by Wallace. The action of the muscle was first correctly described by Helmholtz (1851). A controversy of long standing regarding the existence of a dilatator muscle of the iris appears to have been settled affirmatively by the researches of Kölliker, Retzius, and Juler. The structure of the lids, the lachrymal apparatus, and the retina was specially studied by H. Müller (Müller's muscle, Müller's fibres). The layer of rods and cones (Jacob's membrane) was discovered by A. Jacob, of Dublin, in 1819, the visual purple by Boll in 1876. Recently important comparative studies of the retina have been made by W. Krause and Ramón y Cajal.

The complicated anatomy of the ear has been the object of research by a great number of observers, only a few of whom can be mentioned here. The membrana tympani has been carefully investigated by O. Shrapnell (1832), Jos. Toynbee (1851), Rüdinger (1867), and Prussak (1868); the anatomy of the auditory ossicles and the mechanism of their movements has been elucidated by Helmholtz (1868); the Eustachian tube has been specially studied by Rüdinger, Huschke, and Kölliker; the membranous labyrinth by Böttcher, Henle, and Hyrtl. The organ of Corti was discovered by the Marchese di Corti in 1851. Additional details of its structure were established by E. Reissner (1854), M. Claudius (1856), O. Deiters (1860), and Hensen (1863). Special memoirs on the anatomy of the ear have been written by Rüdinger, Wharton Jones, Ayers, and Retzius.

As to the organ of smell, the olfactory cells were first described by Max Schultze in 1862, although they were probably seen previously by Ecker and Eckhardt. The tracing of the olfactory fibres has been effected by the labors of Kölliker, van Gehuchten, and Ramón y Cajal. The general anatomy of the passages of the nose has been carefully studied by Zuckerkandl.

The taste buds of the tongue were discovered by Schwalbe, of Strasburg, in 1867, and at about the same time by Lovén, of Christiania.

The tactile corpuscles of the skin were first seen by Meissner and Wagner in 1852, the end bulbs by W. Krause and Kölliker (1850–1858). Pacini discovered the corpuscles that bear his name in 1836, and they were described by Vater somewhat later (1841). Other nerve endings recently described are those of Golgi in tendons (1878), those of Ruffini in the fingers (1893), and the "muscle spindles" of Kühne and others found in the substance of muscle.

Most of our accurate knowledge of the minute anatomy of the viscera has been developed during the present century. Space does not permit a detailed account of the discoveries, but mention should be made of the work of Neumann, Lent, and Röse upon the teeth, and the attempts of Ryder, Osborn, Cope, and others to obtain from palæontological and other evidence a connected account of the mechanics of their development; of the work of Flemming, of Kiel, upon the principles of gland construction; and that of Heidenhain of Breslau upon the anatomy of the pancreas, the salivary and peptic glands. Investigations of the development of the peritoneum by Toldt, His, Treves, Brösike, and others have greatly aided our comprehension of that complicated structure. The liver has been specially investigated by Kiernan, Hering, Heidenhain, and Ranvier, and in the anatomy of the kidney great advances have been made. Henle described the loops of the uriniferous tubules that bear his name in 1862, Ludwig and Heidenhain have done much in elucidating the structure of the tubules, and Disse has studied the changes of the epithelia during secretion.

In the generative organs of the male researches in spermatogenesis have been carried on by La Valette St. George, Nussbaum, Flemming, Hermann, and Minot. In the female organs Pflüger and Waldeyer have investigated the structure of the ovary and the development of ovules, and Nagel has given the first exact description of the human ovum. The situation of the pelvic organs has been carefully determined by B. Schultze and Waldeyer, and an exhaustive examination of the human placenta has been made by Minot. *Frank Baker.*

ANCHYLODYNIA. See *Foot (Surgical)*.

ANCHYLOSIS. See *Ankylosis*.

ANCHYLOSTOMA DUODENALE. See *Nematodes*.

ANDERSON MINERAL SPRINGS.—Lake County, California.

Location.—Nineteen miles from Calistoga, five miles from Middletown, and ten miles from the Great Geysers.

Access.—By stage from Calistoga and Cloverdale.

The worshipper at nature's shrine, the lover of grand and varied scenery, will find all that can be desired at the Anderson Mineral Springs. The mountain stage ride is one of the most picturesque in the State. The ever-changing picture of hill and dale, of forest and shrubbery, and of brooks with ferns and mosses forms one of those pleasing panoramas which the spectator loves to recall in after days. The springs with the hotel and cottages are located in a cozy nook in a small cañon surrounded by forests abounding in picturesque waterfalls. The cool, leafy dells and the profound silence and solitude of the dense forests form an ideal combination to attract the early morning rambler. The atmosphere here is balmy and exhilarating and free from humidity. Fish and game abound all the year round. The accommodations offered to guests are excellent, and visitors come by the thousand to enjoy the numerous advantages of the spot. There are nine important springs. The principal drinking-spring, known as the Cold Sulphur, is located about one hundred and fifty yards from the hotel. It was analyzed by Dr. Winslow Anderson and found by him to have the following composition:

One United States Gallon Contains:

Solids.	Grains.
Sodium chloride	1.09
Sodium carbonate	9.27
Sodium sulphate	6.18
Potassium salts	Traces.
Magnesium carbonate	11.73
Magnesium sulphate	16.95
Calcium carbonate	20.40
Calcium sulphate	9.10
Ferrous carbonate	0.46
Arsenious salts	Traces.
Silica	2.45
Organic matter	Traces.
Total	77.63
	Cub. in.
Gases { Carbonic acid gas	243.50
Gases { Sulphureted hydrogen	4.20

This may be characterized as a saline sulpho-carbonated water. It has been found very beneficial in chronic skin diseases of strumous and syphilitic origin. In liver and bowel troubles, in uterine and ovarian engorgement, and in glandular congestions, the water has also proved to be of much value. It is aperient, diuretic, and alterative in its action.

The "Sour Spring" is one of the few California mineral springs containing free sulphuric acid. Its sour taste is supposed to be due to alum, but the following analysis by Mr. George E. Colby, of the California State University (1889), shows that no alum is present:

One United States Gallon Contains:

Solids.	Grains.
Sodium chloride	0.08
Sodium sulphate	0.49
Potassium sulphate	0.87
Magnesium sulphate	4.76
Calcium sulphate	2.07
Ferric sulphate	0.63
Aluminum sulphate*	7.11
Boric acid (with spectroscope)	Strong test.
Lithium (with spectroscope)	Well-marked test.
Ammonia (manganous sulphate)	0.33
Silica	3.94
Organic matter	Traces.
Total	20.28

* A microscopic examination of the residue obtained by slow evaporation fails to show characteristic crystals of alum.

A considerable quantity of free sulphuric acid was also revealed by the analysis. The temperature of the water is 64.3° F. It possesses tonic, astringent, and gently laxative properties, and has proved beneficial in hemorrhages from the lungs, menorrhagia, dyspepsia, etc.

Another valuable water is the "Iron Spring." The following is Mr. Colby's analysis, made in 1899:

One United States Gallon Contains:

Solids.	Grains.
Sodium chloride	0.18
Sodium bicarbonate	0.19
Sodium sulphate	3.42
Potassium sulphate	1.17
Magnesium sulphate	7.35
Calcium sulphate	10.88
Calcium phosphate	0.15
Ferrous carbonate	1.18
Alumina	0.93
Boric acid (with spectroscope)	Strong test.
Lithium (with spectroscope)	Well-marked test.
Manganous carbonate	1.77
Silica	4.22
Organic matter	Small quantity.
Total	31.44

Free carbonic acid gas, 25.80 cubic inches.
Temperature of water, 124° F.

This is a mild calcic-chalybeate water. It possesses tonic and slightly laxative properties, and is useful in anæmia and chlorosis and in conditions requiring restorative agents.

Among other valuable springs in this group may be mentioned the "Cosmopolitan," an excellent drinking water, possessing laxative properties; the "Belmar" Spring, a light saline-sulphur water; the "Magnesia Spring" (known also as "Father Joseph's Spring"), a rich saline water having valuable laxative properties; and the "Hot Sulphurous" or bathing spring. These last waters have a temperature of 145.5° F., and have been found very beneficial in rheumatism, chronic joint swellings, and skin diseases. It is claimed that the inhalation of the hot sulphurous steam of this water is highly useful in cases of chronic bronchitis, incipient phthisis, and catarrhal affections of the nose and throat. There are good facilities for bathing. The incrustations formed by the hot sulphurous vapors on the surrounding rocks are gathered and powdered and used in cases of chronic nasal catarrh, as well as for acute coryza and colds in the throat. This powder represents all of the solid mineral ingredients found in the water.

James K. Crook.

ANDROMEDOTOXIN. See *Ericaceæ*.

ANDROPOGON. See *Citronella Grass*.

ANELECTROTONUS. See *Electrotonus*.

ANENCEPHALUS. See *Teratology*.

ANESON.—Anesin; chloretone; acetone-chloroform. $(CH_3)_3COH.CCl_3$, tertiary trichlorbutylalcohol. Potassium hydroxide is slowly added to a mixture of equal weights of acetone and chloroform, and then steam is blown through. The resulting aneson forms a white crystalline mass resembling camphor and having a camphoraceous odor. It is slightly soluble in cold, more so in boiling water, is fairly soluble in strong alcohol, and freely in ether and chloroform. It is decomposed by strong sulphuric acid, but is not affected by weak acids or alkalies.

Aneson or chloretone is antiseptic, locally anæsthetic, and hypnotic. Combining the properties of an antiseptic and an anæsthetic, it promises to be of considerable value in minor surgery. Kossa and Vamossy found it to be slower in its action than cocaine, and somewhat less penetrating; yet it had more anæsthetic power, as a one-per-cent. solution was equivalent to a 2.8-per-cent. solution of cocaine. A lacerated wound or a burn soaked in a weak solution of aneson soon becomes anæsthetic, and permits of incision, suturing, etc., without pain. For other minor operations, especially about the mouth, nose, and eye, or for circumcision, it is superior to cocaine, and is used in such weak solution as not to have any systemic effect. It is non-irritant and does not dilate the pupil, and being a very stable compound its antiseptic power may be increased by the addition of mercuric bichloride, phenol, thymol, etc.

Houghton and also Albrich have found aneson to be readily absorbed from the stomach, and rapidly distributed throughout the body. It tends to slow the heart without weakening it, and it has no effect on the arteries or on the blood. In animals killed after large doses the greater amount of the drug is found in the brain. It is sedative and readily produces hypnosis, but has little or no effect on the important centres in the medulla oblongata. Aneson is not eliminated as such by the kidneys, the skin, or the lungs, and, as the chlorides in the urine are increased during its administration, it is probable that the drug is broken up in the system. Given by mouth, the mucous membrane of the alimentary canal becomes insensitive, nausea and seasickness are relieved, and in gastric cancer or ulcer, the pain and persistent vomiting are overcome. As a hypnotic, it does not irritate the stomach or the kidneys, and may be used with safety in cardiac and respiratory diseases. Five to twenty grains may be given in powder or capsule half an hour before bedtime, as its action is fairly rapid. Sixty grains have been given without disagreeable effect. As an anæsthetic it may be used in one-per-cent. solution, either directly applied to the surface or subcutaneously.

W. A. Bastedo.

ANEURISM.—An aneurism of an artery is a circumscribed tumor composed of a sac, the cavity of which communicates with the lumen of the artery, and contains liquid or coagulated blood. The sac may be formed in whole or in part of the distended wall of the artery, or of the condensed adjoining tissues.

Definitions and Classification.—The terminology of the affection has been much confused by a lack of agreement in the use of terms and in the meaning attached to them. Most of these terms are intended to indicate differences in the composition of the wall of the sac, some of which cannot even be recognized with certainty on direct examination, and are not marked by any corresponding clinical differences.

Internal and External.—*Internal* aneurisms are those situated within the thoracic or abdominal cavity; *external* aneurisms are those formed at the expense of arteries lying outside these cavities. (*Medical* is sometimes used as a synonym of internal; *surgical*, of external.)

Spontaneous and Traumatic.—*Spontaneous* aneurisms are those that have arisen in consequence of disease or gradual change in the wall of an artery. A *traumatic* aneurism is one which has formed in consequence of sudden mechanical division or injury of the wall of an artery, as by a knife or splinter of bone.

The following anatomical classification, adopted by Holmes, is the one in common use. The distinction made between "true" and "false" aneurisms is anatomically justified, but the terms are likely to mislead, for "true" aneurisms, in the narrow sense of the term—*i.e.*, aneurisms whose walls are everywhere composed of all the coats of the artery—are rare and always small. The common form of aneurism belongs to the class termed "false," those in which only one of the coats of the artery takes part in the formation of the wall of the sac.

I. Common or encysted aneurism, subdivided into—

(*a*) Aneurismal dilatation, or fusiform aneurism. The artery is dilated for some distance, and the wall of the dilated portion preserves its three coats.

(*b*) True aneurism. The sac is formed throughout by all the coats of the artery dilated at only one point.

(*c*) False aneurism. The sac is formed by only one or two of the coats of the artery, the middle one having disappeared or being unrecognizable in consequence of change.

(*d*) Consecutive or diffused aneurism. The wall of the sac is formed of the condensed adjoining tissues, and the communication of its cavity with the artery is therefore through an actual opening in the wall of the latter. A traumatic aneurism is the type of this class, but most, if not all, large aneurisms would be included under the definition, rather than in class (*c*), because of the substitution of condensed connective tissue in the wall for the distended external coat of the artery. The presence of a lining coat similar to the intima of the artery is not proof of the persistence of the latter; it may be of new formation.

II. Arterio-venous aneurism, formed by abnormal communication between an artery and a vein; subdivided into—

(*a*) Aneurismal varix, in which there is no sac intermediate between the artery and the vein; and

(*b*) Varicose aneurism, in which there is an intermediate sac.

III. Cirsoid aneurism (or arterial varix), formed by the general dilatation of an artery and its branches.

IV. Dissecting aneurism, formed by the effusion of blood between the coats of an artery after ulceration of the intima.

I. Common Encysted Aneurism (mainly Spontaneous).—The formation of a spontaneous aneurism appears to be preceded by a degenerative change in the wall of the artery by which both its elasticity and its power to resist a distending strain are diminished. This change is in the nature of an endarteritis and mesarteritis, and consists in a hyaline degeneration of the intima and a disintegration of the elastic and muscular tissues forming the middle coat. It may begin without known cause, or may follow the lodgment of an embolus or some mechanical injury to the vessel, as the overstretching of the artery, the application of a ligature,* or even, as in one case, prolonged digital pressure. Under the influence of the blood pressure, increased at every contraction of the heart, the degenerated wall yields, and becomes stretched; if the degeneration has involved the entire circumference and a considerable length of the vessel, the dilatation is uniform (fusiform aneurism) or irregularly pouched; if only a small portion of the wall is involved, it expands and forms a pouch which communicates, either largely or by a narrow opening, with the lumen of the artery. The elongated forms, or dilatations, are common in the aorta, the pouched forms in the arteries of the limbs. In small, bud-like aneurisms the persisting three coats can be identified; in the larger ones they cannot be traced for more than a very short distance beyond the neck of the sac. It is reported that Haller produced aneurisms in frogs by dissecting away the outer coat of the artery (the mesenteric), but similar attempts made by Hunter upon the carotid and femoral of the dog were unsuccessful, although the dissection was carried so far that the color of the blood could be seen through the thin remaining portion of the wall.[1] The effect of local inflammatory conditions in producing aneurism is best seen in the small ones due to infected emboli coming from the heart in endocarditis, and in those due to the extension to the vessel of tuberculous processes on the outside; in these it appears that dissociation of the elastic bundles of the media is a necessary preliminary.

Examination of the wall of a sacculated aneurism of considerable size (Fig. 198) shows that it is composed of condensed connective tissue, with a lining membrane in its inner surface that resembles the intima of an artery to this extent, that it has an epithelial surface of flat cells and a deeper structure of flat cells separated by a fibrillary substance. A similar structure is found also upon the surface of thrombi, as after the ligature of an artery, and it must, therefore, be deemed not simply a distended intima, but rather a layer of newly formed tissue. Traces of the middle coat may be found at different parts of the aneurismal sac, especially in the neighborhood of its neck, where, indeed, they may form a continuous layer with that of the artery; but in the more distended portions of the sac they are entirely absent, and it appears to be well established that there is no hyperplasia of the muscular and elastic tissues which compose this coat, but that their elements undergo not only degeneration but also mechanical separation, and they have practically no share in the formation of the wall.

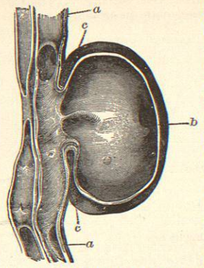

Fig. 198.—Aneurism of the Femoral Artery. The walls of the sac consist only of the *adventitia* (*a*) and *intima* (*b*); the *muscularis* (*c*) remains only at the entrance of the sac. (Weber.)

The new tissue may itself either undergo fatty degeneration, or become atheromatous or calcified. As the sac enlarges it may become thinned at some point and burst, with escape of its contents into the adjoining tissues ("ruptured aneurism"); and when, in its growth, it reaches and presses upon firm, unyielding tissues, like bone, the latter undergo absorption. Bone disappears under this pressure by rarefaction; that is, a general rarefying osteitis is set up, characterized by the enlargement of the vascular canals of the bone, by multiplication of the cellular elements, and by disappearance of the earthy salts, but without production of pus. Other tissues may become inflamed under the same irritation, and the inflammation may be plastic, with production of adhesions, or ulcerative. Thus, adjoining serous surfaces unite (pleura, pericardium, peritoneum), or rupture may take place through ulceration of the walls of the trachea or of the œsophagus, or of the wall of any other cavity that is pressed upon. These openings may be large or small, and may give rise to repeated small hemorrhages, or may cause death instantly by a free one, either external or internal.

The growth of the sac takes place in the direction of least resistance, but this direction is determined rather by the distensibility of the wall itself than by the resistance of the surrounding parts. Thus, the wall may be comparatively firm on the side adjoining a cavity, and growth may be slow in that direction, while at another point where it rests against bone the latter may be rapidly absorbed and even perforated, as is seen in the sternum, and this perforation will be followed by rapid enlargement of the aneurism through the opening. Aneurisms of the limbs seldom rupture through the overlying skin, probably because they receive treatment before their growth has reached such a point; but those of the thoracic aorta and innominate not infrequently end by ulceration of the skin and fatal external hemorrhage. An aortic aneurism reaches the surface either by growth upward into the neck or through the sternum, or between the ribs to the surface of the chest. The absorption of the bodies of the vertebræ by thoracic or abdominal aneurisms gives rise to some of the most painful symptoms of this fatal and painful affection. In two cases quoted by Mr. Holmes from Dr. Gairdner the spontaneous opening of an aneurism through the skin was followed by the healing of the opening, and in one of them apparently by the cure of the disease; but such a result is so entirely exceptional that it deserves mention only as a surgical curiosity. When an aneurism has ruptured externally or internally, the progress, in the immense majority of cases, is from bad to worse if the hemorrhage is not immediately fatal. The bleeding may be arrested by syncope or by the plugging of the orifice

*See cases quoted by Follin ("Pathologie Externe," vol. ii., p. 339), in one of which three aneurisms formed after three successive ligatures, of which the first was in an amputation just above the elbow, the second of the brachial, to cure the first; the third, to cure the second; a fourth ligature, on the axillary artery, was not followed by dilatation. The case was Warner's, in the first half of the eighteenth century, and the aneurism was laid open in each operation.

by a clot, but it recurs again and again, and ultimately proves fatal, unless the recurrence can be prevented by treatment.

The pressure of the growing tumor not only leads to the condensation and absorption of the tissues pressed upon, but it also causes much pain, either by stretching nerves or by provoking a neuritis, and it may interfere with the circulation of a part or limb by closing a vein or even an artery, and thus lead to gangrene.

The blood contained within an aneurism is usually in part liquid and in part clotted, and the inner surface of the wall of the sac is lined with layers of grayish, opaque fibrin of irregular thickness and extent. These layers may be comparatively thin, or they may fill the greater part of the cavity. They are produced by gradual deposit of the fibrin on the wall, so that those layers that are nearest the wall are the oldest, and also the shortest, because the sac has usually increased in size since they were deposited. They occasionally undergo degeneration and break down into a granular detritus, forming small cavities filled with a pulpy mass. Ordinarily the connection between the wall of the sac and the adjoining layers of fibrin is one merely by contact, and there is no growth of tissue from the former into the latter. This seems to be true at least of all growing aneurisms, but in those that have undergone spontaneous cure, or have been cured by treatment, the development of new tissue is observed. This firm, laminated fibrin is called the "active clot"; the soft, dark clot, or "passive clot," which is frequently found loose in the cavity of the sac, is probably a post-mortem formation in most cases.

The growth of an aneurism may be stayed, and a practical cure obtained, by the deposit of sufficient laminated fibrin either to fill its cavity or thoroughly to protect its wall from the distending effect of the blood pressure, and this is thought to be the mode of cure by most methods of treatment. It seems extremely improbable that this laminated fibrin is a later stage of a "passive" clot; there is every reason to believe that it is gradually deposited as such by the blood in consequence of changes or peculiar conditions in the lining membrane of the sac, or in the rapidity of the circulation. Under ordinary conditions this deposition does not take place rapidly enough to effect a cure; it occurs at some parts of the sac and not at others; its union with the sac is slight, and the blood can readily insinuate itself between the two at the edge of the layers, and as the sac enlarges fresh portions are created and left uncovered to undergo subsequent distention. If the conditions are modified by operative or other treatment that diminishes the volume and force of the stream of blood, time may be given to the tissues of the sac at the edge of the clot to become more intimately adherent to the latter, and thus to make the clot a permanent protection against further increase. This is effected by granulations from the lining membrane, which spread into the clot and over its surface, making it, as it were, a part of the wall of the sac, binding down its edges, and covering it with a smooth epithelial layer. The union between the walls and the layers of fibrin appears to be very slight, and limited to those layers immediately adjoining the wall, and there is no evidence that new vessels extend from the wall or between the layers of the fibrinous clot. Some aneurisms, after a long period of rest and apparent cure, have begun again to pulsate and to enlarge, and this fact can be explained only on the theory of a simple mechanical obstruction that has persisted during the period of quiescence, and has then yielded and allowed the re-entrance of blood, the insinuation of blood between the layers of fibrin and the wall.

A cure may also follow the sudden formation of a soft "passive" clot. This fact has only recently been demonstrated by examinations made after the rapid cure of aneurisms by the use of the elastic bandage. The first case is reported by Mr. Wagstaffe in the Transactions of the London Pathological Society, vol. xxix., p. 72; it was a case of popliteal aneurism cured a few months before the patient's death. At the autopsy the sac was found to measure two inches in length and one inch in diameter, and to contain a central blood clot measuring one by one-half inch, and surrounded by fibrous tissue which was continuous with the sac and artery. This tissue was abundantly supplied with blood-vessels, and the artery was permanently closed above and below. The process I conceive to be as follows: In consequence of the arrest of the current of blood, whether by a distal plug, or by ligature, or by compression, the blood within the sac clots, and it probably does so more promptly than within normal vessels because of the character of the inner surface of the wall of the sac. This clot fills the sac, and probably extends for a variable distance into the artery above and below the opening. This extension prevents the re-entrance of blood into the sac even if the obstruction that led to the formation of the clot is afterward removed, and the latter then undergoes those changes with which we are familiar in clots formed outside the body. It divides into two portions, a central, shrunken, firm clot, composed of corpuscles and fibrin, and an external layer of serum. The latter is absorbed by the neighboring tissues, and the sac correspondingly retracts, and its wall thickens by this retraction and possibly by a hyperplasia of its cellular elements, provoked by the irritation excited by the clot. This irritation involves also the adjoining wall of the artery, as is proved by the changes that occur even in normal vessels into which clots have extended. The intima thickens and sends out cellular prolongations, which perforate the clot and spread over its surface; these new cells soon constitute a completely formed and resistant plug structurally continuous with the wall of the artery, and provided with a smooth epithelial surface. The artery is now as completely and permanently closed on each side of the aneurism as if ligatures had been placed upon it there, and the clot is left free to undergo its natural retrogressive changes, and the aneurism is relieved from the distending pressure of the arterial stream. Complete absorption of the serum reduces the clot to less than half its original size, and this reduction is slowly carried further by molecular disintegration and absorption of the corpuscles and fibrin.

Fig. 199.—Section of an Aneurismal Sac Containing a Clot Surrounded by Organized Fibrous Tissue. (Wagstaffe.)

This conception of the process is supported by our knowledge of the changes which occur in blood that has clotted within the body under other circumstances, by certain clinical features observed in aneurisms that are undergoing or have undergone cure, and by the examination of specimens. Thus, in a case of popliteal aneurism cured by the application of the rubber bandage, a nonpulsating area of fluctuation appeared in the sac a day or two after the operation, and slowly disappeared as the tumor diminished; there can be but little doubt that it was due to the pressure of serum exuded from the clot more rapidly than it was absorbed by the surrounding tissues. Again, in Mr. Wagstaffe's case above referred to, there was found a central blood clot of comparatively small size, closely surrounded by the thickened sac, and the artery was permanently occluded by fibrous tissue continuous with its wall and with that of the sac; and in Dr. Reid's case (*Lancet*, August 5, 1876), the first one cured by the use of the elastic bandage, a similar condition of the parts was found: a central blood clot, dark in color and of cheesy consistency; a contracted but thin sac with a few partly adherent layers of laminated fibrin; and the artery occluded by fibrous tissue for a distance of two and one-half inches above the sac.

The transformation of an obliterated aneurism into a blood cyst after many years has been observed in one

case, which is apparently unique. It is reported by Reinhold ("Inaug. Dissert.," Marburg, 1882; abstract in *Centralblatt für Chirurgie*, 1882, p. 571). It was a traumatic varicose aneurism of the popliteal artery and vein successfully treated by ligature of the femoral artery and by compression of the sac. Nine years afterward a large, tense cyst formed, containing crystals of cholesterin and hæmatin, and suppurated after multiple punctures; it was then laid open, and several old blood clots and a few calcified fragments were turned out.

Causes.—Anything which reduces the power of resistance possessed by the arterial wall below what is sufficient effectively to oppose the distending force of the blood may be an immediate or a predisposing cause of aneurism. A sudden increase of intravascular pressure may combine with pre-existing weakness of the wall to produce an aneurism, but in the great majority of cases the change which leads to this production lies in the wall alone. Mr. Holmes quotes two cases in which the formation of an abdominal aneurism appeared to have been the direct consequence of the emotion experienced by a criminal on receiving a severe sentence. Weakness of the wall may be limited to a single large or small area, or may exist at many points, with the production of a corresponding number of aneurisms. This latter condition is termed the *aneurismal diathesis*, and although the affection is usually single, as many as sixty-three aneurisms have been found in one individual. The weakness of the wall is the result of change in the inner, and especially the middle, coats of the artery, and this change may be either the hyaline degeneration above described, or the one known as atheroma. Among the predisposing causes, therefore, must be counted all those which lead to degeneration of the arterial wall. The statistics collected by Mr. Crisp show that of 551 spontaneous aneurisms of all kinds, only 2 were of the pulmonary artery, 175 of the thoracic aorta, 59 of the abdominal aorta, 137 of the popliteal artery, 66 of the femoral, 24 of the carotid, 23 of the subclavian, 20 of the innominate, and 18 of the axillary. The disease is most common between the ages of thirty and fifty years, and is very rare in childhood; cases have been operated upon at eight and nine years. Broca claimed that the liability to aneurism increased with advancing years in the arteries above the diaphragm, and diminished in those below it. Aneurisms of the arteries of the extremities are much less frequent in women than in men, but there appears to be no such difference as regards internal aneurisms. This unequal distribution as regards the artery, the age, and the sex, indicates some of the causes, both general and special. Among the general causes are habits of life and peculiarities of constitution which increase the arterial tension or diminish the strength of the arterial walls; the special ones are anatomical peculiarities and local lesions, changes, and injuries.

The habits of life which act as predisposing causes are excess in the use of alcoholic drinks, and occupations which call for the exertion of much muscular effort. The influence of syphilis has been alleged, but not proven, and the same is true of its mercurial treatment. The gouty or rheumatic diathesis predisposes to it. The influence of muscular effort, so far at least as regards external aneurisms, is shown by the greater prevalence among males than among females, and the greater frequency during the prime of life, notwithstanding the fact that degenerations of the arterial walls are more common in advanced life. Follin quotes in support of the influence of alcohol a remarkable statement made to him by the Dublin surgeon, Colles, to the effect that while the Father Mathew Temperance Societies flourished in Ireland, aneurisms were much less frequently seen than before or since that time.

The anatomical peculiarities which influence the occurrence of an aneurism are changes in the direction of an artery (as the arch of the aorta), normal enlargements of its calibre (as at the upper end of the carotid), bifurcations, and the neighborhood of joints which are habitually and violently extended and flexed (as the knee and hip). The local changes which are to be regarded as exciting causes are the changes already described as occurring in the arterial wall, and other changes or injuries which diminish its power of resistance or break its continuity. Thus the sharp edge of a calcified atheromatous patch may cut through the intima and admit the blood into the rent, with the subsequent formation of a real aneurism, or of the variety known as dissecting aneurism. Or the middle coat may be ruptured by being overstretched, and the part thus weakened will be expanded to form an aneurism; or ulcerative inflammation outside the vessel may weaken, or even perforate its wall, leading, in the former case, to the formation of a typical aneurism, and, in the latter, to the transformation of an abscess into an aneurism. Or, rarely, the process set up by a ligature upon an artery may extend beyond what is needed for the sealing of the vessel, and so weaken the adjoining portion by modifying its middle coat that it yields under the pressure of the blood and expands into an aneurism. Or an embolus may lodge in an artery and lead to the same result by the same process; this seems to be especially probable when the embolus has formed during ulcerative endocarditis, and the explanation is to be found in the septic or virulent qualities then possessed by the embolus. Four cases of this kind were reported by Dr. James F. Goodhart, in the Transactions of the London Pathological Society, 1877, vol. xxviii., p. 98: in three of them the aneurism occupied the middle cerebral artery, or one of its branches; in the others, the posterior cerebral artery.

Symptoms and Progress.—When an aneurism forms suddenly by rupture or perforation of an artery, or in consequence of a violent effort or emotion, its formation is accompanied by sharp pain and the more or less prompt appearance of a tumor, if it is so situated that a tumor is recognizable. But ordinarily the formation is slow, and the patient's attention is first attracted by the presence of a tumor. This is situated in the line of an artery, is not adherent to the skin, is slightly movable, smooth and regular in outline, usually globular or ovoid, soft and compressible, and pulsates synchronously with the heart. If steady pressure is made upon it, its size may be more or less diminished while the pressure is made, but it immediately regains its former volume when the pressure is removed. If it is grasped between the thumb and fingers or between the two hands, the pulsation is found to be expansile, that is, the fingers or hands are pushed apart by it, not simply lifted by it. If the ear is placed upon it a sound is heard corresponding to the pulsation; this is the *aneurismal bruit*; and while it may vary somewhat in character in different cases, it is usually harsh rather than soft or blowing; it may be limited to the time occupied by the pulsation, or may extend over the entire interval from the beginning of one pulsation to that of the next. If pressure is made upon the artery above the tumor, the latter diminishes somewhat in size, and the pulsation and bruit cease. The pulsation in the distal branches of the artery may be normal or diminished; and if the tumor presses upon the corresponding vein, the limb may be œdematous and swollen. The compressibility and softness of the tumor are modified by the amount of laminated fibrin within the sac.

In thoracic and abdominal aneurisms many of these signs are unrecognizable because of the inaccessibility of the tumor to palpation. The objective symptoms of thoracic aneurism are abnormal dulness on percussion over the region occupied by it, an impulse communicated by it to the sternum or ribs, aneurismal bruit, and possibly the presence of a tumor at the root of the neck or on the front of the chest. Other symptoms are pain and those produced by pressure on various adjoining organs: dysphagia, diminished respiratory murmur on one side, alteration of the voice by pressure on the recurrent laryngeal nerve, and perhaps differences in the pulse when the two carotids or the two radials are compared. In abdominal aneurism the size, shape, and peculiarities of the tumor can sometimes be recognized.

Pain may accompany aneurism, when once formed, and is due either to stretching of nerves or to pressure upon, and inflammatory processes excited in them and other adjoining tissues.

The tendency of an aneurism is to increase in size; for the absence from the wall of the sac of a muscular coat, the most efficient agent to withstand the expanding blood pressure, leaves the wall unprovided with any tissue able successfully to oppose this pressure. The growth may be rapid or slow, according to circumstances, chief among which are the size of the opening by which the sac communicates with the artery, the firmness of the surrounding tissues, and the readiness with which the blood in the aneurism clots or deposits laminated fibrin upon its wall. The enlargement may be uniform, or more marked at some points, and may take place more rapidly at certain times than at others.

The natural tendency of an aneurism is to spread and finally to rupture, either by gradual weakening of its wall or by ulceration into a natural adjoining cavity or through the skin. As it approaches the surface the skin becomes tense, adherent, and inflamed, and may ulcerate or become gangrenous. The subcutaneous tissues may be similarly affected, and thus an abscess may form between the sac and the skin, into which the aneurism may rupture either before or after the abscess has opened externally. The inflammatory process outside the sac has been thought to favor coagulation of the blood within it, and thus to lead to a temporary or even a permanent arrest of the disease; but ordinarily free hemorrhage follows the rupture and requires extreme measures for its arrest, if indeed arrest is possible.

The most favorable, and one of the possible terminations of aneurism, is its *spontaneous cure* by coagulation of the blood within it. Some of the conditions which provoke or favor this occurrence have already been referred to. They may all be classified under three heads: (1) Those which favor clotting in the sac by retardation or arrest of the current through it; (2) those which increase the coagulability of the blood; (3) those which provoke coagulation through change in or about the wall of the sac.

(1) *Retardation or arrest of the current*; and (2) *Conditions which increase the coagulability of the blood.* It has been abundantly proved, both clinically and by the study of specimens, that total arrest of the current in the sac is not necessary for the coagulation of the blood contained in it, but that a partial arrest or slowing, effected by influences acting upon the general circulation or only upon the blood occupying portions of the sac, may either begin the process or promote the extension of the process after it has been begun. Most aneurisms of any size contain laminated fibrin adherent to some portion of the wall, and some are found completely filled with it, or so nearly filled as to leave only a small canal through which the current is maintained. When these clots are small, they habitually occupy those portions of the sac in which the circulation was apparently the least rapid, and it has been observed that the adoption of measures or the occurrence of changes which have diminished the rate of flow, or the quantity of blood passed through the vessel upon which the aneurism is situated, has been followed by a gradual cure through the deposition of fibrin. The permanency of such a cure depends upon the maintenance of the reduction in the rate or volume of the blood current, or upon the creation of such relations between the clot and the wall of the sac that the former becomes a permanent part of the latter, and protects all portions of it from the action of the expanding force of the blood. These relations consist in the formation of a membrane by proliferation of the cellular elements of the intima of the artery, and the spread of this membrane over the edges and perhaps over the whole of the exposed surface of the clot, in such a way as to prevent the insinuation of the blood between the clot and the wall, and to give a smooth epithelial surface over which the blood passes without depositing additional fibrin.

The causes of retardation or arrest are various. They may be found in the shape of the sac, in the general condition or habits of the patient, or in special modifications of the flow through the artery itself.

Pouched sacs, or sacs with small necks, are more favorable to the occurrence of clotting than are fusiform dilatations or sacs with large, free openings, because the blood that enters does not immediately leave them, but forms a sort of eddy beside the general stream in which the current is slow or almost nil.

Of the causes arising in the general condition or habits of the patient, the first and most important is continuous rest in bed for weeks or months, combined with a light, non-stimulating diet. Other causes, which may also act by increasing the coagulability of the blood, are bleeding, either large or small and repeated, and the internal use of various drugs, such as digitalis, tartar emetic, veratrum viride, iodide of potassium, acetate of lead, ergot, and the chloride of barium. Cures have followed the use of each of these measures, alone or in combination, but it is not always easy to determine how much credit is to be awarded to the treatment in any one case.

Retardation or arrest of the flow may also be caused by obstruction of the orifice of the sac, if it is small, or of the artery above or below the aneurism. The most common agency in producing this change is the detachment of a fragment of fibrin from the wall of the sac and its lodgment in the neck of the sac, or in the artery below. The latter occurrence is habitually accompanied by severe pain in the limb, and is evidenced by arrest of pulsation in the distal branches of the artery. A cure by this mechanism has been observed a number of times, and it forms the basis of a method of treatment suggested by Sir William Ferguson, in which the forcible detachment of a clot from the wall is sought to be effected. If the detached clot is small, it may lodge on the spur of a bifurcation, and then grow in size by additional deposits of fibrin until it obstructs one or both of the branches, and in such a case retardation precedes complete arrest.

This possibility of the detachment of small clots and their passage into the distal branches of the artery involves the risk of other changes far different from the cure of the aneurism. The arrest of the circulation may lead to gangrene of the lower portion of the limb, total or partial, according to the seat of the obliteration; and if the aneurism is situated upon the arch of the aorta or upon one of the vessels going to the head, the emboli may lodge in the vessels of the brain and cause death promptly. I once saw a surgeon examine a patient with an aneurism of the aorta that had perforated the sternum and formed a large tumor over it. He made pressure upon the tumor and reduced it through the opening; as he did so, the patient was seized with convulsions and became unconscious, and after his death, on the following day, the brain, kidneys, and spleen were found filled with emboli, fragments of the laminated clot that had lined the wall of the projecting tumor.

When there is merely retardation of the current the cure takes place by the gradual deposit of laminated fibrin; and when there is total arrest, it takes place probably by coagulation in mass of all the blood within the sac, and the subsequent shrinking of the clot and sealing of the vessel by the production of fibrous tissue, as has been described above.

Another alleged cause of retardation of the stream is pressure of the tumor upon the proximal portion of the artery, but no cases have been reported in which this mechanism has been demonstrated. Its supposed possibility rests upon theoretical grounds alone, and while it may be admitted as a possibility, there is but little reason to believe it has ever taken place.

(3) *Conditions which provoke coagulation through change in or about the wall of the sac.* Inflammation of the sac, or of the tissues immediately overlying it, is alleged by Broca and others to be a cause of coagulation within it and of consequent cure. Mr. Holmes thinks this has