mia leads to contraction of the vessels, through which means the pressure is gradually raised. The fluid mass of the blood is quickly replaced by absorption of the tissue fluids through the activity of the cells of the blood-vessel walls. After a longer period of time complete regeneration of the corpuscular elements takes place. In anhydræmia caused by the sudden removal of large quantities of fluid from the body, as in excessive diarrhœas, etc., the effects upon the circulation are practically the same as those resulting from hemorrhage. In chronic anæmias due to long-continued decrease in the mass of the blood, blood pressure is lowered and the heart beat slowed. The circulatory apparatus gradually adapts itself to the new conditions, and both heart and blood-vessels become atrophic. Atrophy and fatty degeneration of all the internal organs also take place. Similar changes take place in the forms of anæmia due to changes in the red cells, hæmoglobin, etc. (see *Anæmia*).

Local anæmia or ischæmia is the result of a diminished flow of blood to certain organs or tissues. The total mass of the blood may be normal, or the local anæmia may be coincident with a general anæmia due to changes in the blood as a whole. Local deprivation of blood supply may be caused by ligation, obstruction, embolism, thrombosis, compression, or disease of the vessel walls, or any cause which may act as an obstruction to the afferent arteries of the parts affected. Collateral local anæmia may also occur, but plays but a slight pathological rôle. Local anæmia may also be of neurotic origin, dependent upon increased resistance due to vaso-motor contraction.

In the anæmic area there results a slowing and diminution of the blood stream, which is directly proportionate to the degree of obstruction of the artery. If the vessel is completely occluded the blood current comes at once to a complete stasis. If the part is well supplied with anastomosing collaterals the anæmia may be compensated by an increased flow through the branches, which eventually become greatly dilated and hypertrophied, so that the restoration of the circulation may be complete. If the anæmic area is supplied by a terminal artery which is poorly supplied with collaterals, the blood of the part beyond the point of obstruction is gradually pressed out by the contraction of the tissues about the vessels until the pressure in the anæmic vessels reaches that of the capillaries or is even less than it, so that a backward flow may take place to a slight extent. This is not sufficient for the nourishment of the affected area, so the tissues thus shut off from nutrition either gradually or quickly die. The parenchymatous cells first undergo necrosis, later the endothelium and connective-tissue cells. Such a dead area is spoken of as an anæmic infarct.

If the obstructed artery is supplied with collaterals, the blood pressure in the vessel beyond the obstruction after sinking to a certain minimum will rise again because of the afflux of blood from the collateral veins and capillaries. If these are capable of dilatation, as is frequently the case under normal conditions, the nutrition of the part may not suffer and no anæmic necrosis follow. For this reason the obstruction of a terminal artery in the lung or in the mesentery will not, under normal conditions, lead to infarction; but if the circulation in these regions is already so disturbed that the collateral anastomosis does not yield sufficient nutrition to the part, death will result. Since the blood-vessels under these circumstances contain a greater or less amount of blood, the damage to the vessel walls leads to escape of the blood either by diapedesis or rupture. The necrotic tissue then becomes infiltrated with blood, and the area thus affected is known as a hemorrhagic infarct. The latter is in fact only an anæmic infarct which has become infiltrated with blood which comes into the damaged area from neighboring collaterals. Anæmic infarcts occur in the brain, heart, liver, spleen, kidneys, and muscles; hemorrhagic infarcts only in the lungs and mesentery.

About the periphery of the anæmic infarct there is always seen a narrow zone of hemorrhage and congestion. This is in reality a narrow line of hemorrhagic infarct surrounding the anæmic area, and is produced by the limited collateral anastomosis. To the naked eye all anæmic tissues appear pale. They are also softer than normal, and the color proper of the tissue is more distinctly brought out. An anæmic infarct is grayish or yellowish in color, and cheesy in consistence. In the early stage it is elevated and outlined by a narrow zone of bright red. Later the necrosed area becomes contracted from the absorption or organization of the infarct. Through the contraction of the newly formed tissue deep fissures or grooves may be formed in the surface of the organ. The fresh hemorrhagic infarct of the lungs is dark red, elevated, and firm; later it becomes brown and undergoes softening and shrinking. Since the tissues are dead in both conditions the sequelæ of both anæmic and hemorrhagic infarctions are the same as those of necrotic tissue in general: absorption, organization, sequestration, calcification, etc. Obstruction to the venous outflow produces a damming back of blood, which may be followed by extensive and destructive extravasations. The term hemorrhagic infarct has also been applied to this condition, but it seems best to restrict its use to the anæmic necrosis which is followed by hemorrhage, and to classify this condition under hemorrhage. The rapidity and completeness of the development of a collateral circulation after arterial obstruction depend entirely upon the size and distensibility of the collateral branches. In some cases small capillaries or arterioles become changed in a few weeks to large-sized vessels.

Changes in Current.—As a result of varying conditions in and around the blood-vessels, retrograde currents may at times be produced. These occur most frequently in those veins subjected to pressure resulting from muscular activity. Removal of abnormal pressure by the withdrawal of pleuritic and peritoneal effusions, decrease in the intrathoracic pressure, etc., may cause a reversed current in the large veins near the heart. Compression, ligation, and obstruction of the veins from any cause may lead temporarily to the production of abnormal currents. Collateral local hyperæmia may have the same effect. Such abnormal currents play an important part in the retrograde metastasis of tumor cells and infective emboli. Eddies are also occasionally set up in the veins at the point where a rapid current from one branch is mingled with a slow one coming from another. As these currents of different velocities come together eddies are formed which may favor the formation of a thrombus.

With the slowing of the blood current in any vessel the difference between the peripheral and axial zones of the stream becomes less and less distinct. When a certain degree of slowing is reached, the red cells pass into the peripheral stream and this difference is lost altogether. A clot formed on the vessel wall under such conditions will be composed chiefly of red cells. If the current is but slightly slowed, the peripheral stream contains only leucocytes, and a thrombus formed upon the wall under these conditions will be composed only of fibrin, blood plates, and leucocytes. Alternations in the velocity of the current will lead to the formation of laminated thrombi. However, such changes in current do not always in themselves lead to the production of thrombosis.

Stasis.—This term should be applied to the complete stoppage of the blood current. This may take place either when the vessels are full of blood or when they are empty; hence we may distinguish an anæmic and a hyperæmic stasis. In the former the vessel is collapsed and the red cells are not crowded together. Such stoppage may occur in collateral anæmia or in a general or local anæmia due to any cause. Hyperæmic stasis is more common and is subsequent to excessive hyperæmia, in which the red cells are forced into the vessels under such great pressure that their outlines cannot be made out. The dilated vessel appears as if filled with a homogeneous scarlet mass. If the obstruction is removed before coagulation takes place, the red cells become separated and the circulation is restored. If the stoppage is of long duration, thrombosis takes place. Stasis is also produced by evaporation (exposure of the intestines during laparotomy), heat, cold, alcohol, chloroform, concentrated sugar and salt solutions, or any substance which has the power of quickly abstracting fluid from the blood, injuring the vessel wall, or causing destruction of the blood corpuscles. As a result of stasis a gangrenous necrosis of the affected part may take place.

Coagulation.—Sooner or later after death the blood in the vessels and heart undergoes coagulation. As this usually takes place when the red cells are uniformly distributed throughout the blood, a red clot is formed (cruor, currant-jelly clot, post-mortem clot). In leukæmic blood the post-mortem jelly clot may be yellowish or grayish throughout, having an appearance like that of pus. In the heart and in the large veins the red cells may settle to the lower side of the vessel before coagulation takes place, so that after clotting, the upper layer of the clot, which may contain but few red cells, has a yellowish or grayish appearance (post-mortem mixed clot). Ante-mortem clots are also formed in cases of slow death, where there is a gradually progressive heart failure. These form only in the peripheral stream of the heart and larger vessels, chiefly in the auricles and venæ cavæ. They contain, therefore, few red cells and have a grayish or yellowish appearance. The greater the number of leucocytes contained in the clot the more yellow its color. Toward the axial stream the clot may contain red cells; or when the current is greatly slowed, or there are alterations of rapid and slow velocity, the ante-mortem clot may be mixed or laminated. Such clots are not infrequently mistaken at autopsy for thrombi. They can be distinguished from these by their consistence, size, position, and the fact that they are nowhere adherent to the vessel wall. Coagulation of extravasated blood may also take place either in the tissues or in any of the body cavities. Such clots are usually red, but may be mixed as a result of the settling of the red cells before coagulation begins.

Thrombosis.—If coagulation takes place within the heart or vessels during life as a result of changes in the vessel wall, in the blood current, or in the quality of the blood the process is known as thrombosis, and the resulting clot a thrombus. The first stage of the process consists in the deposit of blood plates upon the intima at the point of injury. These become fused into a granular mass, leucocytes collect and are included in the blood-plate mass, and finally stringy fibrin is deposited. If the blood current is strong and the thrombus does not extend into the axial stream, a white thrombus will be formed, composed only of hyaline and stringy fibrin with a greater or less number of leucocytes. If the current is very slow, a red thrombus results through the inclusion of red cells, which with the slowing of the current pass into the peripheral stream; or, if there are alterations of velocity, laminated thrombi may be formed.

The causes of thrombosis are to be referred primarily to changes in the vessel wall: arteriosclerosis, fatty degeneration, calcification, inflammation, necrotic processes, the various changes in the vessel wall produced by the infections and intoxications, direct injury, etc. Impaired efficiency of heart's action from any cause, dilatation, compression, or stricture of the vessels may also lead to thrombosis. Further, changes in the blood, as in superficial burns, intoxications, anæmia, cachexia, etc., favor coagulation.

The sequelæ of thrombosis are: contraction, simple softening, absorption, purulent softening, organization, or calcification. Embolism may result if the thrombus becomes dislodged and is carried away with the blood current. Obturation of a terminal artery by thrombosis leads either to hemorrhagic or anæmic infarction. Thrombosis of veins produces venous stasis, hemorrhage, etc. The most favorable sequela is organization, the most unfavorable purulent softening, since this leads to the metastasis of emboli containing pus organisms.

Embolism.—The dislodging of a portion of a thrombus or ante-mortem clot and its metastasis by the blood current to other parts of the vascular system where it is unable to pass the lumen is called embolism, and the portion of clot transported is called an embolus. This will have the same structure as the parent thrombus; but after lodgment in a vessel a secondary thrombus of different structure may be formed upon the embolus. The sequelæ of embolism are essentially the same as those of thrombosis. Metastasis may take place either in the direction of the current (direct) or in the reversed direction (retrograde), or the embolus may pass from the venous to the arterial system through a patent foramen ovale or ductus Botalli (paradoxical or crossed embolism).

Hemorrhage.—Any escape of blood from the vascular system is styled a hemorrhage. This may take place either by rupture (rhexis) or by diapedesis. In the latter form the constituents of the blood pass through the vessel walls without appreciable lesion of the latter. Hemorrhage from arteries occurs by rhexis only, from veins and capillaries by both rhexis and diapedesis. The size of the hemorrhage bears no distinct relation to the manner of escape. Large hemorrhages may be caused by either rhexis or diapedesis. The causes of hemorrhage by rhexis are: mechanical injury, diseased conditions of vessel wall, increase of intravascular pressure, and decrease of external pressure. In the arteries changes of pressure are not in themselves sufficient to cause hemorrhage, but in the small veins and capillaries marked disturbances of pressure may lead to rupture. Diapedesis is caused by an increased permeability of the vessel wall in association with a rise of pressure in the veins. Passive hyperæmia, stasis, mechanical, thermal, and chemical lesions of the walls, disturbances of nutrition, etc., may all lead to diapedesis. Neurotic diapedesis also occurs in connection with disturbances of the vaso-motor system, arising either from the central nervous system, lesion of the conducting nerve fibres, or reflexly. Vicarious menstruation, stigmatization, etc., are to be included in this class. A tendency to hemorrhage is called hæmophilia; this may be either congenital or acquired. The former is an inherited condition, the pathology of which is unknown. Acquired hæmophilia occurs in all forms of severe infections, in the various purpuras, in disease of the blood, in many intoxications, etc. The sequelæ of hemorrhage are anæmia which is directly proportionate to the amount of blood lost, absorption or organization of the extravasate, formation of hæmatoidin and hæmosiderin from the broken-down hæmoglobin, etc.

Œdema.—The fluid of the tissues (lymph) is essentially a secretion of the cells of the blood-vessel walls. When the amount of this fluid is increased to an abnormal degree so that the tissue spaces are over-filled with it, the resulting condition is known as œdema (dropsy, hydrops). This pathological increase of the lymph is in all cases chiefly due to an increased activity of the secretory cells of the vessels. This is shown by the fact that the fluid of œdema contains less albumin and frequently more extractives than the blood, that its chemical nature in different portions of the body differs, and that its production is under nervous control. Further, the specific function of the capillary walls may be excited by injection of certain substances, hypnotic suggestion, etc. The accumulation of the vascular secretion in the tissues may be due to stagnation of the blood stream, obstruction to the outflow of lymph, pathological alterations in the walls of the capillaries altering their secretion, and stimulation of the vascular secretion by decrease of the pressure external to the vessels (*œdema ex vacuo*). Under ordinary conditions neither stagnation of the blood nor obstruction to the outflow of lymph is sufficient in itself to cause œdema; but in the first case it is probable that the high pressure stimulates the vessel walls to increased activity. The pathological changes in the walls of the capillaries and veins leading to increased vascular secretion may be either toxic, infectious, mechanical, thermal, nutritional, or nervous in origin. Clinically the following varieties of œdema are described: stagnation, inflammatory, hydræmic, cachectic, and neuropathic. The fluid of inflammatory œdema contains more albumin, and is probably partly derived from the direct escape of the blood constituents. It may be collateral, occurring in the neighborhood of inflammations.

Hydræmia only favors the production of œdema, but

does not in itself cause it. The œdema of nephritis is more probably due to the changes in the vessel walls, caused by the alterations in the blood. The œdema of anæmia may be explained in the same way. Œdema ex vacuo occurs chiefly in the central nervous system following a loss of a part of the nerve tissue. The local defect becomes filled with fluid resembling in character that of the cerebro-spinal fluid. It is produced by an increased secretion from the capillaries which is probably caused by the decreased pressure around the vessels. Œdematous tissue is swollen, boggy, inelastic, and pits when pressed upon by the finger. Extensive œdema of the skin and subcutaneous tissues is known as anasarca or hyposarca. When the condition is very extreme rupture of the skin may take place and the fluid escape. Incision or trocar puncture may be of service in allowing escape of the fluid. Collections of fluid in the thorax (hydrothorax) and peritoneal cavity (ascites) may be removed by tapping.

Disturbances of Lymphatic Circulation.—The lymphatic circulation through the thoracic duct or larger lymph vessels may be obstructed by pressure from tumors, inflammations, invasion of lumen by new growths, etc. Local œdemas may result, but this is not always the case even when the thoracic duct is obstructed, as the lymph vessels have elaborate anastomoses, and collateral channels may be opened which suffice to carry off the excess of the lymph. Obstruction of large lymph trunks will, however, increase an œdema already resulting from excessive vascular secretion. Escape of lymph (lymphorrhagia) is caused by interruption of the continuity of a large lymphatic. Since the pressure in the lymphatic circulation is practically the same as that of the tissues, lymphorrhagia can take place only from a surface, either externally or in a body cavity. The escape may be checked by very slight pressure. Rupture of the thoracic duct is the most important and dangerous. It is usually traumatic in origin, but may be the result of inflammatory processes involving the duct, or it may be caused by obstruction or compression of the duct by tumors. As a result of such a lesion lymph may be poured out into the thoracic or abdominal cavity (chylous hydrothorax, chylous ascites, etc.). If the opening in the lymphatic vessel is permanent, a lymph fistula may result from which there is a permanent oozing of lymph. In this way large quantities of fluid may be lost and serious disturbances of nutrition may result.

Chyle may also appear in the urine as a result of obstruction of the abdominal lymphatics, which in turn leads to dilatation of the lymphatics of the bladder wall. From the rupture of the latter chyle escapes and becomes mingled with the urine. Chyluria is associated with the presence of the filaria sanguinis; but the exact relations between the parasite and the changes leading to chyluria have not as yet been satisfactorily cleared up. Metastasis of tumor cells, micro-organisms, etc., also takes place through the lymph channels (lymphogenous metastasis), and may be either direct or retrograde. The latter plays a very important rôle in the metastasis of carcinoma. (See *Coagulation, Embolism, Hæmorrhage, Œdema, Thrombosis,* etc.)

Aldred Scott Warthin.

CIRCUMCISION.—(Synonyms: Posthectomy; Lat., *Circumcisio;* Fr., *Circoncision;* Ger., *Beschneidung.*) Circumcision is the partial or complete removal of the prepuce or foreskin. The operation is performed as a religious rite, and as a hygienic or therapeutic measure in congenital or acquired phimosis and in some other diseases of the prepuce and penis. Among females in Arabia, Egypt, Nubia, and some other portions of Asia and Africa, travellers have observed a similar custom, consisting of amputation of the labia minora or mutilation of the clitoris.

As a religious rite, circumcision is a practice of great antiquity and geographical extent. While most commonly performed among the Hebrews and Arabians, it is frequently observed by travellers in other portions of Asia and Africa where the origin of the custom is easily traced, and it is also seen in parts of South America, in the islands of the Pacific Ocean, in Madagascar, and in other places where no clue to its origin has yet been discovered. The Hebrews are supposed to have derived their knowledge of circumcision from the Arabians or Egyptians, most probably the latter, among whom the practice originated, according to Herodotus. An interesting account of the operation as performed in Algiers is given by Tarneau, in *Gazette des Hôpitaux,* 1855, and referred to by Hamdy in his monograph on circumcision, which also gives much valuable information of the history of the operation (Hamdy, "De la circoncision"). The age at which the operation is performed varies in different places. The Hebrews operate on the eighth day, as is also done in Algiers, while generally the Arabians wait until the tenth or thirteenth year, the age of puberty. The operation consists essentially of three parts: circular section of the extremity of the prepuce, tearing of the remainder of the prepuce to the corona, and denudation of the glans, and suction of the wound and penis by the operator. Hemorrhage is arrested by styptics, powdered coral, or generally tannin; and simple dressings are applied to prevent the inner layer of the prepuce from again covering the glans penis. As the operation has too frequently been performed by unskilful and dirty hands, serious complications and fatal results have often followed. Cases of death from excessive hemorrhage and inflammation can be found in the literature of the operation, as well as cases of various untoward complications, for example, wounds of the glans penis or urethra. Much has been said, too, of the danger of transmission of syphilis in the disgusting third part of the operation, whether from the operator to the patient, or occasionally from the patient to the operator.

The prepuce consists of two folds of integument, separated by very loose connective tissue, that cover and protect the glans penis. They meet and become continuous at the preputial orifice or end of the foreskin, which is usually the narrowest part of the prepuce, although sufficiently large in adults to allow the prepuce to be retracted so as to expose the glans. At birth the prepuce is longer than the penis, and its orifice narrow, often preventing exposure of the glans, so that a greater or less degree of congenital phimosis is pretty constant. The inner surface of the prepuce at this time embraces the glans closely, especially at the corona where there is generally a little circle of adhesions. Sometimes the prepuce and penis are adherent over a much greater extent of surface, either in little patches at various parts of the glans, or from the corona to the meatus. The adhesions are generally easy to subdue, partaking more of the nature of agglutinations than of firmly organized tissue. They are apt to be the source of local irritation, and to cause the retention of the secretion of the glands of Tyson, or smegma præputii. It occasionally happens that the prepuce is imperforate, a condition that soon makes itself manifest and is easily recognized. The child passes no urine, and a soft, fluctuating, transparent tumor forms slowly on the penis. As the tumor grows it pulls upon the skin of the scrotum and pubes, and an inspection shows that there is no preputial orifice. An incision must be made as soon as possible to allow the escape of urine, but circumcision had better be deferred to a later period, when it may be unnecessary to do more than trim up unsightly flaps. In childhood there are not many changes in the prepuce; the few erections rupture a part of the congenital adhesions. Attacks of balanitis or posthitis may result from the decomposition of retained smegma and give rise to contraction of the preputial orifice, or new adhesions between the prepuce and the glans. A child with phimosis and an unhealthy local condition may have no symptoms, and at puberty, with the changes that occur in the penis, the phimosis may disappear. But if there are dysuria or reflex symptoms arising from the phimosis, operative interference is essential, not only to relieve the immediate trouble, but as a prophylactic against various conditions apparently dependent on it, as varicocele, hernia, some cases of masturbation, etc. (see article *Phimosis*). At puberty the glans increases in size, erections become more frequent and strong, and usually destroy any remaining adhesions, while they dilate the preputial orifice so that the phimosis disappears. If any adhesions still persist they are strong and firm, and may prevent the development of the glans, and be the cause of painful erections, tenderness of the penis, etc., as well as of other symptoms more or less evidently dependent on the local condition. Such a condition is best remedied by circumcision, and, indeed, it is a question of considerable interest and importance whether it is not desirable to perform circumcision at an early age as a hygienic and prophylactic measure. The outer lamella of the prepuce is continuous near the corona glandis with the integument that covers the body of the penis. One should remember the elasticity of this integument and the laxity of its subcutaneous tissue, and avoid undue traction upon it in performing circumcision, or it may happen that when the skin has been drawn forward and amputated, an unexpectedly large surface of the prepuce will be found denuded, or there may be a circular wound near the middle of the penis while the parts covering the glans have entirely escaped the knife. Such an accident is usually a surprise to the operator, and may be followed by disagreeable deformities from subsequent cicatricial contraction. Under the circumstances, the best course to pursue is to relieve the phimosis for which the operation has been undertaken, by a dorsal incision, without removing any more integument, and after careful arrest of hemorrhage to approximate the edges of the circular wound as well as possible by sutures and endeavor to secure primary union. The inner surface of the prepuce is reflected forward from the corona glandis to the orifice of the foreskin. In its ordinary condition, protected from friction, and lubricated by the natural secretions of the part, it is soft, pliable, moist, and so much resembles a mucous surface that it is usually referred to as the "mucous membrane," in distinction from the "skin" or outer layer of the prepuce. This layer, and not the skin, is usually affected in phimosis. The constriction is usually at the meatus, although it may be anywhere between there and the corona. The frænum præputialis is a small triangular fold of the inner layer of the prepuce, is inserted near the meatus of the urethra, and when too short may interfere with retraction of the prepuce, pulling the penis downward and producing pain. On each side of it, near the median line, are the arteries of the frænum. When these are divided in circumcision they may be the source of troublesome hemorrhage; accordingly the operator is directed in most of the textbooks to avoid them if possible. Where the frænum is short, it is better to disregard this precept and divide the frænum thoroughly, or remove a portion of it, and be prepared to place fine catgut ligatures on the arteries that bleed.

A considerable number of operative procedures have been suggested and practised by different surgeons, having in view the more accurate removal of the redundant prepuce, and the coaptation of the wound surfaces. They fall into three classes: preliminary incision of the prepuce, dilatation, and amputation of the prepuce without previous incision or dilatation. The preliminary incision is usually made on the dorsum of the prepuce, as far back as the corona, and the two flaps thus formed are carefully trimmed away. The incision may be made with a pair of blunt-pointed scissors or a bistoury. The bistoury may be inserted into the preputial orifice on a director, or the point may be protected by a little lump of wax and the bistoury passed with the side to the glans as far as the corona, then, turning the back to the glans, the point is pushed through the foreskin, which is divided by drawing the bistoury forward. The trimming of the flaps may be facilitated by the use of curved forceps applied so as to leave out the portion that is to be removed. Instead of making this division the prepuce may be dilated and cotton stuffed between the prepuce and penis, but this is a tedious and sometimes impossible process, and has no advantages beyond the greater facility of applying needles and sutures in the prepuce, so as to render the coaptation of the surfaces more accurate. The incision, however, is of great value, especially where there is reason to believe that a constricted prepuce conceals a venereal sore that cannot be disinfected. Here a comparatively small incision allows the sore to be exposed, and after it has been disinfected or has cicatrized, the unsightly flaps may be trimmed off. In such cases it often happens that so much tissue has been destroyed by the ulcerative process that there is no excess of tissue when the wound has healed. The most simple and rapid method of circumcision is by a transverse incision made obliquely from behind forward and above downward, in the direction of the margin of the corona glandis, the glans being carefully protected from the cutting instrument. The prepuce may be drawn forward with the fingers or with forceps, and the glans may be protected by Ricord's forceps or the handles of a pair of scissors or other instrument applied obliquely. To insure division of the prepuce at the proper place, if the eye cannot be trusted, it is well to mark with ink or iodine on the prepuce at the point where the corona glandis underlies in a natural condition of the parts, and to make the section a very little in front of this level. Ricord made the mark two lines from the corona, and after applying forceps transfixed the prepuce with needles along the line so as to secure accurate apposition of the two preputial layers after removing the prepuce. Other surgeons, however, sometimes remove more or less of the two layers. Dieffenbach and Redreau removed all the mucous surface, leaving enough of the outer to cover partially the glans. After the prepuce has been drawn forward and divided, the forceps or other protecting instrument is removed, and the integument slides back on the penis, while the inner layer of the prepuce still covers the glans. This may either be turned back if there is no constriction, or divided as far as the corona by cutting or tearing on the dorsum. The two flaps thus formed may be left or trimmed away; it is generally preferable, with a pair of curved scissors and forceps, to cut away evenly on all sides, leaving about a quarter of an inch. At the same time that this layer is turned back any adhesions are carefully broken, or if firm, cut through, doing as little damage to the penis as possible. All hemorrhage should be carefully arrested, fine catgut ligatures being used for the purpose. The edges of the two layers are then united by fine sutures, which may be either of horsehair, silk, or catgut. A continuous catgut suture is very useful, but if many stitches are used there is apt to be considerable swelling of the parts from the contusion and handling. In young children it is not necessary to use sutures, as the dressing prevents the inner layer from again covering the glans, and retains the parts in sufficiently close apposition for union to take place readily. If silk or horsehair sutures are used they should be removed on the fourth day. Instead of sutures, the parts may be held together by serresfines, but these are apt to fall off as the patient recovers from ether and may excite erections. If they are used they should all be removed by the end of twenty-four hours. Before the operation an attempt should be made to disinfect the parts, and antiseptic dressings should be applied. In children it is impossible to prevent the soiling of the wound by urine, but in adults, with care, pretty thorough asepsis may be preserved. After urine has been passed the wound should be thoroughly cleansed with some disinfecting solution and a new dressing applied. In children, cold-water compresses and weak carbolic acid solutions, or a light dressing of iodoformized gauze are as useful as any elaborate dressing, and may be frequently renewed.

The operation should not be performed without some anæsthetic. For the surgeon's comfort a general anæsthetic is the most satisfactory, but there are many cases in which local anæsthesia may well be employed. Cocainé or eucaine may be used, or Schleich's mixture