Solids.	Grains.
Magnesium carbonate	3.17
Magnesium sulphate	7.80
Calcium carbonate	5.32
Calcium sulphate	6.47
Iron peroxide	Trace.
Silica	.92
Alumina	.85
Organic matter	Trace.
Total solids	83.82

Gases.	cu. in.
Free carbonic acid gas	9.20
Free sulphureted hydrogen	1.60

The water has valuable antacid and aperient properties. It belongs to the alkaline-carbonated class. The other springs at Paso Robles are not extensively used as yet, although some of them will no doubt be found of value.

James K. Crook.

EMBALMING. See *Dead Bodies, Disposal of.*

EMBELIA.—The fruit of *Embelia ribes* Burm. (fam. *Myrsinaceæ*). The fruits of this Indian shrub are nearly as large as black-pepper fruits, globular, slightly beaked, dull red and striate, becoming brown on keeping. They are said to be used to adulterate pepper. The drug is largely used in its home as a stomachic, carminative, stimulant, and anthelmintic, and also as a parasiticidal application to ringworm and similar affections. It appears to be specially useful as a tænicide. Although it is said usually to kill the worm, a purgative should be taken both before and after the administration of the remedy. Its important constituent is *embelic acid*, which is found to possess the same properties as the drug itself. Embelic acid, the formula for which is $C_9H_{14}O_2$, occurs in orange-yellow scales, soluble in alcohol, odorless and tasteless. It is given in doses of 0.2 to 0.4 gm. (gr. iij.–vi.), to be both preceded and followed by castor oil. The fruit contains also resin, fat, tannin, and a small amount of an alkaloid, which has been called *christembine*. The powdered fruit is given in doses of from 4 to 16 gm. (℥ i.–iv.) or the fluid extract in corresponding amount.

Henry H. Rusby.

EMBOLISM.—That form of metastasis in which insoluble substances are transported by the blood current and lodged in some part of the vascular system is known as embolism. The material transported is an embolus. The process may occur in lymph vessels (lymphogenous embolism), as well as in blood-vessels (hæmatogenous embolism). The latter is, however, the more common and important form, and embolism is usually understood to mean this when no special modifying designation is used.

The substances which may enter the vascular system and be carried in the blood stream as emboli are of the most varied character. They may be gaseous, liquid, or solid. The most common emboli are those composed of the products of coagulation of the blood, and unless specially designated an embolus is usually understood to be derived from a thrombus. Tumor cells, living or dead tissue elements, animal or vegetable parasites, parenchymatous cells, placental cells, fat, pigment, masses of calcification, air, and various extrinsic substances may also gain entrance into the blood stream and form emboli.

The lodgment and impaction of the embolus in the vessel is due to the fact that on account of either its size or its physical properties the material transported is caught upon an obstruction in the vessel or cannot pass its lumen, and so remains either partially or wholly blocking it. Metastasis in the direction of the normal current is known as direct embolism, in the opposite direction as retrograde. The transportation of emboli from the veins into the systemic arteries without passing through the pulmonary circulation is designated crossed or paradoxical embolism. This may occur in case of a patent foramen ovale or a persistent ductus Botalli.

The importance of the embolism depends upon the nature of the embolus, its size, and the anatomical location and relation of the obstructed vessel. Emboli containing tumor cells or infective agents, as bacteria, are the most injurious (septic emboli); those consisting of dead tissues or substances incapable of growth act only mechanically in obstructing the circulation (bland or aseptic emboli). Hard or sharp-pointed substances may injure the vessel wall and lead to the formation of an aneurism. Toxic or infective emboli may weaken the wall of the vessel and also produce aneurisms. Large emboli may seriously interfere with the nutrition of the part supplied by the obstructed vessel. Embolism of small arterioles or capillaries may produce a very slight disturbance of nutrition or none at all. If the embolus does not completely occlude the vessel no serious circulatory disturbance may be caused, but the complete closure of the vessel usually follows from the formation of an induced or secondary thrombus. If the obstructed vessel has abundant collateral anastomoses beyond the point of obstruction no disturbance of nutrition of any consequence may occur; but if the obstructed vessel is a terminal artery poorly provided with collaterals, degeneration or necrosis of the part supplied by it will result from an insufficient supply of nutrition (anæmic and hemorrhagic infarction).

Direct Embolism.—Embolism in the direction of the normal blood current is of most frequent occurrence. The substance forming the embolus is carried along by the blood stream until it is caught by some obstruction or swept into a channel so narrow as to prevent its farther passage. It follows, therefore, that direct embolism can occur only in the pulmonary and systemic arteries, heart, and portal vein. Emboli found in the right side of the heart or pulmonary arteries have their origin in the systemic veins; those found in the systemic arteries arise in the pulmonary veins or left heart. Portal vein emboli take their origin from thrombi either in the main trunk or in its contributing branches. The course and site of lodgment of the embolus is determined by the size, form, weight, and character of the material composing it, as well as by the force and volume of the blood stream, the size of the blood-vessels, the angle at which they are given off, the position of the body, etc. Consequently emboli arising in the systemic veins or right heart are more frequently carried into the pulmonary arteries supplying the lower lobes of the lungs, and more frequently into those of the right lower lobe than into those of the left. Emboli from the left heart more frequently pass into the thoracic aorta than into the carotid or subclavian arteries. The left carotid arising from the highest point of the aortic arch is more likely to receive emboli than the right carotid. Likewise the left common iliac artery, being more directly in the line of the current in the abdominal aorta, receives emboli more often than the right iliac. Since the conditions in the systemic veins are much more favorable for thrombosis, embolism of the pulmonary arteries is much more common than of the systemic arteries. Of the latter the small branches in the lower extremities are most likely to receive emboli, but in the great majority of cases this event is not diagnosed clinically, inasmuch as the symptoms produced are of much less importance than those caused by embolism of the renal arteries, splenic, etc.

If the embolus completely closes the lumen of the obstructed vessel, it is styled an obturating embolus. Spherical emboli are most likely to do this, but an elongated portion of a thrombus may be folded several times upon itself and pushed into a vessel several times its own diameter, completely obturating the lumen. This happens very often in the case of long pieces of thrombi washed out of the femoral veins and in this manner blocking up the largest branches of the pulmonary artery. Similarly, irregular thrombi of soft consistence may be pushed into a vessel so as completely to fill it. If the embolus is hard and firm there may still be some space left for the blood to flow, but this is usually soon closed by secondary thrombosis. Cylindrical, elongated, flat or irregular emboli may be caught at an arterial bifurcation and be pushed partly into both branches, either wholly or partly blocking them (straddling or riding embolus).

Large soft thrombi may break up and the resulting emboli may fill a large number of small branches. Emboli arising in the systemic veins small enough to pass the pulmonary capillaries may lodge in the cerebral or renal capillaries.

Retrograde Embolism.—A retrograde transportation and impaction of an embolus is more likely to occur in the lymph stream than in the blood, and plays a very important part in the lymphogenous metastasis of tumors. Under especial conditions retrograde embolism may occur also in the veins, usually in the large veins near the heart. The cause of the retrograde transportation may lie either in a reflux flow from the heart and great veins into the hepatic veins or the veins of the neck, head, or arms; or in case of extreme venous stasis an embolus may be gradually pushed backward in the vein by abnormal venous pulse waves proceeding from the heart. This event is most likely to occur in dilatation of the right heart with tricuspid insufficiency. Any condition which increases the pressure in the veins near the heart favors also a retrograde embolism—decreased negative pressure in the thorax, disturbances of respiration, prolonged and difficult expiration, cardiac insufficiency, etc. If the venous valves remain adequate the embolus may be caught on them, but in the extreme distention of the vessels they become insufficient and allow the embolus to pass. Retrograde metastasis has been observed in the hepatic, cerebral, mesenteric, renal, pulmonary, axillary, and coronary veins, pampiniform plexus, and dural sinuses. It has been most frequently observed in the case of tumor metastasis, but retrograde transportation of liver cells and infective emboli has also been reported. In the case of non-infective thrombotic material it would be very difficult to establish a diagnosis of retrograde embolism.

Paradoxical Embolism.—The passage of emboli through an open foramen ovale or persistent ductus Botalli without passing through the pulmonary circulation is known as paradoxical or crossed embolism. An opening in the foramen ovale in the shape of a narrow oblique slit is found in a relatively large number of autopsies. Under conditions of disturbed pulmonary circulation in which the pressure in the right auricle becomes greater than that in the left, the slit in the foramen is widened and its membranous edges are bulged into the left auricle. The opening may be so enlarged that not only small emboli may pass through it, but thrombi arising in the right side of the heart may extend through it into the left auricle. Masses of tumor cells and liver cells, the latter in cases of traumatic laceration of that organ, have been found obstructing the opening. Aside from such autopsic evidence the diagnosis of crossed embolism is impossible. The metastasis of certain large-celled tumors is best explained by the assumption of paradoxical embolism, but the possibility of even large tumor cells passing through the pulmonary capillaries without setting up lung secondaries must be borne in mind.

Varieties of Emboli.—*Bland Emboli.*—These are composed for the greater part of thrombotic material which does not contain toxic or infective agents; but no sharp line can be drawn between them and septic emboli, inasmuch as emboli may contain pathogenetic organisms and yet not give rise to septic processes, because of the action of the protective forces of the body inhibiting their growth. The practical criterion is not the absence or presence of micro-organisms in the embolus but the effect of the latter upon the vessel wall at the point of lodgment. A bland embolus is accordingly one whose effects are purely mechanical. Besides thrombotic material, pigment granules, dead cellular elements, fat, masses of calcification, extrinsic substances such as carbon or metal dust, etc., may produce only mechanical effects.

Infective Emboli.—Because of their frequent occurrence and the serious conditions resulting from them infective emboli containing pathogenetic micro-organisms are of very great pathological importance. In addition to the purely mechanical effects produced by them they show at the point of lodgment the characteristic effects of the bacteria which they contain. These effects are chemical in nature, the result of the specific products of the organism, and are characterized by degeneration or necrosis of the vessel wall, hemorrhages, inflammation, suppuration, gangrene, etc. As a result of the weakening of the wall through the injury or destruction of its inner coat an aneurism may be formed (aneurysma mycotico-embolicum). The metastasis of emboli containing pyogenetic organisms gives rise to the condition known as pyæmia. The primary focus of the embolism is usually an infective venous thrombus formed at the point of entrance of the bacteria, which may be in any part of the body. From this thrombus emboli may arise and convey the bacteria to other parts of the vascular system, where having lodged they may multiply and produce poisons which exert their characteristic effects upon the vessel wall and surrounding tissues. From these secondary foci other emboli may be given off and the process indefinitely repeated.

There may be no evident primary focus and the secondary lesions may form the only features of the disease; or, as in the case of malignant endocarditis, infective thrombi may form upon the valves of the heart and give origin to infective emboli, though the primary entrance of the bacteria into the vascular system cannot be discovered (cryptogenic infection). Embolism of bacteria alone from the primary focus, either singly or in masses, may occur without the formation of an infective thrombus at this point. These may become attached to the vessel wall, multiply, and set up their characteristic changes. In this manner bacteria arising in infective thrombi in the systemic veins may pass the lung capillaries and produce their effects in the systemic arteries or capillaries. As mentioned above, not all emboli containing pathogenetic bacteria give rise to secondary lesions at the point of lodgment. This may be explained by an increase in the vital resistance of the body or a diminished virulence of the bacteria due to conditions arising either in them or in the body.

Tumor-cell Emboli.—Very rarely benign tumors may extend into blood-vessels and give rise to emboli which may develop into secondary growths of the same nature as the primary. In the metastasis of malignant tumors embolism plays a much more important rôle, as it is the chief factor of their spread through the body. Both carcinoma and sarcoma very frequently break into blood-vessels, loosened tumor cells are carried away in the blood stream, and from the emboli thus produced secondary growths arise. It may be emphasized here that this embolism is always one of tumor cells and not of parasites which may cause the formation of such cells. The structure of sarcoma particularly favors hæmatogenous metastasis, inasmuch as the cells of a sarcoma not infrequently form the wall of the blood-vessels of the tumor, or are separated from the blood stream by an endothelial layer only. On the other hand, the spread of carcinoma into the lymph spaces favors lymphogenous metastasis, and in general it may be said that sarcoma and carcinoma are characterized respectively by hæmatogenous and by lymphogenous metastasis. Large masses of tumor cells coming from the systemic veins may block the orifices of the right heart or the pulmonary artery and cause sudden death. As a rule, however, the mechanical effects of emboli composed of tumor cells are unimportant when compared to the significance of the tumor metastasis. More frequently the secondary growths of malignant tumors arise from capillary emboli rather than from those of large size. Tumor cells of small size may pass the pulmonary capillaries without lodgment and give rise to secondary growths in the systemic capillaries. Both the retrograde and the paradoxical varieties of embolism of tumor cells have been observed.

Emboli of Animal Parasites.—The Filaria sanguinis, Bilharzia hæmatobia, and the Plasmodium malariæ may be found in the blood of the human body, and under certain conditions may block the capillaries or smaller arterioles. Echinococcus cysts in the heart wall may

rupture into the blood stream and give rise to general metastasis. The embryos of trichina pass from the intestine to the muscles through the lymph and blood, and the amœbæ found in the intestine may reach the liver by way of the portal vein.

Fat Embolism.—This is probably a very common form of embolism, but in the majority of cases it is of little pathological significance. Occasionally it is fatal, in conditions when so much fat is set free into the circulation as to block up many capillaries and small arteries; or a moderate fat embolism may aid in causing death in cases of shock, cachexia, anæmia, etc. The source of the fat is usually the fatty bone marrow, but fat may enter the circulation as the result of injury to any tissue or organ of the body which contains fat. Amputations, fractures, crushing, etc., of bones containing fatty marrow are the most common causes, but even slight concussion of the bones may set free some fat into the blood-vessels. Congestion or inflammation of the marrow may also lead to fat embolism. Injury to adipose tissue in breast amputations, in laparotomy, during child-birth, in laceration or necrosis of fatty liver, in destruction of brain tissue, in fatty degeneration of sclerotic plaques, etc., may also cause the entrance of fat into the circulation. Fatty embolism has also been observed in the lipæmia of diabetes, and has been supposed to be the cause of the dyspnœa and coma in this disease.

The fat droplets floating in the blood stream collect for the greater part in the lung capillaries, and in severe cases the majority of these vessels may be found greatly distended and filled with plugs of oil. The capillaries of the brain, kidneys, and heart suffer next in proportion. Layers of fat may occasionally be present in the post-mortem clots found in the heart and great veins. The results of fatty embolism are purely mechanical. In a moderate degree the fat may be disposed of by saponification, emulsion, phagocytosis, etc.

Cellular Embolism.—Pulmonary embolism of the giant cells of the bone marrow is of very frequent occurrence, being found in many toxic and infective conditions, pernicious anæmia, splenic anæmia, leukæmia, congestion and inflammation of the bone marrow, injury to the bones, etc. It occurs to the greatest extent in puerperal eclampsia, but is also commonly found in the lungs of women dying in the puerperium of diseases other than eclampsia. The significance of the process is not yet known; within a limited degree it may be physiological, or it may occur just before death as a pre-agonal phenomenon. In the acute infections the existing hyperæmia of the bone marrow may be the underlying cause of the metastasis. Injury to the bones may cause a metastasis not only of the fat and giant cells of the marrow but also of portions of marrow tissue and bony fragments. The giant cells appear in the lung capillaries as deeply staining masses of chromatin having a very irregular knobbed shape, rarely multinuclear. As a rule the cells are crowded so tightly into the capillaries that no cell protoplasm can be made out, but in the cells found in the small arterioles the protoplasm is usually intact. In puerperal eclampsia the number of giant-cell emboli may be as great as from six to eight in every low-power field. This variety of embolism is also found in association with other forms of cellular embolism—*e.g.*, that of liver cells, placental cells, etc. Giant cells resembling those of the marrow may originate in the hæmolymph glands in fatal cases of anæmia, and it is possible that these may also give rise to pulmonary embolism.

Giant cells arising from the syncytium of the chorionic villi have also been found in the pulmonary capillaries in cases of puerperal eclampsia, hydatid mole, etc. According to Schmorl, this form of embolism plays an important rôle in the etiology of eclampsia; but this is probably not the case, as it is not constantly found in this disease and may be present in the lungs of puerperal women not suffering from eclampsic symptoms. Further, since the differential diagnosis of multinuclear bone-marrow cells and syncytial cells is practically impossible, it is probable that many of the emboli found in the pulmonary capillaries and thought to be syncytial cells were in reality bone-marrow cells. In case of hydatid moles portions of syncytial buds or villi may break into the uterine sinuses and be carried to the lungs, but this process must be regarded as being of the nature of a tumor metastasis.

After traumatic laceration of the liver, large portions of liver tissue have been found in the pulmonary arteries. Small pulmonary emboli of liver cells may occur after hemorrhage and necrosis of the liver. Paradoxical embolism of liver cells has also been observed, and portions of liver tissue have been found blocking the foramen ovale. In connection with liver-cell embolism pulmonary emboli of bone-marrow giant cells are usually present.

Splenic cells, sometimes containing malarial parasites and pigment, red blood cells, or blood pigment, may be found under certain conditions in the liver capillaries. Portions of endothelium, bits of the heart valves, etc., may also be loosened into the blood stream and form emboli in the capillaries of various organs: brain, spleen, kidneys, etc.

Air Embolism.—Air may enter the circulation through wounds or surgical operations about the neck, upper part of thorax, shoulders and head, in which conditions the air is sucked into open veins by means of respiratory aspiration. Air may also enter the circulation through the uterine veins after separation of the placenta in abortion, placenta prævia, uterine injections, etc.; or open veins in ulcers of the stomach and intestines may be the point of entrance. Gaseous embolism may be produced by infection with gas-producing bacilli. The cause of death in caisson disease has been thought to be emboli of nitrogen which has been absorbed by the blood under high atmospheric pressure and suddenly released on return to normal atmospheric conditions. It is probable that many of the cases reported as air embolism, especially those in which the air has been supposed to enter the uterine or stomach veins, have been in reality cases of gaseous embolism due to infection with the Bacillus aërogenes capsulatus.

Effects of Embolism.—The metastasis of dead or inert substances incapable of growth produces chiefly mechanical effects, inasmuch as their chemical properties are not usually such as to cause further alterations in the vessel wall or its neighborhood. Of the purely mechanical effects the most important is the obstruction of an artery by an embolus. If the embolized vessel has abundant anastomoses, as is the case with the arteries of the voluntary muscles, skin, bone, uterus, thyroid, etc., there may be no appreciable disturbance of the circulation: but if the obstructed vessel has few anastomoses and a collateral circulation is not quickly established, the tissues supplied by the occluded artery will quickly degenerate or necrose as the result of the anæmia. The area of anæmic necrosis produced by the occlusion of an artery by either thrombosis or embolism is known as an infarct. Two varieties of infarcts are distinguished: anæmic or white, and the hemorrhagic or red infarct. The former occurs as the result of the occlusion of terminal arteries in the kidneys, spleen, heart, brain, and rarely in the liver. The hemorrhagic infarct occurs regularly as the result of the obstruction of the terminal branches of the pulmonary and superior mesenteric arteries. It differs from the anæmic only in that into the necrosed area there is an infiltration of blood. It is therefore a hemorrhagic-anæmic necrosis. The hemorrhage into the necrosed tissue takes placed by diapedesis from the injured walls of the vessels lying within or at the border of the infarcted area. These become partly filled with blood from collateral capillaries and arterioles, and the vessel walls, weakened by disturbance of nutrition from the lack of their accustomed blood supply, permit of hemorrhage by diapedesis. The low tissue-resistance in the lung and mesentery and submucosa of the intestines also favors hemorrhage. Surrounding the anæmic infarcts of the spleen, heart, kidneys, etc., there is always a narrow zone of congestion and hemorrhagic infarction which is visible in the fresh specimen as a red line surrounding

the gray or yellowish dead anæmic area. This may be explained as being due partly to collateral congestion and partly to hemorrhage from the small anastomosing vessels at the border of the infarct, whose walls also suffer disturbance of nutrition from the anæmia or stasis, and permit the escape of blood by diapedesis. In cases of extreme venous congestion this border-line of hemorrhage may be greatly increased in size and an anæmic infarct may for the greater part or wholly become hemorrhagic. Thrombosis in the veins also favors the production of hemorrhagic infarction. It is probable that under normal conditions of the circulation occlusion of a terminal branch of either the pulmonary or the mesenteric arteries will produce no infarction owing to the rich collateral anastomosis. Under conditions in which the circulation in these vessels is already impaired obstruction of their terminal branches will produce a hemorrhagic infarct. The fact that infarcts of the lung and intestines are invariably hemorrhagic has not yet been satisfactorily explained, but it is supposed that the rich anastomoses of the pulmonary and superior mesenteric arteries lead, through the establishment of a collateral flow, to the refilling of the vessels of the infarcted area, and that the walls of these vessels having been damaged by the anæmia permit of diapedesis.

Embolism of the coronary arteries, the valvular orifices, the pulmonary artery or its main branches, or the bulbar arteries may cause immediate death. Fat embolism of the cerebral, pulmonary, or renal capillaries may be so extensive as to cause death by obstruction of the circulation and disturbances of nutrition. Less severe cases may cause coma, dyspnœa, etc., while moderate degrees may produce no symptoms at all. Likewise air embolism in small amount may produce no effect. Larger amounts of air may collect in the right heart and pulmonary artery in a frothy mass which the heart is unable to force through the pulmonary capillaries, death resulting from paralysis of the heart. In caisson disease death may follow the embolism of bubbles of nitrogen which have been suddenly liberated in the blood by the too sudden reduction of the high atmospheric pressure under which it had been absorbed. The effects of embolism of cellular elements, animal parasites, etc., are purely mechanical. Pulmonary embolism of placental cells has been thought to be the cause of eclamptic convulsions. Tumor cells not only act mechanically but give rise to secondary growths as well. Sharp-pointed masses of calcification, etc., not only cause obstruction of the circulation but may so injure the vessel wall as to lead to the formation of an aneurism.

Toxic and infective emboli, in addition to their purely mechanical effects, produce changes in the vessel wall and its neighborhood due to the action of the poisons which they contain or which are produced by the bacteria contained within them. Degeneration, necrosis, and inflammation of the vessel at the point of obstruction may lead to a widespread purulent arteritis, formation of abscesses, etc., or as a result of the weakening of the wall aneurisms may be produced.

Sequelæ.—In the case of embolism of thrombotic material organization, simple and purulent softening, or calcification of the embolus may take place. The sequelæ of infarction are essentially the same as those of necrosis in general: organization, encapsulation, cyst formation, calcification, abscess, gangrene, etc. Emboli of liver cells quickly necrose, the dying cells forming centres of coagulation and thrombus formation. Bone marrow and placental giant cells undergo a slow necrobiosis, and may also give rise to thrombosis. Small bits of dead tissue, pigment granules, and bacteria may be removed by phagocytes. Fat in small amount is saponified or emulsified, or disposed of by phagocytosis. Small amounts of air may be absorbed.

Symptoms.—The general symptoms of non-infective embolism depend upon the anatomical position and relations of the occluded vessel, the specific function of the part supplied by it, and the degree of anæmia produced by the obstruction. The passage of the embolus through the vessels or heart causes no definite symptoms. Pain may follow the impaction of an embolus in an artery, particularly in the abdominal aorta, mesenteric, iliac, and femoral arteries. At times it is very severe, having the character of that of a painful blow. It is probably due to the irritation of sensory nerves in the vessel wall caused by the sudden impaction and distention. Pain may follow the embolism as a result of local anæmia, inflammation, and the various sequelæ of infarction. In rare cases a chill or fever may accompany the impaction of non-infective emboli. Fever, however, usually follows embolism as one of the accompanying phenomena of secondary inflammation, sepsis, gangrene, etc. Embolism of vessels supplying visible parts of the body may be manifested by signs of local anæmia, gangrene, etc. Infarction of the lungs, kidneys, and intestines may be shown by hemorrhage from these organs. Infarction of any organ will lead to disturbance of its function. In the case of infective emboli, besides the symptoms due to the mechanical obstruction of the circulation, those of local and general infection are present: chills, fever, etc. These are usually of such great importance as completely to cast into the background the symptoms of local anæmia or infarction.

The order of frequency of arterial embolism is given by Welch as follows: pulmonary, renal, splenic, cerebral, iliac and arteries of lower extremities, axillary and arteries of upper extremities, cœliac axis with hepatic and gastric branches, central artery of the retina, superior mesenteric, inferior mesenteric, abdominal aorta, and coronary arteries. This order is based not so much upon the actual occurrence of emboli as upon the symptoms of embolism. It is very probable that embolism of the small arteries of the lower extremities would come next to the pulmonary in the order of frequency, but the signs or symptoms attending this event may be so unimportant as to be overlooked.

Pulmonary Embolism.—Embolism of the main trunk or both of the main branches of the pulmonary artery is usually followed by sudden death from syncope. In other cases there may be a sudden attack of intense precordial pain and dyspnœa followed quickly by death. In less extensive pulmonary embolism the chief symptoms may be intense dyspnœa, cyanosis, great disturbance of heart action and respiration, sweating, chills, syncope, convulsions, coma, etc. Embolism of the smaller terminal branches may produce no symptoms, but very often hemorrhagic infarction is accompanied by chills, dyspnœa, fever, pain in the side, pulmonary hemorrhage, etc. Infective emboli give rise to pyæmic abscesses, purulent and fibrinous pneumonia, and the local and general symptoms arising from these conditions.

Renal Embolism.—Large infarcts of the kidney may cause pain; small ones are unaccompanied by painful sensations. Hæmaturia is the most important sign of renal infarction. Infective emboli cause pyæmic abscesses. These are manifested by severe constitutional symptoms of intoxication, pyuria, etc.

Splenic Embolism.—Infarction of the spleen may occur without signs or symptoms, but frequently there are sudden pain in the splenic region, chills and fever, splenic enlargement, perisplenitic friction, etc. Infective emboli may cause splenic abscess with symptoms of pyæmia.

Cerebral Embolism.—The left middle cerebral is most commonly involved. Sudden hemiplegia and aphasia are the chief symptoms. Embolism of the vertebral or basilar artery produces symptoms of acute bulbar paralysis.

Mesenteric Embolism.—Infarction of the intestines is usually manifested by sudden attacks of severe colicky pain, abdominal tenderness, bloody or fæcal vomiting, blood in the stools, gangrenous odor of stools, sweating, subnormal temperature, and collapse. Death usually occurs within a very short time. Very rarely the symptoms are less pronounced and of such vague character as to make diagnosis impossible. The condition may very much resemble that of intestinal obstruction. Embol-

ism of the smaller branches of the intestinal arteries gives rise to the formation of intestinal ulcers.

Aortic Embolism.—The occlusion of the thoracic aorta by embolism is of very rare occurrence. It may be caused by the dislodgment of large thrombi in the heart or in aortic aneurisms above the point of embolism. Death is instantaneous or occurs within a very short time. Embolism of the abdominal aorta is not of such rare occurrence. In the majority of cases the embolus arises from a heart thrombus. The symptoms may develop suddenly or the onset may be gradual. Pain in both legs, often extreme, is usually present; with this there is associated paralysis, absence of femoral pulsation, and ascending gangrene. Death may occur within a few hours or be delayed for months, in the latter case the result of slowly progressive gangrene or sepsis. Embolism of the iliacs, or of the femoral or popliteal arteries is manifested by similar symptoms varying in degree with the size of the vessel occluded and the location of the embolus.

Coronary Embolism.—Embolism of the larger branches of the coronary arteries causes sudden death, or an attack of extreme dyspnœa and severe precordial pain which may be followed shortly by death. Embolism of the smaller branches may cause anæmic infarction with symptoms of angina pectoris. As a result of the weakening of the heart wall by the infarction rupture of the heart into the pericardial sac may take place causing sudden death, or aneurismal dilatation of the heart may be produced, leading to serious impairment of cardiac efficiency.

Retinal Embolism.—Sudden blindness is produced by the embolic occlusion of the central artery of the retina. Ophthalmoscopic examination reveals a condition of retinal infarction usually without the occurrence of hemorrhage. In embolism of the smaller retinal branches multiple hemorrhages usually occur with more or less pronounced disturbance of vision.

Embolism of Spermatic Artery.—Occlusion of the spermatic artery by embolism is said to have caused gangrene of the testis in a small number of cases.

Diagnosis.—Since the chief symptoms of embolism are of the same nature as those of thrombosis, the differential diagnosis of these two conditions is of main importance. The sudden development and severity of symptoms of arterial anæmia are more characteristic of embolism than of thrombosis. Absolute dependence cannot, however, be placed upon this point, inasmuch as thrombi occasionally occlude with great rapidity, while on the other hand the symptoms of embolism may be slow of development owing to the fact that the embolus either does not entirely obturate the vessel which is later completely closed by secondary thrombosis, or the embolus may be so situated that the secondary thrombosis plays the chief part in the production of the anæmia. After arterial embolism the establishment of the collateral circulation usually leads to a more decided improvement of symptoms than is commonly the case after thrombosis. Absolute reliance cannot be placed upon this factor, since occasionally after thrombosis very marked amelioration follows the development of the collateral circulation. The occurrence of arterial obstruction in young individuals or in adults showing no signs of arteriosclerosis, especially in cases of valvular lesions of the left side of the heart, points very strongly to the occurrence of embolism. If arteriosclerosis is present, the differential diagnosis between thrombosis and embolism becomes much more difficult.

The establishment of a source for emboli in the existence of a previously formed thrombus, cardiac disease, aneurism, infected wound, etc., is the most reliable factor in the diagnosis of embolism. In pulmonary embolism the source may be sought in thrombi in the systemic veins or right heart. In embolism of the systemic arteries the chief source of the embolus is disease of the mitral or aortic valves. Thrombi in aortic aneurisms may also give rise to emboli. Infected wounds, abscesses, acute endocarditis, etc., form the sources of infective emboli. Abortion, separation of the placenta, uterine injections, pregnancy, etc., are conditions in which embolism may occur. Injury of the bones or fat-containing tissues gives rise to fatty embolism. Though cases may occur in which the source cannot be found, and though the existence of a thrombus in one vessel does not exclude the occurrence of thrombosis in others, it is possible, in the majority of cases, to make a correct diagnosis of embolism whenever definite symptoms are present.

Treatment.—Prophylactic treatment may be of the greatest importance in the prevention of the detachment of an embolus from a thrombus. In the case of venous thrombosis of the lower extremities absolute rest of the affected limb, or better of the body as a whole, should be insisted upon, as a very large percentage of the cases of death from pulmonary embolism occur as the result of the patient's movements in walking, bathing, or going to stool. Walking should not be allowed for at least six weeks after venous thrombosis of the lower extremities. Massage or palpation of the affected vein, or even pressure upon it with a stethoscope, as well as all unnecessary movement and manipulation of the limb, should be avoided. Prophylactic measures may be instituted further against the formation of thrombi by stimulation of the heart's action, improvement of the general nutrition, prevention of extension of infection by surgical interference, etc. In the case of acute endocarditis, heart thrombus, aortic aneurism, etc., absolute rest is also essential as a prophylactic measure against embolism.

After embolism has occurred the general indications are relief of pain, absolute rest, proper nourishment, cardiac stimulation, etc. The development of the collateral circulation should be brought about as soon as possible in order to avoid anæmic necrosis. In case of embolism of accessible portions of the body this may be aided by the local application of heat. When infarction has taken place the general indications of treatment are along lines tending to promote absorption and organization of the dead tissues. In the case of gangrene of the lower extremities and intestinal infarction operation according to general surgical principles may be performed.

Aldred Scott Warthin.

EMBRYOLOGY.—The general history of the embryo up to about the end of the second month is given under *Fœtus, Formation of*. The development of special organs is treated under those organs; *e.g.*, for the development of the brain see under *Brain*. There are also included in the Handbook the following special embryological articles: *Allantois, Amnion, Area Embryonalis, Blastoderm, Blastopore, Chorion, Cœlom, Differentiation, Embryos, Gastrula, Germ Layers, Impregnation, Neurenteric Canal, Notochord, Placenta, Proamnion, Segmentation of the Body, Segmentation of the Ovum, Umbilical Cord*, and *Yolk Sac*.

In this article are given, (1) a brief sketch of the history of embryology; (2) practical directions for the study of embryos.

1. History of Embryology.—Although embryology is the department of morphological science now most in vogue among investigators, it has held this high rank but a very short time. Embryology may date its birth, after gestating for many centuries in the womb of science, from the year 1600, when Fabricius ab Aquapendente published his work, "De Formato Fœtu," followed four years later by his "De Formatione Fœtus." After Fabricius came a series of anatomists, who during the seventeenth and eighteenth centuries slowly added to the knowledge of the development of man and other vertebrates; but it was a time of vague general notions, a period when principles which seem to us elementary were still under debate. It was not until Caspar Friederich Wolff published his dissertation, "Theoria Generationis" (1759), that the mere idea of development by gradual differentiation of unformed material could make its way. Wolff is justly regarded as the initiator of modern embryology, for until his views of gradual differentiation (epigenesis) were established correct embryological conceptions were impossible. The next great advance was due to the influence of Döllinger, of Würzburg, a man who inspired many of the best researches. Under him were trained Pander and von Baer, who first definitely ascertained the existence and traced much of the history of the germ layers. Von Baer was a magnificent intellect—among the great morphologists of Germany easily first. His book (1829) on the development of animals has never been equalled for keen insight and original profound thoughts in the domain of morphology. The author is no less remarkable for his observational powers. Kölliker says, with perfect truth, that von Baer's researches "are to be described as unconditionally the best which the embryological literature of all time and all peoples has to show." The third epoch may be said to have begun with the establishment of the cell doctrine by Schleiden and Schwann (1839), after which began the labor of ascertaining the origin and metamorphoses of the cells in the embryo. Schwann's discovery led to the recognition of the real signification of the segmentation of the yolk, which had been previously discovered. The next great change occurred during 1860–1870, after Darwin had given a mighty impulse to biology by the publication of his "Origin of Species"; in this period the germ layers were discovered and described in invertebrates by Kowalewsky, Metschnikoff, and others, and in the course of a few years it was definitely proved that the germinal layers exist in all multicellular animals. Since 1870 a multitude of researches have been carried out, a wealth of new discoveries made, largely in consequence of the vast improvements in the methods of investigation. These improvements have been—first, in regard to the means of preserving ova and embryos; second, in the manners of making sections and staining them.

Of recent writers the student of human embryology must place His and Kölliker first; the former has worked out the anatomy of very young human embryos with surprising skill, and the latter has contributed a vast series of observations on the development of nearly every organ and tissue.

In the history of embryology, then, the following points mark the chief epochs:

1759.—The doctrine of gradual development, or epigenesis, definitely established by Caspar Fr. Wolff.

1829.—The existence of the germ layers demonstrated, and their most important metamorphoses in vertebrates traced out by Carl Ernst von Baer.

1839.—The cell doctrine applied to animals by Schwann, and embryology turned into the study of histogenesis.

1860–70.—The presence of germ layers in invertebrates proven by Kowalewsky, Metschnikoff, and others.

1870–85.—Constantly increasing number of special researches, and steady perfecting of methods.

2. Embryological Methods.—The student of human embryology cannot obtain his material at will, but can only take advantage of opportunity. A considerable number of abortions and miscarriages, natural and procured, occur in every community, and the ova and embryos thus discharged are, in a minority of cases, normal and fresh; the older the embryo the more likely it is to be in good condition. Embryos less than two inches long are best preserved intact; larger embryos are much better opened, and the parts severed and hardened separately. If the specimen is intended only for the study of gross anatomy, it will suffice to preserve it in seventy-per-cent. alcohol, which must, however, be renewed once or twice at first, and the larger the specimen the more necessary is the caution of changing the alcohol.

If the specimen is good enough to be used for section cutting, it must be preserved with more care. Embryos of more than seventy days should be opened and partially dissected if good preservation of the internal parts is desired. (*a*) For general use Parker's fluid (formalin 16 c.c., water 784 c.c., and 96-per-cent. alcohol 1,200 c.c.) may be recommended, as it has great penetrating power; it should be renewed after twenty-four and forty-eight hours, and the quantity of fluid should be about ten times the volume of the embryo. (*b*) For embryos of nine weeks or less preservation intact in Zenker's fluid (corrosive sublimate, 10 gm., potassium bichromate 5 gm., sodium bisulphate 2 gm., water 200 c.c., glacial acetic acid 10 c.c.) or in von Rath's fluid (saturated solution of picric acid 100 parts, hot saturated solution of corrosive sublimate 100 parts, acetic acid 1 part) may be recommended. Zenker's fluid requires from twelve to thirty-six hours, von Rath's from one-half to two hours, according to the size of the specimen. Both of these require to be followed by treatment with tincture of iodine to remove the corrosive sublimate. (*c*) Of all good methods the most expeditious is to place the embryo or organ for five minutes or less in a mixture of 10 parts strong nitric acid and 90 parts water; transfer to sixty per cent. for an hour or two, and then to seventy per cent. (*d*) The simplest method of all is to put the specimen into sixty-per-cent. alcohol for twenty-four hours, then permanently in seventy to eighty per cent.

In preserving embryological material observe the following rules: Handle the specimen as little as possible; do not on any account put it in water or wash it; if it is necessary to keep it moist, wrap a soft damp cloth gently round it; never put a fresh specimen in strong alcohol; never keep a specimen in strong alcohol, *i.e.*, over eighty per cent. The only time when strong alcohol can be safely used is after a specimen has been hardened; and then only to act for twenty-four hours immediately before embedding.

To cut sections: For very small objects, paraffin is satisfactory, but for most of the work of the human embryologist celloidin is the best embedding material. A specimen to be embedded in this material is put (1) for one day in ninety-five-per-cent. alcohol; (2) for one day in a mixture of equal parts ether and ninety-five-per-cent. alcohol; (3) for one day in a thin celloidin solution; (4) embedded in celloidin. To embed, wrap a piece of glazed paper round a *cylindrical* cork, so as to make a paper cup of which the end of the cork forms the bottom; the paper may be fastened to the cork with a couple of pins; the cup must be considerably deeper than the object to be embedded, because bubbles form in the celloidin, and it is desirable to have the celloidin so deep that the bubbles will rise above the specimen; the specimen is placed in the cup, which is then filled with thick celloidin solution. The object may then be pushed into the right position for cutting. The cup is allowed to stand until a film is formed over the celloidin, and is then (5) transferred to a jar of eighty-per-cent. alcohol, where it remains until the celloidin is thoroughly hardened, a process requiring several days. To keep the cork down and the cup right side up, put into the bottom of the cork a sinker made of a heavy bullet and a stout pin or sharp-pointed wire nail. Celloidin is made by Schering, at Berlin, and sold in ounce boxes; it may be dissolved in equal parts ether and alcohol; two solutions are required, one about the consistence of maple syrup, the other like thick molasses.

Celloidin sections must be made under alcohol. The sections are stained and the celloidin is left on; to mount them, place the sections in alcohol on a glass slide, drain off the extra alcohol, and drop on top of the sections a thin filtered solution (fifteen per cent. is good) of white shellac, enough completely to cover the sections; dry the slide at a gentle warmth, say 30° C., until the shellac is hard; clear up with oil of cloves, and mount in balsam. (This method is new, and has not been published before.)

Staining: Small pieces, not exceeding one-fourth of an inch in diameter, may be stained *in toto* before embedding. The best method, on the whole, because the safest, for *in toto* coloration, is to soak the object for one to two days in alum-cochineal, made by boiling seven parts powdered cochineal and seven parts burnt alum with four hundred parts water for at least one-half hour; the solution must be filtered before using. For staining, Mayer's paracarmine and Mayer's hæmatein solutions