rejoined his command and again underwent the exertions of a soldier's life. He soon noticed that he could not bear them as formerly; he got out of breath, could not keep up with his comrades, was annoyed with dizziness and palpitations; his accoutrements oppressed him, and all this though he appeared well and healthy. Seeking advice from the surgeon of the regiment, it was decided that he was unfit for duty and he was sent to the hospital, where his persistently quick-acting heart confirmed his story, though he looked like a man in sound condition. Any digestive disturbances which might have existed gradually passed away, but the irritability of the heart remained, and only very slowly did the excited organ return to its natural condition. Or it failed to do so, notwithstanding the use of remedies which control the circulation: thus the case might go on for a long time, and the patient, after having been the round of the hospitals, would be discharged, or, as unfit for active service, be placed in the Invalid Corps."

The important symptoms are precordial pain, usually paroxysmal; palpitations, tachycardia, the heart being characteristically more rapid in the upright position or after slight exertion; dyspnœa; and nervous disturbances—headache, dizziness, and sleeplessness. The symptoms may be absent except during exercise or they may come on paroxysmally during rest or sleep.

Examination in the functional cases shows little or no enlargement, the chief physical signs being irregularity or rapidity of action on slight exertion or on suddenly assuming the erect posture. Systolic murmurs may be present, but they are inconstant and variable. The first sound is described as short and valvular. In the cases with permanent dilatation the area is large, the impulse labored and diffuse, the first sound dull and prolonged. Constant murmurs may be present; due to regurgitation from relatively insufficient valves.

In the differential diagnosis it must be remembered that a true valvular lesion from endocarditis may exist without causing a murmur, especially in the case of mitral stenosis, and that the patient with such a lesion may correctly date the onset of symptoms to a period of over-exercise. Renal, pulmonary, and vascular degenerations must be borne in mind. Some of the cases are clinically identical with myocarditis.

The prognosis is uncertain. Complete recovery may occur, but as a rule more or less irritability remains. The quotation from Da Costa given above indicates what seems to be the common result in military cases. It is probable that many cases become virtually ones of valvular disease—relative insufficiency—or of myocarditis. Some go from bad to worse and die within a few years.

For prophylactic treatment training is again of great importance, or at least occupations requiring severe exertion should be entered into gradually so that hypertrophy of the heart muscle may occur before the full strain is put on the organ. Active exercise should be resumed with great care after recovery from acute disease. Excesses, especially of tobacco and alcohol, should be avoided permanently by those whose occupations call for a large amount of work on the part of the heart.

When the condition of irritable or dilated heart is once established prolonged rest is imperative with gradual return to active life after recovery. Among drugs digitalis will be of greatest use. The bromides will also be of service. Iron and other tonics may be called for.

In conclusion the statement must be reiterated that the danger from over-exercise in healthy hearts is not great, especially if the subject be well-trained. Indeed, Darling mentions one oarsman in whom, at the beginning of the season, the heart showed obvious signs of mitral regurgitation from previous organic disease of the valve. As the season advanced the symptoms and signs entirely disappeared, and the man did valuable work in the final hard-fought intercollegiate boat race. Such instances may be rare, but the heart certainly has a vast reserve power.

Ralph C. Larrabee.

Allbutt, T. C.: System of Medicine, vol. v.
Altschul: Münch. med. Woch., December 6th, 1898.
Bäumler: Deut. Arch. f. klin. Med., vol. lxvi., 1899.
Cautley: Brit. Med. Journal, 1893.
Christ: Arch. f. klin. Med., Bd. iii., Hefte i. and ii.
Da Costa, J. M.: Toner Lectures, May 14th, 1874.—Am. Journ. Med. Sciences, January, 1871.
Darling: Boston Med. and Surg. Journal, August 31st and September 7th, 1899; also June 6th, 1901.
Friedreich: Wien. klin. therap. Woch., 1898, Hefte 2 and 5.
Herschell: Lancet, March 2d, 1895.
Herz: Deut. med. Woch., February 22d and March 1st, 1900.
Hill, Leonard: Shäfer's Physiology, 1900, vol. ii.
Jamieson: Intercol. Med. Journal Australia, November 10th, 1896.
McCarthy: Thesis for Degree M.B., Camb., 1898. Quoted in Allbutt's System of Medicine.
Morgan: University Oars, London, 1873.
Powell: Allbutt's System of Medicine, vol. v.
Prince: Med. Record (N. Y.), April 20th, 1889.—Am. Journ. Med. Sciences, February, 1901.
Roy and Adami: Brit. Med. Journ., December 15th, 1888.
Sherrington: Allbutt's System of Medicine, vol. v.
Stengel: Am. Journ. Med. Sciences, November, 1899.
Treadwell: Boston Med. and Surg. Journal, September, 1872.
Weber: Lancet, October 28th, 1893.
Williams and Arnold: Phila. Med. Journal, June 3d, 1899.
Yeo: Edinburgh Med. Journ., July, 1899.
Zuntz: Verh. d. Berl. physiol. Ges. Arch. f. Physiol., p. 378, 1895.

HEART, THROMBOSIS OF.—The blood clots found within the heart cavities may be divided into three classes: *post-mortem clots, ante-mortem* or *agonal clots*, and *true thrombi*. The post-mortem clot is formed after the heart has ceased to beat; it is a soft, dark-red, jelly clot filling up the cavity of the auricle or ventricle and not adherent to the heart wall. It is commonly known as cruor or the currant-jelly clot. Such coagula are found usually in the right auricle and ventricle, but may be present in all four of the cavities. When coagulation occurs rapidly after death the clot is uniformly red throughout; but if coagulation is delayed the uppermost portion of the clot may be light-colored or yellowish from the sinking of the red cells. In themselves the post-mortem clots are of significance only in so far as the character of the blood and its coagulability are concerned. If coagulation takes place while the ventricles are contracted in rigor mortis the clots will be found in the auricles only, but, if coagulation be delayed until the contraction of the ventricles has passed, the ventricular cavities may again become filled with blood which later coagulates.

The ante-mortem or agonal clot is a white or mixed clot found in the periphery of the large veins, right and left auricles, and right ventricle, but rarely in the left ventricle. Such clots consist of fibrin with few or many leucocytes, while that part of the clot toward the axial stream may contain red blood cells. They develop during the slowing of the blood stream in cases of slow death, and are formed only in the peripheral stream where there are no red cells. As the current becomes slower red cells are caught in that portion of the clot toward the central stream. As soon as death occurs and the current comes to a standstill, the blood in the central stream coagulates forming a post-mortem currant-jelly clot. Combinations of agonal and post-mortem clots are thus formed and are distinguished by the presence of a central red clot surrounded by a peripheral layer of white clot. The agonal clot is soft, but much firmer than the currant-jelly clot. It is not attached to the endocardium and is easily removed from between the trabeculæ of the ventricular wall. It is further distinguished from true cardiac thrombi in its moistness, translucency, and greater elasticity. On microscopical examination the agonal clot is found to consist almost entirely of a coarse reticulum of fibrin; on the other hand the fibrin in the post-mortem jelly clot is in the form of fine fibrillæ. The significance of the agonal clot is only that of a slowly progressive heart failure; in cases of sudden death without previous impairment of cardiac efficiency the white peripheral clots are either very small or absent, while in cases of slow death in chronic valvular disease, cachexias, etc., they may be very large. Care should be taken to differentiate between these and the true heart thrombi for which they are frequently mistaken.

The true heart thrombi are yellowish, reddish, or reddish-gray or brown, opaque, dry, inelastic, brittle, and

in the great majority of cases more or less adherent to the endocardium. The conditions under which they are formed are practically the same as those of thrombosis of the blood-vessels, the chief factors being lesions of the endocardium, disturbances of the circulation, and changes in the chemical composition of the blood. Of these the endocardial changes are by far the most important. At the point of lesion blood plates are first deposited, followed by the processes of conglutination and coagulation as in the formation of thrombi in the vessels. The resulting thrombus is usually a mixed clot, very frequently laminated. When formed in the peripheral stream with rapid circulation it may be entirely white; if the current is very much slowed it may be red throughout. In old red thrombi the diffusion of the hæmoglobin from the red cells may give a washed-out reddish or brown color. The latter color is also the result of the formation of blood pigment from the disintegration of the red cells. The surface of the thrombus is not infrequently ribbed; this appearance has been explained as being due to wave-like movements in the blood, the process being analogous to the formation of wave marks on sandy bottoms. The general external appearances of cardiac thrombi are dependent upon the manner of formation, rapidity of development, and the local conditions of the circulation. The size and shape of cardiac thrombi show great variation. They may be so small as to be hidden between the muscle columns, or so large as completely to fill one of the heart cavities. They may appear as small globular masses projecting from between the muscle trabeculæ (globular vegetations); or as flat, circumscribed masses firmly attached to the heart wall. On the valves they may form bead-like excrescences or vegetations. Not infrequently the thrombus is attached to the endocardium by a pedicle (heart polyp); as the result of the tearing of such attachment free globular thrombi may be formed (ball thrombi). In rare cases the thrombus may be spread over the endocardium like a false membrane.

The thrombus when cut into may be found to be made up of dry and brittle laminations, in some instances the central portion of the thrombus is under slight pressure easily crumbled to dust. In other cases the central portion may be cystic, the cavity filled with a thick reddish-brown or pus-like substance. Such secondary degenerations are of frequent occurrence. The most common is a simple softening or liquefaction; when many leucocytes are present a grayish pus-like fluid may be formed; if the degenerating portion is made up of red cells alone a grumous reddish-brown fluid results. Calcification of the thrombus may also take place, resulting in the formation of a cardiolith. As a more favorable sequela of cardiac thrombosis organization of the thrombus may occur, the latter becoming ultimately converted into a nodular or diffuse fibrous thickening of the endocardium. In some cases both the degenerating and the organized thrombi have been mistaken for cardiac neoplasms, cysts, and fibroma respectively. It is also not improbable that some of the growths on the endocardial surface described as myxoma were recently organized thrombi.

The microscopical examination shows the cardiac thrombi to be composed of fibrin, disintegrated blood cells, blood pigment, masses of calcification, etc. In case of simple softening of the central portion the cavity thus formed contains granular débris, blood pigment, etc. In recent thrombi the leucocytes present may still show traces of their nuclei. If organization has begun fibroblasts and new capillaries may be seen extending from the endocardium into the thrombus.

Thrombi are more frequently found in the right side of the heart than in the left. In the auricles the appendix, and in the ventricles the neighborhood of the apex are the favorite seats of location. The pedunculated thrombi (cardiac polyps) are of frequent occurrence in the right ventricle, while the free ball thrombi, in the majority of recorded cases, were found in the dilated left auricle in cases of mitral stenosis. Cardiac thrombi may be single or multiple, and may be found at any age. According to their location in the heart they are also designated as parietal and valvular.

Etiology.—The changes in the endocardium leading to thrombosis are either inflammatory, or degenerative, or necrotic in nature. These lesions may be the result of the local action of pathogenic bacteria or due to general intoxications, disturbances of nutrition, changes in the blood, etc. The pus-forming organisms, gonococcus, and tubercle bacillus have been found in cardiac thrombi under such conditions as to make it very probable that they were the exciting factors of the thrombosis. The valvular vegetations are in the majority of cases due to bacteria. In the acute infections fatty degeneration of the endocardial endothelium not infrequently leads to thrombosis. Local changes in the myocardium, such as anæmic infarction, myocarditis, etc., are usually associated with the formation of thrombi on the overlying endocardium. Aneurismal dilatations of the cardiac wall are very likely to be partly or wholly filled by a thrombus. In many chronic diseases in which there are general disturbances of nutrition, systemic intoxication, etc., as in chronic nephritis, pulmonary tuberculosis, chronic valvular disease, etc., heart thrombi are frequently found. In pulmonary tuberculosis they are of such frequent occurrence as to give rise to the suspicion that in many cases they may be due to the local action of tubercle bacilli which have lodged upon the endocardium. Cardiac thrombosis often occurs in cases of general cachexia (marasmic thrombi). In severe superficial burns of the skin the changes produced in the blood may lead to the formation of thrombi, both in the right side of the heart and in the pulmonary arteries. Small emboli coming from venous thrombi and lodging upon the valves or between the muscle trabeculæ of the right ventricle may give rise to secondary or induced thrombi. Sclerotic changes in the valves or mural endocardium followed by atheromatous degeneration or calcification may also become the seat of thrombosis. Dilatation of the heart and slowing of the blood stream are favoring factors in all of the conditions named above, but are probably not in themselves sufficient to cause thrombosis; the changes in the endocardium must be regarded as of prime importance.

Symptoms.—Thrombi of large size may exist indefinitely without serious impairment of cardiac efficiency. In the majority of cases the symptoms produced cannot be differentiated from cardiac insufficiency due to other causes. The physical signs are those of dilatation; in case of the blocking of a valvular orifice by the thrombus the heart's action is usually too weak to produce audible murmurs. The valvular vegetations may give rise to symptoms of stenosis or insufficiency. In the case of the ball thrombi so frequently formed in the left auricle in mitral stenosis sudden death or the most serious disturbances of circulation may be caused by the complete or partial blocking of the mitral orifice. The occurrence of embolism, either of the pulmonary or of the systemic arteries, is the symptom of greatest value in the diagnosis of cardiac thrombosis. Embolic abscesses may be produced throughout the body in the case of emboli arising from thrombi caused by pyogenic bacteria. In the case of a patent foramen ovale paradoxical embolism from thrombi in the right side of the heart may occur. In general it may be said that the diagnosis of cardiac thrombosis is usually impossible, having been made in but few cases. Aside from the occurrence of embolism the irregularity of the symptoms and physical signs may excite suspicion of a movable body within the heart.

The Prognosis is bad, not only because of the interference of the heart's action and the danger of embolism, but also because of the conditions which have led to the thrombosis. The treatment is directed chiefly to the latter and to the support of the heart.

Aldred Scott Warthin.

HEART, WOUNDS OF.—Wounds of the heart may be either non-penetrating or penetrating—injuring the cardiac wall or opening a cavity. The chief dangers

from the former are shock and injury to a coronary artery. Ninety per cent. are penetrating, and of these only nineteen per cent. are immediately fatal. The right ventricle is most frequently injured, and the left auricle is least so. Auricular wounds are more fatal than ventricular, and injuries to the apex are less dangerous than either. A needle puncture will rarely cause hemorrhage from a ventricle, but excessive bleeding, which is mostly systolic, is liable to follow a like injury to an auricle. A wound inflicted during diastole is less dangerous than a similar injury during systole, perpendicular wounds are more fatal than diagonal, and those of the right heart bleed more profusely than those of the left. The presence of the foreign body in the heart, the size of the wound, the location of the wound, the number of wounds, the connecting cavities, the attending syncope, the involvement of Kronecker's co-ordination centre, are important factors in determining the outcome. Pericarditis, myocarditis, endocarditis, cerebral embolism, and empyema are frequent secondary complications. When the wound heals there is a possibility of cicatricial stretching, and subsequent rupture as in a case reported by Izzo of a man stabbed in the left ventricle, who was conveyed to the hospital from which he was discharged cured on the twenty-eighth day; a few hours afterward, while lifting a heavy body, he fell and quickly died. At the autopsy the cicatrix of the left ventricle was found ruptured. In addition to injuries of the heart caused by bullets, daggers, knives, needles, and various implements of war, it may be wounded by a fractured rib, by a foreign body that has perforated the œsophagus, or by blows upon the chest. Traumatic ruptures are more frequent after a full meal, as the distended stomach pushes the heart upward and forward, and causes a large area of the organ to be in contact with the thoracic wall and receive the impact.

Newton and Gamgee have collected forty-five cases in which traumatisms caused rupture of the heart without opening the pericardium. All were fatal. Only one of Gamgee's cases survived the injury fourteen hours. The fatality is easily understood, for, there being no exit, as soon as the accumulation of blood exceeds the limit of pericardial distensibility, the cardiac movements are mechanically stopped. The surgeon not only would be justified, but it is his bounden duty to operate upon every one of these cases, for it is more important, if possible, to rescue a drowning heart than to relieve a strangulated hernia. It is possible for a foreign body, as a gun missile, to remain quiescent for years in the myocardium, as in the case of a British officer, reported by Stevenson, who carried a bullet encapsuled for eleven years, or the still more remarkable case of Beers (Cincinnati *Lancet Clinic*, 1898), of an American soldier who survived the lodgment of a bullet in the wall of the left ventricle for thirty-seven years.

Symptoms.—There is no certain cardinal sign by which an injury of the heart can be denoted, but when we consider the location of the external wound and the usually profuse hemorrhage from it, if it is large, in connection with other signs and symptoms, it can generally be recognized. The pulse is weak, irregular, and intermittent, and often imperceptible in the extremities. There are pain, pallor, restlessness, and generally dyspnœa. Frequent attacks of syncope are common. The mind is clear but the countenance depicts an anxiety and distress that seemingly would welcome death, with the wintry sterility of the grave, as a sympathizing friend. If there is much blood in the pericardium the heart sounds are indistinct and percussion elicits increased dulness. With the pulsation of the heart a foreign body, as a needle, may be seen to move under the skin, a sign centuries ago enshrined in the magnificence of indestructible verse:

> "He fell, the spear point quivering in his heart
> Which, with convulsive throbbing, shook the shaft."

Treatment.—I presume no one will question the propriety of immediate operative interference, as experience teaches that about thirty-nine per cent. of cases recovered in which the heart had been sutured as against ten per cent. of recoveries in which no operation had been performed. The patient must be kept absolutely quiet. A probe should never be employed, as it is liable to inflict an additional injury upon the myocardium. For temporary hæmostasis some operators have recommended Pean's forceps and various modifications of it, but they all more or less lacerate the myocardium and can never replace digital pressure. Interrupted sutures are preferable in closing a wound of the myocardium. They should not involve the endocardium, and as few as possible should be passed, commensurate with safety against leakage, as they cause a degeneration of the muscular fibre with its tendency to dilatation and rupture. Giordano, from his experiments upon rabbits, concludes that auricular wounds might be closed advantageously with Lembert's sutures. Silk is preferable to any other material. The needle should be the same as that used in suturing the intestines. The sutures should be passed and tied during diastole. If the wound in the heart is sufficiently large a finger should be introduced into it, as Parrozani did, for the double purpose of checking the hemorrhage and to facilitate the passage of the first suture which should be used to steady the heart for the passage of the others. If the pericardial sac is closed either interrupted or continuous sutures may be used. It hardly seems necessary to accentuate the fact of the necessity of perfect cleanliness in these operations whenever the urgency of the case does not require instant intervention, as in the patients of Longo and Ninni. The question of administering an anæsthetic is a mooted one. Ollier says general anæsthesia should not be thought of. Parrozani operated upon his two cases without an anæsthetic, and observed only slight movement when he cut the skin and in passing the sutures in the myocardium. Giordano made the same observation in his case, but thinks Ollier's statement is too sweeping; and if the pulse is strong and the general condition good, chloroform may be administered. Struggling from an anæsthetic is liable to produce detachment of a clot, and renew the hemorrhage, as occurred in Parlavecchio's case. No two operators have made similar external incisions for exposing the heart. Professor Rotter performed a number of experiments upon the cadaver, and concluded that the easiest way to expose the heart was by making an incision four inches in length along the lower border of the third rib, commencing three-fifths of an inch to the left of the sternum. A similar incision was made along the lower border of the fifth rib. The external ends of these incisions were joined by a third. The fourth and fifth ribs were divided and the musculo-osseous flap was turned inward. If more space should be required the third rib could be included. No arteries excepting the intercostal require ligation in this operation. In Parrozani's successful operation an incision through the soft parts one and one-fourth inches from the margin of the sternum, in the fourth intercostal space, was carried for a distance of five and one-half inches, then it descended vertically in the mid-axillary line as far as the superior margin of the ninth rib. The fifth, sixth, seventh, and eighth ribs were cut through, in the mid-axillary line, the incision extending through the pleura. The flap, or door, was raised with the cartilages of the ribs acting as hinges. Parrozani closed the pericardial sac and sutured the flap in position. As the uncleanliness of the weapon and often the urgency of the operation preclude the proper preparation of the patient, it is considered safer to drain the pericardial sac with iodoform gauze as was done in the successful cases of Rehn, Ramoni, and Rosa. There are on record eighteen cases of heart suture and one of ligation of the left coronary artery. Five of these eighteen cases were injuries to the right ventricle and were stab wounds. In two of the cases the surgeons stated that the wounds were one-fourth of an inch and three-fifths of an inch in length. In two cases three interrupted stitches, in two four interrupted stitches were taken, and in the fifth case the wound was closed with a continuous suture of five stitches. Two of the patients recovered. Of the three

unsuccessful cases, broncho-pneumonia caused the death of one on the sixth day, and in the other two cases the operators were not explicit as to the cause of death.

In one patient the left auricle was opened to the extent of four-fifths of an inch. It was closed with four silk stitches. Death occurred on the nineteenth day from abscesses in the lungs.

In three cases the dagger entered the apex, and in two of these it extended into the left ventricle. In these two cases the wounds were respectively three-fourths of an inch and one and a half inches in length; both were closed with four interrupted sutures. The wound which did not enter a cavity was closed with one stitch. All ended in recovery.

There are seven cases of stab wounds of the left ventricle. The length of these wounds varied from three-fifths of an inch to one inch and two-fifths, and required from two to four stitches. Two patients recovered, one died on the operating table of hemorrhage, one in fifteen minutes after reaching the bed, one of pericarditis several days after the operation, and the other two within twenty-four hours, never having reacted.

In two of the cases no mention is made as to the location of the wound. One was a shot and the other a stab wound of the heart. The patient that was stabbed died of empyema on the twenty-second day after suturing.

The left coronary artery was cut and ligated two hours after the injury. The patient had pericarditis and died thirty minutes after the second operation, which was performed on the fifth day for the purpose of drainage. After most of these operations stimulating hypodermic injections were used and hypodermoclysis and autotransfusion practised.

Dr. John B. Roberts, of Philadelphia, in 1881, suggested heart suture, and Dr. Del Vecchio, of Naples, in 1894, demonstrated its feasibility before the Eleventh International Medical Congress in Rome, by his experiments on dogs. Two years later the human heart was sutured by Farrina and Cappelen. It is surprising to find the statistics collected by Fischer, of Breslau, in 1868, quoted as late as 1901 as authoritative in computing the prognosis in this class of injuries.

The successful suturing of the heart by Rehns, Parrozani, Parlavecchio, Pagenstecher, and others has revolutionized the treatment and changed the probable outcome. Fischer's statistics, though a monument to his untiring zeal and energy, are now like an antiquated signboard on a discarded highway; but

> "Faith, fanatic faith, once wedded fast
> To some dear falsehood, hugs it to the last."

Luther L. Hill.

HEAT, ANIMAL.—See *Calorimetry*.

HEAT-STROKE.—(Synonyms: Sunstroke, insolation, thermic fever, *siriasis*, *coup de soleil*, *coup de chaleur*, *Hitzschlag*, *Sonnenstich*, *ictus solis*, *insolatio*.)

Definition.—Insolation is a disease whose main features are, in its milder forms, intense exhaustion, with or without rise of temperature, and in its severer forms, with an enormous rise of temperature and coma probably caused by acute auto-intoxication due to the presence in the blood of toxic decomposition products or toxic substances produced at the unusually high temperature, the exact nature of these substances being at present unknown.

From the time when it is recorded that Elisha restored the Shunammite woman's son to life after a sunstroke the frequency of this affection has often been noted. Armies at times have been almost decimated by it, especially when operating in sub-tropical or tropical countries. Sunstroke occurs most frequently in the tropics, particularly where the climate is damp. In the United States it occurs during summer in the Mississippi Valley, around the southern shores of the Great Lakes, and especially along the Atlantic coast. In the Western and Southwestern States it is rare, owing to the dryness of the climate.

Any cause that diminishes the resistance of the body predisposes to sunstroke. Alcoholism is undoubtedly the most frequent predisposing cause, not only chronic alcoholism but even a single excess. All causes producing extreme fatigue, such as severe muscular exertion and sexual excesses, predispose to an attack. Elderly people suffering from chronic ailments and invalids of any age, even if taking no bodily exercise and remaining indoors, are sometimes overcome by the heat and suffer true attacks of heat-stroke. It is not so common amongst children as amongst adults. Men are more frequently attacked than women because their occupations more often subject them to different forms of exposure to heat and require of them greater physical exercise, and because men are more often given to alcoholic excess than women.

Exposure to the direct rays of the sun is usually the exciting cause, but this is not necessary; some of the worst epidemics in the tropics have occurred during the night. While soldiers on the march and laborers working in the sun have given the greatest number of victims to this disease, men working in superheated environment such as firemen, stokers, and laundrymen have often been attacked. The main factors in the weather which are specially prone to assist in the causation of insolation are high humidity with continuously high temperature and lack of motion of the air. Humidity and especially the absence of cooling breezes are as important factors as is high temperature, although the cause of sunstroke is usually attributed to the high temperature alone. It has long been noticed that a hot, sultry, lifeless atmosphere is the one in which most cases of sunstroke occur. Impurity of the atmosphere is not unimportant, as is shown by the fact that men sleeping in crowded barracks or small tents are more often victims than those in well-ventilated quarters.

Sunstroke occurs with greater frequency in the tropics amongst unacclimated individuals than amongst those of long residence, who have acquired a certain amount of immunity to the effects of heat. This immunity, however, is by no means absolute, as even the natives of India, Egypt, Arabia, and Africa are at times overcome. The light-skinned races are more susceptible than the dark-skinned.

Pathological Anatomy and Pathology.—The post-mortem appearances depend on the time that has elapsed since death occurred. When the post-mortem examination is made shortly after death the following conditions are usually present: When the ante-mortem temperature has been high the viscera feel noticeably hot; the lungs are congested, often extremely so; the liver is large, and the abdominal veins are filled with blood and there is intense congestion of the brain and meninges. The left ventricle of the heart is contracted and empty; the right ventricle is filled with semi-fluid blood, sometimes giving an acid reaction. Rigor mortis has set in early. When several hours have elapsed since death decomposition has rapidly set in. The heart, instead of being contracted, is then flabby and filled with grumous, semi-fluid blood. The spleen is slightly enlarged and soft. The meninges of the brain are congested, and there is more or less abundant subpial fluid. The brain itself is usually firm and not congested, with sometimes a little bloody fluid in the ventricles. The membranes of the spinal cord are congested, the cord being firm and about normal in appearance. Small hemorrhagic extravasations are sometimes found in the viscera, pleuræ, and pericardium. The most constant changes seem to be marked congestion of the meninges and viscera, the fluidity of the blood, and the unusual rapidity with which rigor mortis and decomposition supervene.

In the minute cellular structure of the brain we find changes which are much more constant and which are fairly characteristic. Dr. Ira Van Gieson, of New York, has described these lesions in the neurons of the brain and cord, as an acute parenchymatous degeneration of the ganglion cells best shown by the Nissl stain and counterstain with eosin. In the normal cell stained in this manner the chromophilic plaques, or Nissl bodies,