclavian artery and its coursing in front of or behind the œsophagus as it passes over to the right side may cause difficulties in swallowing, according to Eppinger. Differences in the size of the radial arteries and in the relative depths of their situation frequently give rise to apparent differences in the pulse on the strength of which the existence of aortic aneurism may be erroneously diagnosticated.

Vascular Hypoplasia.—Congenital smallness of the entire cardio-vascular system is an interesting condition. Morgagni, Meckel, Rokitansky, and Virchow have drawn attention to this arrest of development, especially in chlorosis. The walls of the aorta are thin, the intima is the seat of wavy lines, and the lumen scarcely admits the little finger at a period when the aorta usually is twice or more as wide. The condition is frequently associated with arrested development of the reproductive organs and of the sexual characteristics of the body. Apparently the anomaly is probably more frequent in the female than in the male sex. It has been thought that a hypoplasia of the entire mesoblast is present, including the blood-forming organs; hence the peculiar anæmia which is thus explained according to Virchow's theory. The explanation is no longer regarded as satisfactory. "That a disorder so common and for the most part so curable should depend upon a malformation so grave and so incurable as this aortic and general vascular hypoplasia is on the face of it highly improbable" (Clifford Allbutt).

The developmental defects in the vessels in hæmophilia are considered under the latter heading.

Retrogressive Changes in Blood-Vessels.—Retrogressive changes of blood-vessels pure and simple are not of so much importance as when associated with other processes in the condition known as angiosclerosis.

Atrophy.—In chronic anæmia and marasmus simple atrophy takes place in the walls of the blood-vessels, especially in the muscular coat. Atrophy of individual organs is followed by or associated with atrophy of the corresponding vessels. When an extremity is removed the arteries and the veins that nourished the part decrease in size by an atrophy that may be called adaptive; the

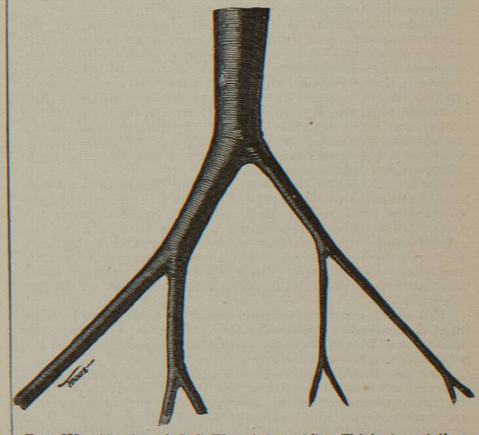

Fig. 626.—Atrophy of Left Iliac Artery After Thigh Amputation. (Two-fifths natural size.)

vessels become less and less until they carry no more blood than necessary for the nutrition of the remaining tissues (Fig. 626). In the beginning this adaptation is favored by contraction of the arteries and a little later by the development of new connective tissue in the intima, so that the lumen of the arteries is reduced in size.

Fatty Changes.—Changes in the composition of the blood or disturbances of circulation may induce fatty changes in the walls of otherwise healthy blood-vessels, especially in the epithelial cells and the muscle cells of the arteries. Fatty changes are of common occurrence in arteriosclerosis. A vessel the seat of fatty change presents upon its surface opaque white or yellowish-white patches and irregular lines, running longitudinally or forming a network, sometimes striated, sometimes of a uniform color. The surface is no longer smooth but velvety. These appearances are due to fatty granules and drops of fat in and between the epithelial cells. The change occurs also in the endothelial cells of capillaries. Cells may accumulate around and in areas of fatty change, taking up the oily detritus, and proliferation of cells also occurs.

Disintegration of the degenerated areas results in small defects ("fatty ulcers") which may become the starting point of thrombosis or traumatic and dissecting aneurisms; on supervention of trauma or severe strain the deeper layers of the intima are ruptured and the blood current makes its way between the layers of the arterial wall. According to Paget fatty degeneration of the media is responsible for many instances of cerebral apoplexy. Fatty changes in the media are common in various intoxications. Fatty changes are frequently followed by calcareous infiltration, the arterial wall becoming rigid and inelastic.

Fatty changes occur most commonly in the aorta and the pulmonary artery, and then in the arterioles and capillaries, especially of the brain.

In veins fatty changes in the intima form white spots, but the veins are not as frequently affected as the arteries.

Amyloid Degeneration.—The vascular system is especially prone to amyloid change, which takes its beginning in the media of the arterioles of parenchymatous organs. It also develops in the intima of the larger arteries in extensive widespread amyloid change, appearing as fine striæ and points, which are difficult to recognize with the naked eye unless first subjected to the action of Lugol's solution (see *Amyloid Degeneration*).

Hyaline Degeneration.—Hyaline degeneration of the smallest arteries and veins and of capillaries is frequently observed in the spleen, the lymph glands, the renal glomeruli, and elsewhere. The epithelium, at first intact, is finally destroyed and the lumen narrowed and occluded. In the larger vessels hyaline degeneration appears first in the intima, and it forms an early step in the complicated degeneration of arteriosclerosis. Hyaline material tends to become calcified. A good example of a hyaline degeneration of the muscular coat is seen in the pial arteries in tuberculous leptomeningitis.

Calcareous Infiltration.—Calcareous particles in glistening granules and as compact masses are frequently deposited in hyaline and fatty areas; it probably does not occur as an isolated primary process, and is most frequent and most extensive in senile arteriosclerosis. Gazert found that in a normal aorta the amount of earthy material is .43 per cent.; in a sclerotic and calcified aorta the calcareous material equalled 8.79 per cent. of the dry residue.

Metastatic calcification as a result of resorption in bone is said to involve especially the abdominal vessels, large calcareous plates forming which are separable from the surrounding tissues.

In some instances *true bone* has formed in the walls of arteries the seat of petrification. Ossification means preliminary vascularization, absorption of pre-existing calcareous material, and the formation of genuine bone. Von Schrötter and Falk describe with illustrations extensive ossification of arteries in senile gangrene; Cohn found true bone in the aortic valves and in the media of peripheral vessels.

Desquamation of the Epithelium.—In microscopic sections the epithelium of the vessels, both arteries and veins, is found occasionally to have separated from the intima, the peculiar epithelial cells lying curled up in the lumen. This condition is not constant, and the peculiar circumstances under which it occurs have not been established. The loosened epithelial cells have been mistaken for sarcomatous emboli.

Hypertrophy and Hyperplasia.—Hypertrophy and hyperplasia of arteries are observed in connection with various forms of tissue overgrowth, *e.g.*, the uterus in pregnancy, large tumors, and the adaptive hypertrophy of one kidney in case of extensive disease or absence of the fellow kidney. Obstruction to the blood current and increased arterial tension, as in cases of hypertrophy of the heart and of chronic nephritis, produce more or less hypertrophy of the muscular coat of the arteries. The power of the arteries and veins to adapt themselves to new conditions is best shown by the changes that take place in the development of collateral circulation. Indeed, this is one of the richest fields for the study of pathological adaptation. Vessels and vascular systems adjust themselves with wonderful precision to changes in pressure, in velocity, and in quantity of circulating blood. Readjustment is accomplished by virtue of the vaso-motor nervous mechanism and the physical properties of the arterial walls, aided under special conditions by structural changes. The physiological prototype of collateral circulation is seen in the changes that occur in the fetal circulation after birth. The ease with which the important rearrangements to the conditions of extra-uterine life are accomplished indicates that the mechanisms for adaptive changes on the part of the circulatory system are good; as in almost all pathological adaptations, the mechanisms are in better working order in the young than in the adult and the old.

The extent of the changes incidental to the development of collateral circulation will depend naturally upon the location of the obstruction and upon the size and the number of the collateral branches. Formerly it was taught that the increased pressure above the obstruction was the cause of the development of collateral circulation; increased pressure is now regarded as a factor of little consequence. Nothnagel has shown that there is really no rise of pressure in the vessels above the obstruction or ligature unless they stand in communication with branches below the obstruction. As advanced by von Recklinghausen, the increase in the extent of the capillary bed of the collateral branches, through which the blood flows into the capillaries of the obstructed vessel, results in increased rapidity of the current; and Thoma has demonstrated that increased rapidity of the current is followed by a widening of the lumen of the vessels, increased thickness of the wall, and growth in length. In this way the need of the tissues for blood is satisfied. The structural changes that take place in the walls of the collaterals may be regarded as the expression of a work-hypertrophy. And conversely, the gradual thickening of the intima of the occluded vessel as far back as the nearest collaterals is to be regarded as the result of diminished need of work.

In obstruction to the portal circulation by cirrhosis of the liver the dilatation and hypertrophy of the veins through which collateral circulation is gradually established may become marked. In exceptional cases the œsophageal veins become enlarged to many times their normal size and afford so complete escape for the portal blood that the congestions and the ascites of cirrhosis fail to appear and the clinical picture of the disease is masked.

Inflammation (Angeiitis, Vasculitis).—Inflammation may affect principally the adventitia, the media, or the intima (periarteritis and periphlebitis, mesarteritis and mesophlebitis, endarteritis and endophlebitis). On account of the structure of the venous wall, these distinctions are not as marked in veins as in arteries. Angeiitis may assume various types, such as productive, suppurative, tuberculous, and syphilitic. Angeiitis may result from the extension of inflammatory processes in the neighborhood (consecutive angeiitis); it may be caused by wounds of arteries or veins and by other forms of trauma, such as tearing and crushing (traumatic arteritis and phlebitis); it develops as a result of infections (pyogenic, syphilitic, and tuberculous) and of intoxications; in angeiosclerosis arteritis and phlebitis are observed as consecutive processes, secondary to nutritive and mechanical disturbances in the vessel wall. Arteritis and phlebitis sustain a double relation to thrombosis inasmuch as thrombosis in an artery or a vein induces inflammation (thrombo-arteritis, thrombo-phlebitis) while angeiitis not uncommonly leads to thrombosis. The causes of infectious or toxic angeiitis may reach the part of the vascular wall first involved through either the vasa vasorum, the lymph stream, or the main blood stream within the lumen of the vessel, or they may come directly from without.

It was the custom of pathologists in the early part of this century to look upon imbibition and reddening of the tunica intima as evidences of acute inflammation; in 1847 Virchow proved the incorrectness of this view.

Because the same agent may produce more than one form of arteritis and phlebitis, and because the same form of angeiitis may result from more than one cause, it becomes rather difficult to make at once a comprehensive yet simple classification of arteritis and phlebitis. When we omit from consideration periarteritis nodosa and the specifically tuberculous and syphilitic varieties, then the remaining forms belong to one of two large groups, the suppurative and destructive or the productive, the first characterized by disintegration and the formation of pus, the second by the production of new tissue.

Suppurative Angeiitis; Suppurative Thrombo-Arteritis.—The form of disease of the arterial wall induced by thrombosis or by the lodgment of emboli is determined by the nature of the thrombus or embolus. In the case that pyogenic bacteria are present in their interior, purulent inflammation is set up in the walls of the artery, resulting in a more or less extensive and destructive panarteritis and periarteritis, the final outcome being an abscess. This is what occurs in the development of metastatic embolic abscesses in the lungs. At other times the destructive action of the bacteria in infected emboli may be more limited in its extent, thereby producing a local weakening of the arterial wall, at which point aneurismal bulging occurs and there results a so-called embolic or mycotic aneurism (Goodhart, Eppinger). Mycotic embolism, the result of embolic suppurative arteritis, is observed most frequently in the cerebral arteries. Purulent thrombo-arteritis, with or without mycotic aneurism, is liable to result in hemorrhage from the weakening and destruction of the vessel wall.

Suppurative Thrombo-Phlebitis.—This may result from the extension of a suppurative process about a vein to the adventitia, media, and intima, which is more frequently the case than thrombosis upon the basis of a primary mycotic endophlebitis. In either case the resulting thrombosis undergoes purulent or purulent and putrid softening under the influence of the microbes that gain entrance into it from the walls of the vein. Beginning with John Hunter, in 1793, who described inflammation of the inner walls of veins after blood-letting and in the uterine and femoral veins of women in the puerperal period, it gradually became established clearly that pyæmia commonly depends upon suppurative thrombophlebitis, the circulating blood being the medium of transference of infected particles from the thrombus to various parts of the body, the emboli on lodgment producing metastatic abscesses. Among veins that are liable to suppurative thrombo-phlebitis may be mentioned the umbilical veins in the new-born, infection taking place at the navel; the lateral and other sinuses of the dura mater in suppurative mastoiditis and middle-ear disease; the mesenteric veins in appendicitis and ulcerative processes in the intestines, the process giving rise by extension or embolism to suppurative pylephlebitis and abscesses of the liver; the subcutaneous veins in the vicinity of the foci of infection, etc. Suppuration originating in a diverticulum of the œsophagus has extended to the superior vena cava and given rise to suppurative thrombo-phlebitis in this vein.

Mycotic and Toxic Endangeiitis.—Acute verrucose and ulcerative endangeiitis, similar to acute endocarditis, most frequently occurs in the aorta and the pulmonary artery and is usually associated with infective endocarditis of the semilunar valves. It has also been described in the larger and medium-sized branches of the aorta and the pulmonary artery. The lesions usually assume the form of warty eminences, composed of a cellular and vascular granulation tissue and capped by thrombotic deposits. There is more or less cellular infiltration into the tissues of the vessel wall near such warty outgrowths, and at times the process assumes more of an ulcerative and destructive type. Osler has described an interesting instance of multiple mycotic aneurisms of the aorta the result of infective endaortitis associated with acute infective endocarditis. Schmey has recorded the sudden development of aneurisms, one upon the radial artery and one upon the posterior tibial, in a boy of twelve with acute articular rheumatism, showing that in all probability the microbe or toxin of this disease may also cause this rare form of endarteritis. Such aneurisms, although not embolic, belong etiologically in the same general category as the embolic-mycotic aneurisms just referred to as caused by embolic destructive arteritis. Oliver demonstrated B. anthracis in an ulcerative aortitis.

It is now recognized that the acute and chronic forms of arteritis occur in various infectious diseases—typhoid fever, smallpox, scarlet fever, measles, acute articular rheumatism, influenza, pneumonia, syphilis, tuberculosis, and leprosy. In the acute forms cellular infiltrations, generally circumscribed, are found in the outer coats of the vessels together with more or less extensive proliferation of the subepithelial connective tissue of the intima. Nodular and more diffuse accumulations of lymphoid and epithelioid cells occur beneath the epithelium. These changes are explained as due to the circulation in the blood of the microbes and the toxins of the diseases mentioned. When the endarterial changes result in destruction of the living epithelium thrombosis takes place, and if it is in a larger vessel a typical verrucose endarteritis may be established. The endarteritic lesions are due to a direct implantation of micro-organisms upon the intima in the same way as infections of the endocardium take place; inasmuch as bacteria are often absent, it seems that endarteritis may be caused by toxins also. These changes occur probably more frequently in the veins than in the arteries. Arterial thrombosis in the diseases mentioned is often referable to infectious arteritis.

It is quite evident that there might be more or less difficulty in distinguishing between toxic and infectious verrucose endarteritis and thrombo-arteritis pure and simple. The formation of a parietal thrombus upon a rough spot or thickening of the intima might induce small nodular outgrowths of new tissue. The presence of microbes in the thrombus would indicate that the acute and destructive changes in the walls of the vessels were primary.

In many instances of multiple venous thrombosis it is not unlikely that an infectious or toxic endophlebitis is the primary change in the vessel wall. In typhoid fever, diphtheria, variola, influenza, and other infectious diseases there is found a nodular, sometimes a more diffuse, accumulation of lymphoid and epithelioid cells in the intima, even more frequently than in the arteries. Many of the large cells in such foci seem to have marked phagocytic properties (Mallory). The accumulated cells, as well as the epithelium, may undergo necrosis and become the starting-point of a thrombus. Inasmuch as bacteria are often absent, it would seem that the vascular lesion may be caused by toxins—a toxic endangeitis.

Lancereaux and others believe that there is a malarial aortitis which occurs in the form of gelatinous plaques, situated generally in the ascending part of the arch of the aorta; the lesion is described as beginning in the adventitia with cellular infiltrations which result in atrophy of the media and an adaptative thickening of the intima. Saccular aneurisms may result. It seems that there