FAT EMBOLISM*

WITH STUDY OF TWO FATAL CASES

By Arthur W. Elting, M.D.
AND
Charles E. Martin, M.D.
OF Albany, N.Y.

It has long been known that free liquid fat may under certain conditions gain access to the blood-vessels and be transported in the blood stream. Fat in this form, of course, is to be more or less sharply distinguished from fat in the minute particles of emulsification or saponification, which plays little, if any, important rôle in the production of embolism. Fat embolism is essentially a surgical complication and usually occurs as a sequel to trauma, especially of the long bones or fatty structures of the body. In any instance in which fat is set free from ruptured fat cells, more or less embolism may result, the symptoms of which will depend not only upon the amount of fat which enters the blood stream, but also upon the localization of the fat emboli in the capillaries.

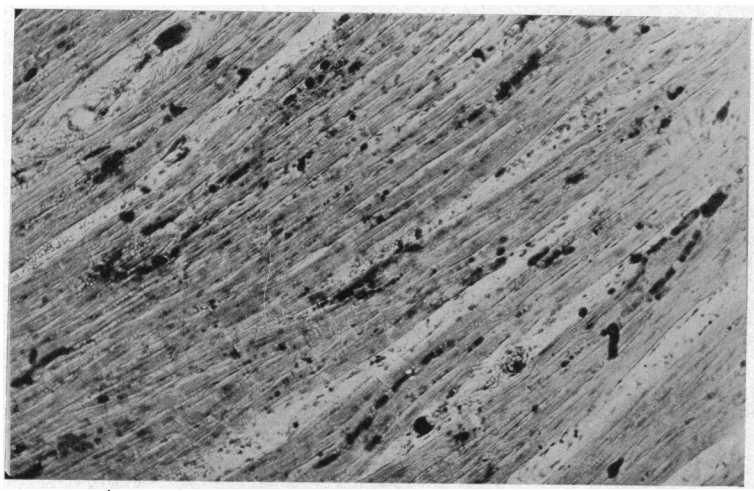

FIG. 1.—Case I. Heart. Scharlach R. and hæmatoxylin, showing numerous fat emboli, congestion and interstitial œdema.

Fat gains access to the circulation mainly through ruptured blood-vessels, but it is also probable that a small amount of fat may reach the circulation through the lymphatics by way of the thoracic duct. In the great majority of instances it is probable that the amount of fat entering the circulation is small and the localization not serious, so that few or no symptoms may ensue and no suspicion of the condition be had. In a smaller number of cases, but probably far more frequently than is generally recognized, fat emboli do produce symptoms which are usually attributed to other

* Read before the American Surgical Association, May 5, 1925.

causes, and in a still smaller number these symptoms are of great severity, and death ensues. Comparatively few correct clinical diagnoses of fat embolism have been made, and the pathologists, too, have undoubtedly overlooked the condition in many instances.

The object of this paper is to call to the attention of the surgical profession a condition of great importance, and in our judgment of relatively frequent occurrence, and to present the clinical picture and the pathological findings in two cases which have been carefully studied. Historically, much has been written about fat embolism, chiefly by the German pathologists beginning with Virchow in his classical work on embolism in 1862. Zenker, in 1862, observed the first case of fat embolism of the pulmonary capillaries in man, and during the succeeding years many articles were written and much experimental work done, chiefly on the continent. During the past few years certain American writers have given especial attention to this subject. The most complete survey of the literature, together with the most exhaustive study of the condition made up to that time was the article published by Warthin in 1913 in the International Clinics. To Warthin's summarized observations and conclusions practically nothing of importance has since been added. It is a striking fact that this condition seems to have been almost entirely overlooked by American surgeons, and the references to this condition in American surgical literature are singularly lacking and inaccurate.

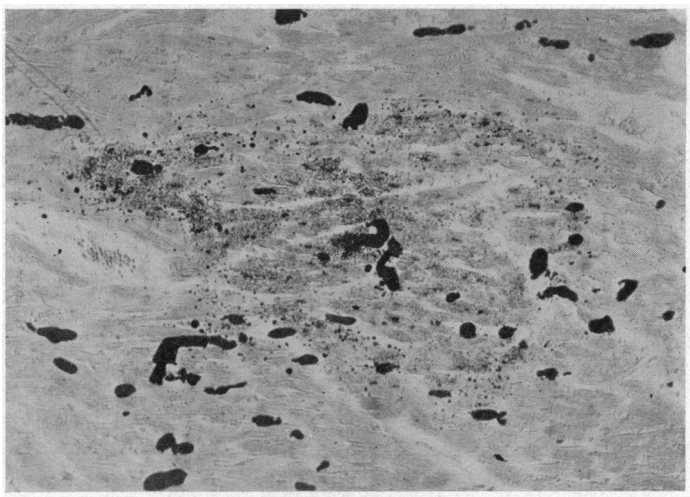

FIG. 2.—Case I. Heart. Osmic acid preparation. Colony of fat emboli with muscle fibres undergoing fatty degeneration.

CASE I.—J. M. H., aged twenty, a strong, healthy, athletic young man, whose past history was negative, was riding on the front seat of an automobile beside the driver at about 3 A.M., October 12, 1924, when the car crashed into a telephone pole, and the patient sustained closed fractures of both femora in the middle thirds. He was transported with all possible care to the Albany Hospital, where he was admitted to the surgical service about two hours after the accident. Retentive dressings were applied with the minimum of manipulation and the patient given a small dose of morphine. The patient was carefully examined by Doctor Elting at 10 A.M., at which time the temperature was normal, the respirations 20, and the pulse 100. There was considerable swelling and ecchymosis of both thighs, but no manipulation was indulged in. There was

some evidence of contusion over the lower abdomen in the region of the symphysis pubis, but no definite fracture could be demonstrated. There were no abrasions of any part of the body.

The patient seemed perfectly normal mentally, conversed rationally and expressed himself as feeling quite comfortable. There was no evidence whatever of shock and no disquieting symptoms. The patient's condition remained about the same until 3 P.M., when he began to complain of increased pain and became quite restless. He was given a small dose of morphine, which lessened the complaints and restlessness somewhat, but soon after this he became quite excited and irrational, which state in a few hours was followed by the onset of stupor. During the night this developed into a deep coma, from which the patient could not be aroused. Coincident with this was a rise in temperature and an increase in the pulse and respiratory rates.

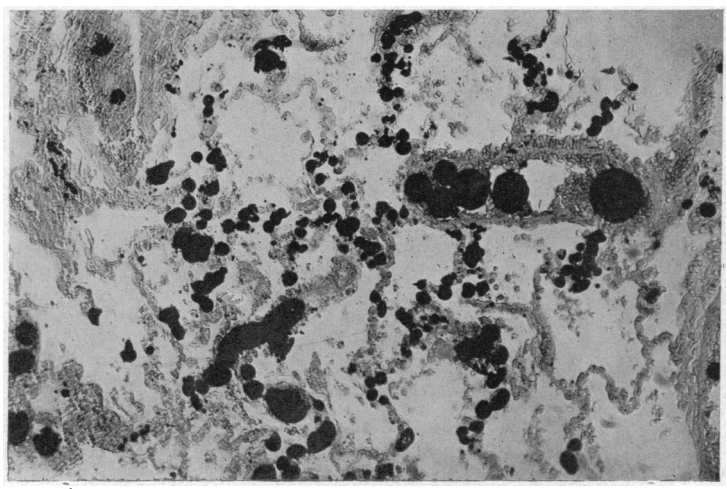

FIG. 3.—Case I. Lung. Osmic acid preparation. Fat emboli lodged in most of the smaller vessels.

When seen at 10 A. M., October 13, the patient was quite comatose with typical Cheyne-Stokes respiration. The temperature was 103, pulse 140, and respirations 28. Cyanosis and dyspnœa were pronounced. A lumbar puncture was done, and 22 c.cm. of normal spinal fluid under normal pressure was withdrawn. A diagnosis of cerebral fat embolism was made by Doctor Elting, and the patient was seen by Doctor Archambault, whose neurological note was as follows: "The patient is profoundly comatose, and no response occurs to any form of sensory stimulation. The upper extremities are flaccid and inert, but the tendon reflexes are present and of symmetrical amplitude. The eyeballs occupy the midplane. There is no strabismus, and the pupils moderately contracted are equal and reactionless. The eye grounds show perfectly normal findings. The breathing is of the typical Cheyne-Stokes type."

The patient remained in a comatose state with temperature rising to 105°, pulse 150, and respirations 30, until 7.30 P.M., when he died, approximately 40 hours after the accident. A urinary examination was negative, but unfortunately no special examination for the presence of fat in the urine was made, nor was the sputum examined for the presence of phagocytic cells containing fat. No skin petechiæ were observed during life.

Autopsy performed by Drs. L. J. Early and C. E. Martin, one and one-half hours post-mortem.

The body is that of a young, white male adult, 178 cm. long. Both femurs are fractured in the upper portion of the lower third, and the thighs are considerably swollen and contused. There are contusions and abrasions over the chest, buttocks and legs. Petechial hemorrhages are noted on the chest, arms, thighs and back.

The pleural cavities contain no fluid. The lungs are firm and do not collapse. The pleural surfaces are dotted with petechial hemorrhages, up to 6 mm. in diameter. Both lungs are extremely œdematous, and from the cut surface a thin, blood-stained fluid

drips. In either lung are several irregular hemorrhagic areas, 1 to 2 cm. in diameter and having a lobular distribution.

The pericardial cavity contains 50 c.cm. of a clear yellow fluid. Scattered over the epicardial and endocardial surfaces, but most abundant in the right ventricle, are numerous petechiæ 1 to 2 mm. in diameter. They also occur in the myocardium, where they are often surrounded by a narrow, yellow, opaque zone.

In the pelvis is an extensive subperitoneal hæmatoma. It covers the superior surface of the bladder and continues up over the brim of the pelvis well into the tissues of the posterior abdominal wall. The hæmatoma is most marked over the superior ramus of the right pubis, which is fractured. The bone is fragmented, dislocated, and the edges much roughened.

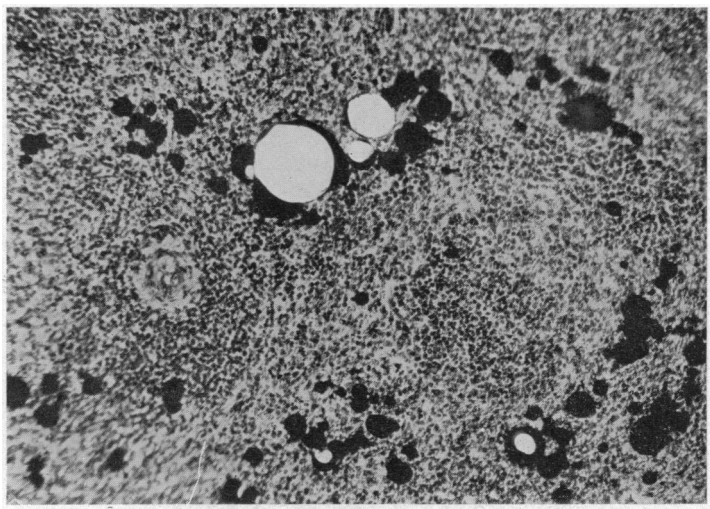

FIG. 4.—Case I. Spleen. Scharlach R. and hæmatoxylin, showing tendency for the emboli to group themselves about the periphery of the Malpighian bodies.

Pin-point hemorrhages scattered throughout the mucosa of the entire intestinal tract. In caecum are numerous streaky mucosal hemorrhages.

The pancreas is normal in the gross. Near the tail of the pancreas is a lymph-node, 1 cm. in diameter, showing much central caseation. The liver weighs 1780 gms. The cut surface is opaque, and the markings are indistinct. The gall-bladder and ducts are normal. The weight of each kidney is 150 gms. The vessels at the junction of the cortex and medulla are injected. The adrenals appear normal. In the mucosa of the bladder, mostly about the neck, are numerous small hemorrhages.

Brain: The weight of the brain is 1580 gms. There is the slightest suggestion of a cerebellar pressure cone, and the uncus projects into the interpeduncular fossa somewhat more prominently than normal. There is no other gross evidence of œdema, and no hemorrhages are seen.

Microscopic Examination.—Heart: There are numerous small areas of hemorrhage in which large numbers of fat emboli may be seen. The emboli often appear as long cords, and their diameter varies with that of the vessel in which they are lodged. The cells surrounding small groups of emboli often show considerable fatty degeneration. Phagocytic cells are not noted in such areas. Interstitial œdema and congestion are extreme. These changes are most marked in the left ventricle.

Lungs: Fat emboli are exceedingly numerous in the lungs. The alveolar walls are congested and most of the capillaries are stuffed with fat. A small amount of free fat is present in the alveoli. The alveoli are, in most instances, filled with transudate, but there is also considerable hemorrhage. There is a small amount of hæmosiderin in the alveoli, some of which has been phagocyted. There is an acute bronchiolitis and also small patches of early bronchopneumonia. The exudate consists almost entirely of polymorphonuclear leucocytes, a small amount of fibrin and a few macrophages.

Spleen: Congestion and hemorrhage are extreme. Fat emboli, which are scattered throughout are, however, most numerous and largest about the periphery of the lymphoid follicles.

Gastro-intestinal tract: There is œdema and congestion of the submucosa in every portion of the intestinal tract. Fat emboli are most often seen in the small capillaries of the mucosa. The cells of the gastric mucosa are secreting a large amount of mucus, much of which has been deposited on the surface. There is an occasional small area of submucosal hemorrhage.

Pancreas: There is marked interstitial œdema. The islets of Langerhans are congested, swollen, and in many the capillaries are stuffed with fat.

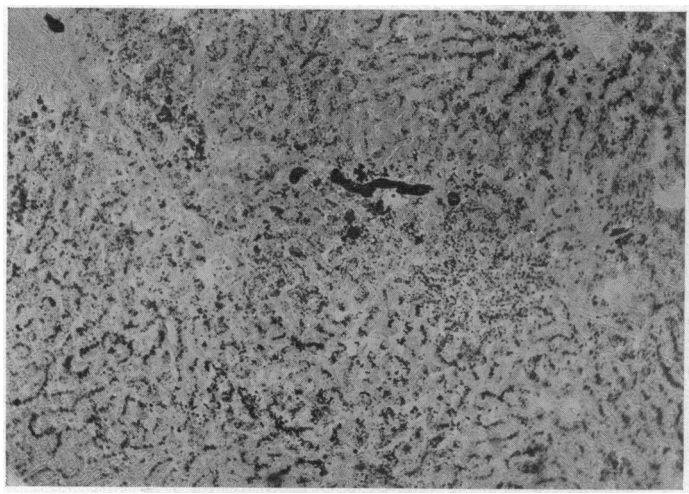

FIG. 5.—Case I. Liver. Osmic acid preparation. A few emboli and marked fatty metamorphosis, which is most marked along the bile capillaries.

Liver: Congestion is extreme and most marked about the central veins. Small droplets of fat are abundant in the liver cells along the bile capillaries. Fat emboli are numerous and most often seen in the capillaries of the periportal spaces.

Kidneys: The glomerular capillaries contain long cords of fat and are distended with red blood-cells. There is cloudy swelling of convoluted tubules. There are many small areas of hemorrhage, particularly in the pyramids. Many fat emboli are lodged in the extra-tubular capillaries.

Adrenals: The adrenal vein contains numerous variously sized fat droplets. Similar emboli are found in the cortex, the cells of which show fatty degeneration. The affected areas in the cortex appear to be the result, at least in some instances, of the plugging of a single vessel of the end artery type.

Bladder: There is a large amount of hemorrhage into the retro-peritoneal connective tissue. In the submucosa and muscularis, œdema and congestion are quite marked. Fat emboli are frequently seen in the mucosal vessels.

Genitalia: Spermatozoa are present in the seminal vesicles and emboli are occasionally noted in the mucosa.

Lymph-nodes: The vessels in the substance of the lymph-nodes are extremely engorged. Fat emboli are numerous and most often located immediately beneath the capsule in the peripheral lymph sinuses. The node removed from near the tail of the pancreas shows tuberculosis.

Brain: Sections from the frontal, temporal, parietal and occipital lobes, motor area, motor speech area, basal ganglia, corpus callosum, optic chiasm, pons, region about the Sylvian aqueduct, floor of the fourth ventricle, various levels of the bulb, upper cord and cerebellum are examined. Emboli and areas of focal necrosis are found in every section. One particularly large area is found in the hypoglossal nucleus. In most instances there is vacuolization, destruction of myelin and fragmentation of the axons. The cells are shrunken and pycnotic. Fat emboli are numerous in and about

these areas. There is little or no phagocytosis. Areas of degeneration are seldom seen in the fibre tracts. The remainder of the brain tissue is extremely œdematous.

CASE II.—W. L., aged twenty-one, was a healthy young man of good habits. At 2 A.M., on April 29, 1923, he fell from a window, a distance of 30 feet, and struck on the pavement, sustaining a closed fracture of the left hip and a compound comminuted fracture of the right humerus. He was removed at once to the Troy Hospital, where he was seen by Doctor Elting at 10 A.M. The fractures were carefully immobilized and the patient removed to the Albany Hospital, where he was admitted to the surgical service at 12 noon on May 1. At that time he appeared to be somewhat pale and shocked but quite rational. The temperature was 98.8°, pulse 120, and respirations 23. The patient was placed in a fracture bed and extension applied to the left leg. The right upper arm presented an irregular wound about 10 cms. in length, situated on the external surface at about the mid-region of the arm and just over the site of fracture of the humerus. Wet antiseptic dressings were applied and the arm immobilized. There were numerous contusions of the chest and abdomen, but none of the head. The patient was restless and uncomfortable, but his condition remained fairly satisfactory. The temperature was practically normal for three days, and the pulse ranged from 90 to 100. The respirations were about 25, and there were no cerebral, cardiac or respiratory symptoms of any significance. For the first two days the patient was restless and difficult to manage at times, but on the third day the general condition was much improved. He was less restless, quite rational, and sleep was more natural. Early on the fourth day the temperature rose in a few hours to 104°, the pulse to 140, and the respirations to 35. The patient was at first restless and delirious, but later in the day he became stuporous and during the night lapsed into coma. When seen on the morning of the fifth day the temperature was 105°, the pulse 150, and the respirations 40. He was quite comatose, with Cheyne-Stokes respiration. The reflexes were abolished. The breathing was labored. Cyanosis was pronounced, and there was evident pulmonary œdema. The pulse was weak and the arterial pressure low. The eye grounds were negative. The wound on the right arm was unchanged except for a slight amount of sero-purulent discharge. Cultures from this wound made on the fourth day showed a hæmolytic streptococcus and a hæmolytic staphylococcus aureus. Blood cultures made on the fourth day were negative. Urine examination showed some albumin, a few granular casts and a few red blood-cells. Unfortunately no special examination was made for free fat in the urine. The temperature, pulse and respiration remained elevated. The coma continued, and the patient died on the evening of the fifth day, approximately four and three-fourths days after the accident.

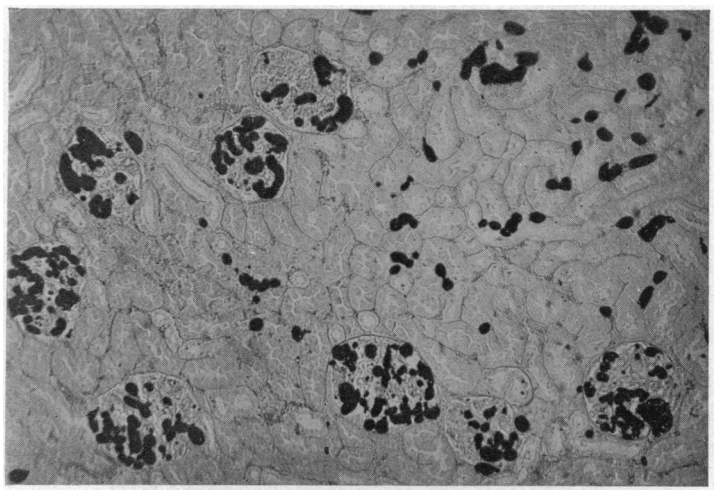

FIG. 6.—Case I. Kidney. Osmic acid preparation. Fat emboli in the glomeruli and extra-tubular capillaries.

Autopsy performed by Drs. V. C. Jacobson and L. Sutton, one and one-half hours post-mortem.

The body is that of a well-developed and well-nourished young, white, male adult, 183 cm. in length. There are numerous petechial hemorrhages in the skin and conjunctivæ. These hemorrhages are most marked over the anterior abdominal wall, chest and arms.

Over the outer aspect of the right arm is a laceration 3 x 1 cm. It is several centimetres in depth, and from it a blood-stained, turbid fluid exudes. The tissues for several centimetres about are œdematous and indurated. A comminuted fracture of the humerus, with much crepitation, can be easily felt just beneath the surface. Numerous blebs with a reddish base are scattered over the outer surface of the lower half of the arm. The circumference of the right arm is 30 cm. and of the left arm 25 cm.

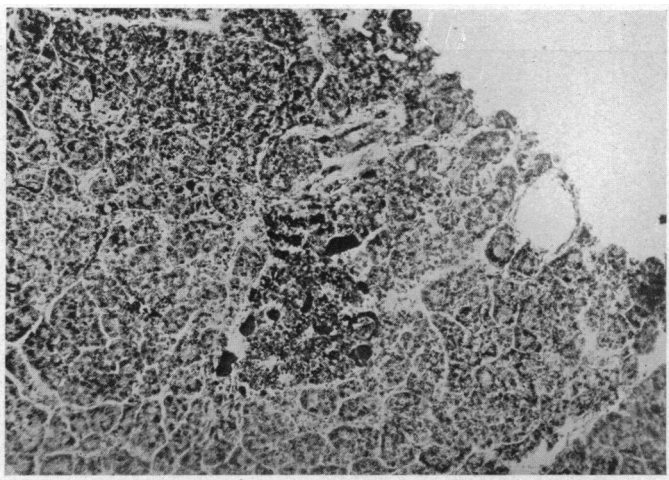

FIG. 7.—Case I. Pancreas. Scharlach R. and hæmatoxylin. In centre is an islet of Langerhans, swollen, congested and containing fat emboli.

The left femur is fractured at about its middle. Measuring from the anterior superior spine of the ileum to the internal malleolus, the left leg is 91 cm. and the right leg 99 cm. in length. Beginning 4 cm. below the right iliac crest is a large area of ecchymosis and yellowish discoloration 29 x 14 cm. There are various ecchymoses of the right knee, right leg and left leg.

An extensive hemorrhage in the pelvis covers the bladder, sigmoid and psoas muscles. There is about 10 c.c. of free blood in the pelvis. In the anterior abdominal wall is a diffuse retro-peritoneal hemorrhage and the peritoneum shows a few small lacerations. The mesenteric lymph-nodes are enlarged, some of them being 2½ cm. in diameter.

The left pleural cavity contains 50 c.c. of thin, reddish-brown fluid and the right cavity 10 c.c. of blood. The pericardial cavity and heart are apparently normal.

The lungs are acutely congested throughout, but more so in the lower lobes. Projecting above the surface of the lower right lobe is a bleb 3 cm. in diameter, filled with blood. Within the substance of the lung are several similar pools of blood. There are no large hemorrhages in the left lung, but digestion of considerable tissue about the hilum has occurred from a perforation in the œsophagus. Many of the bronchioles contain a mucopurulent material.

The spleen (370 gms.) is grayish-red, firm, and the lymphoid follicles prominent. The lower two-thirds of the œsophagus is thin and necrotic. An agonal perforation which communicates with the mediastinum and left pleural cavity has occurred in the middle third.

The liver weighs 2150 gms. The cut surface is opaque. In the right lobe are several linear fractures, 4 to 6 cm. in length, filled with clotted blood. The surrounding tissue is necrotic. The kidneys are congested. The adrenals, bladder, genitalia and aorta appear normal.

The brain is congested and œdematous. There are pin-point hemorrhages diffusely distributed throughout the gray and white matter of the cerebrum, cerebellum, pons and basal ganglia.

Microscopic Examination.—Heart: Fat emboli are scattered throughout the myocardium. In many instances they form small groups about which the muscle fibres have undergone fatty degeneration. Rarely is there any phagocytosis. There is congestion and interstitial œdema. Leucocytic thrombi are present in several of the coronary vessels and necrosis of the adjacent muscle fibres has occurred.

Lungs: Massive hemorrhage has disrupted the lung tissue, the blood filling the alveoli and bronchi. Fat emboli are exceedingly numerous, being located principally in the alveolar capillaries. There is a small amount of free fat in the alveoli. The alveolar walls are intact. Hæmosiderin-laden phagocytes are numerous. There is a slight purulent bronchitis and polymorphonuclear exudate about some of the hemorrhages.

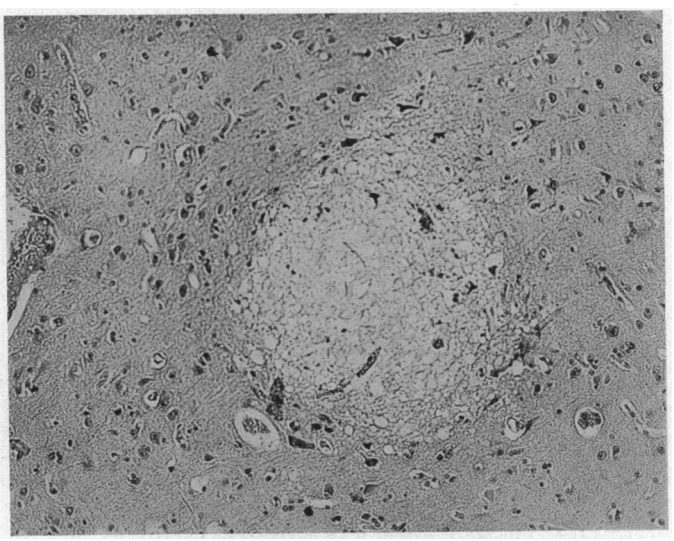

FIG. 8.—Case I. Frontal lobe. Hæmatoxylin and eosin. Focal necrosis with vacuolization and rarefaction of the area. The cells are shrunken and pycnotic. There is no phagocytosis.

Spleen: There is an acute splenitis of the hyperæmic type. Fat emboli are numerous and located chiefly in or about the lymphoid follicles.

Liver: Fat emboli are numerous in the small vessels of the periportal spaces. Fine fat droplets are abundant in the liver cells along the bile capillaries. There are large areas of necrosis, sometimes involving whole lobules or the periportal portions, but mostly of irregular size and shape. Phagocytosis by polymorphonuclear leucocytes is in progress in small foci. Rents in the liver-tissue, probably from trauma, are filled with blood.

Pancreas: Fat emboli are most numerous in the Islets of Langerhans. There is slight œdema and congestion.

Gastro-intestinal Tract: Fat emboli are most often seen in the capillaries of the submucosa. The mucosa of the duodenum shows a mild acute inflammation.

Kidney: Fat emboli are abundant in the glomeruli, which are swollen, congested and often contain an excess of polymorphonuclear leucocytes. There is an acute tubular nephritis and interstitial leucocytic infiltration is conspicuous in many places in the medulla and cortex.

Adrenal: The cortex has undergone considerable fatty degeneration with necrosis and disappearance of many cells. There are many emboli, some of large size, principally located deep in the cortex.

Skin of Arm: There is a purulent inflammation of the skin and subcutaneum, with tracts of exudate dissecting between the muscle and fascia. Fat emboli are unusually abundant, some large vessels being completely occluded.

Brain: Several sections of the cerebral cortex, cerebellum, pons and bulb are examined. In all sections fat emboli are present. There are many small foci of necrosis. Some foci show simply rarefactions with destruction of myelin and pycnosis of the cells. In other areas hemorrhage alone is most prominent. Some foci of necrosis are surrounded by a rim of hemorrhage. Phagocytes are not common. Often the centre of a

necrosed area is occupied by a vessel plugged with fat. Emboli are less prominent in the fibre tracts. Several small hemorrhages are noted in the floor of the fourth ventricle and in the cerebellum.

NOTE.—Shortly before death, cultures of the material from the wound in the right arm showed the presence of hæmolytic streptococcus and hæmolytic staphylococcus aureus. The autopsy findings were regarded as typical of streptococcus septicæmia, although unfortunately a blood culture was not made after death. The focal lesions so widely distributed in the body, the bronchopneumonia with hemorrhage and the bacteriological findings in the arm wound seemed sufficient for a diagnosis of probable septicæmia.

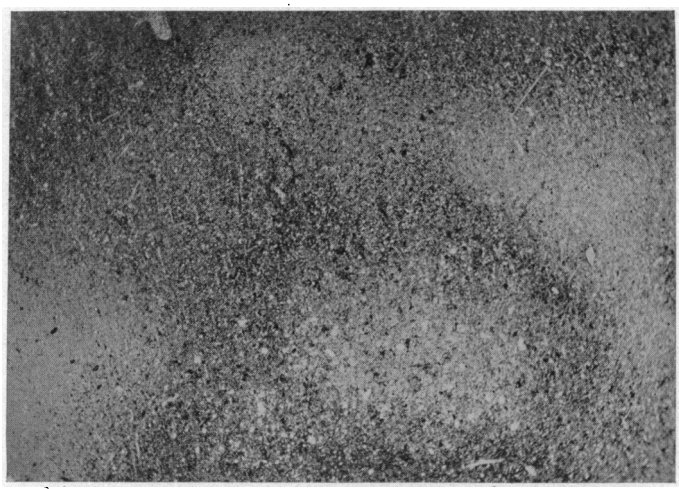

FIG. 9.—Case I. Motor area. Osmic acid preparation. Semi-confluent foci of necrosis.

After Case I was revealed as one of fatal fat embolism, Case II was studied from the same angle and proved to be one of fat embolism, the focal lesions being not due to bacterial emboli, but to occluding fat droplets.

There has been a remarkable tendency to regard cases of fat embolism as cases of shock, and it would appear that this term has borne the brunt of the incorrect diagnoses. W. T. Porter, of Boston, in 1917, both asserted and demonstrated that the so-called shock following war wounds of the long bones was in the great majority of instances due to fat embolism. His experiments conclusively proved that all of the symptoms of shock could be readily caused by fat embolism of the lower structures of the brain, the clinical and pathological pictures presented by his experimental animals being exactly the counterpart of those observed in the human subject. Porter further demonstrated in 1919, that the injection of a very small amount of olive oil into the vertebral artery of animals would cause fat embolism of the vaso-motor centre, thus bringing about vaso-motor paralysis and shock. Porter even went so far as to declare that fat embolism is the most frequent cause of wound shock upon the battlefield. The amount of fat which may be found in the capillaries is at times astounding, and it is difficult to conceive of where all the fat comes from. There is, furthermore, no definite relationship between the amount of trauma and the extent of the fat embolism. As a rule the trauma is rather severe, but in some instances it is comparatively trivial.

It is not altogether uncommon for surgeons to lose cases of fractures of the long bones a few days after the accident when everything appeared to

be going well. The temperature, pulse and respiration begin to rise. The patient becomes at first restless, then perhaps delirious, then comatose, and death ensues. The surgeon usually regards these as cases of shock, toxæmia, infection or concussion, but they are practically always cases of fat embolism.

It is probable that fat embolism in the absence of infection is the most common cause of death after fracture of the long bones.

Warthin has especially emphasized the medico-legal importance of fat embolism after injuries in which the usual diagnosis by the coroner's physician is shock, apoplexy, alcoholism, concussion, heart disease, etc. It may be positively stated that fat embolism occurs only during life, and its presence in the body after death is presumptive, and in the majority of cases, positive evidence of trauma before death.

Fat emboli are bland, and their effects are purely mechanical. For their production three conditions must be fulfilled:

1. The fat must be liberated from the fat cell.

2. It must be liquid and accessible to the circulation.

3. There must be some force to drive it into the blood-vessels.

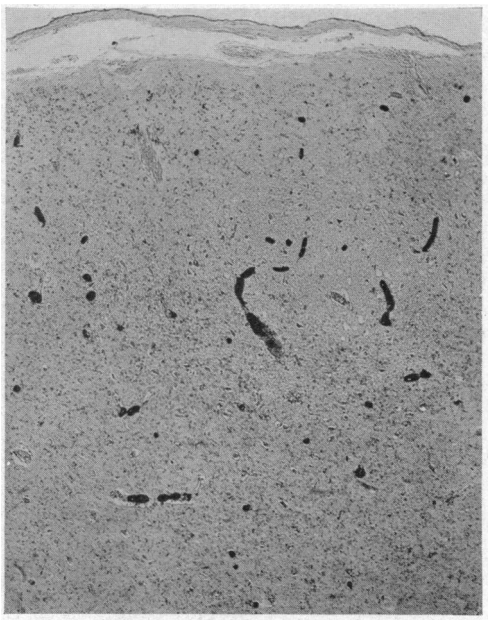

FIG. 10.—Case I. Occipital lobe cortex. Scharlach R. and hæmatoxylin. Numerous fat emboli. An area of focal necrosis is surrounded by several large emboli.

Trauma and the accompanying hemorrhage, with at times infection, easily bring about the liberation of fat from the cell. The second condition is met by the patent vessels in the bone-marrow, and Haversian canals, which show much less tendency to close after trauma than do other vessels. Hemorrhage, œdema, inflammation, manipulation or tight bandages may readily supply the increased pressure to force the free fat into the vessels. The amount of fat which may be, and in fatal cases usually is, deposited in the capillaries is apparently out of proportion to what would be regarded as the normal amount of fat in the traumatized area. Some observers have thought there must be some other source for the free fat, but none has as yet been demonstrated.

In an effort to determine whether or not the fat content of the organs was actually increased, the lungs and spleen of the two cases of fat embolism and the same organs of five other diseased states were extracted. The lung was chosen because most of the fat must at some time pass through or lodge

in it. As the spleen may be considered a filter in the systemic circulation, it was also extracted.

The tissue was first dehydrated over concentrated sulphuric acid, then powdered, weighed and mixed with fat-free plaster-of-Paris. Using sulphuric ether as solvent, the fat was extracted by means of the Soxhlet apparatus. Extraction was continued for at least 20 hours in every case. After extraction the filtrate was diluted to a known volume [usually 100 c.c.] and 10 c.c. placed in a weighed crucible. The ether was then evaporated and the crucible again weighed. From this the fat content of the whole and the percentage composition of the dehydrated specimen was computed. The following are the estimations.

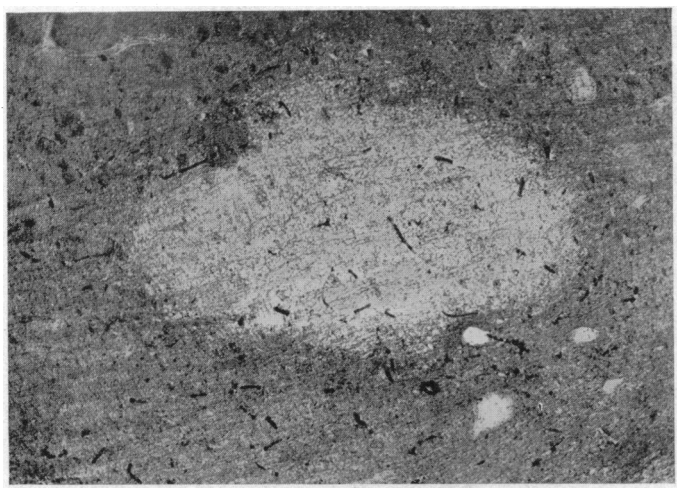

FIG. 11.—Case I. Section lateral to the Sylvian aqueduct at the level of the third nerve. Scharlach R. and hæmatoxylin. Large focal necrosis with many emboli in and about it.

Disease	Lungs	Spleen
Fat embolism [Case I]	45.5%	52.5%
Fat embolism [Case II]	35.4%	29.1%
Extensive superficial burns [lye]	3.5%	3.4%
Acute alcoholic poisoning	11.7%	8.7%
Fracture of skull with maceration of brain	13.8%	12.1%
Peritonitis following bullet wound	9.1%	23.8%
Peritonitis following perforation of jejunum	8.1%	19.0%
Normal content [Wells]	17.3%	14.2%

This, so far as we are able to learn, is the first actual demonstration of the quantitative increase of fat in the organs of cases dead of fat embolism and confirms the microscopical findings. What is true of the lungs and spleen will naturally hold true of the other organs.

Welch states that fat embolism is the commonest form of embolism. It is our feeling that all fractures involving the fatty marrow of bones are probably associated with some degree of fat embolism, but that in the great majority of cases the embolism is so slight that no symptoms are produced, while in a lesser number of cases true symptoms do result which are usually incorrectly interpreted.

In all of these cases recovery takes place, and in only a few of them may it be possible to prove the diagnosis. In the cases that result fatally,

however, a carefully performed autopsy will always demonstrate a most striking and characteristic picture and one that cannot possibly be confused with any other known pathological condition.

Fat embolism may be caused—

1. By all forms of trauma affecting the marrow of bones, especially fractures.

2. All kinds of operations upon bones, especially the so-called orthopædic operations.

3. Trauma, inflammation, necrosis or operation upon fatty tissue in any part of the body.

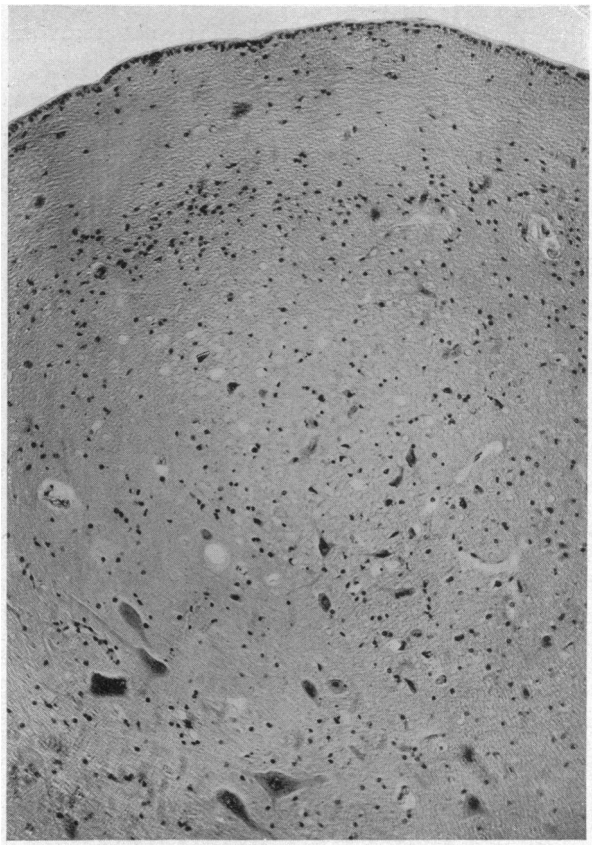

FIG. 12.—Case I. Floor of the fourth ventricle. Hæmatoxylin and eosin. Large area of rarefaction occurring in the nucleus of the hypoglossal nerve. The ganglion cells show various stages of degeneration.

Fat embolism resulting from other causes is rare and of pathological interest only, as there are rarely any definite symptoms.

The vast majority of cases of fat embolism are due to trauma of the bone and are most frequently observed after injury to the bones containing the greatest amount of fatty marrow, as the femur, tibia and humerus. The osteoporotic bones of the aged and the atrophic bones of any age, containing more than the normal amount of fat, are naturally of etiological importance, but it is especially apt to occur after serious injury to the long bones of the healthy young adult, particularly in the third decade. The fat may pass into the blood stream rapidly and in sufficient quantity to produce symptoms almost immediately, although in the majority of instances the accumulation of the fat in the capillaries is a slower process, and the symptoms do not appear for some hours, it may be several days after the trauma. Tight bandages, manipulation, movement and jarring are factors which may favor fat embolism.

The oft-repeated and generally accepted observations of the lessened shock observed in the late war, as the splinting and transportation of cases

of fracture became more expert, skilful and expeditious, furnish in our judgment not only clinical evidence of the importance of fat embolism as a cause of the so-called shock in these cases, but also striking evidence of the importance of prophylaxis in possible cases of fat embolism. There can be little doubt that many cases of death from so-called post-operative or traumatic shock are cases of fat embolism, for unless autopsies are conducted with this possibility in view, many, if not most cases, of fat embolism will be overlooked.

The manner in which fat droplets produce embolism has been much discussed. Gauss, by adding olive oil to citrated human blood, claims to have increased its viscosity approximately four times. This was confirmed by Bissell. In attempting to repeat these experiments we were at once confronted with the extreme difficulty of keeping the fat droplets in the emulsion of sufficient size to form emboli in a capillary tube. When they were of large size and could be induced to enter the capillary tube, the findings agreed with those of Gauss and Bissell, and even when a single droplet of fat was made to enter a capillary tube ahead of a column of normal salt solution, the time for the passage was increased to the same degree as was originally found. Obviously the apparent increase in viscosity was due, not to the increased viscosity of the whole mixture, but to the presence of the fat alone. In an effort to make the fat remain emulsified, it was shaken in a mechanical shaker, and to aid in the emulsification ground glass was added. When this was done and the viscosity tested it was found not to be increased over that of the salt solution without fat. In this case the emulsion was very fine and did not settle out.

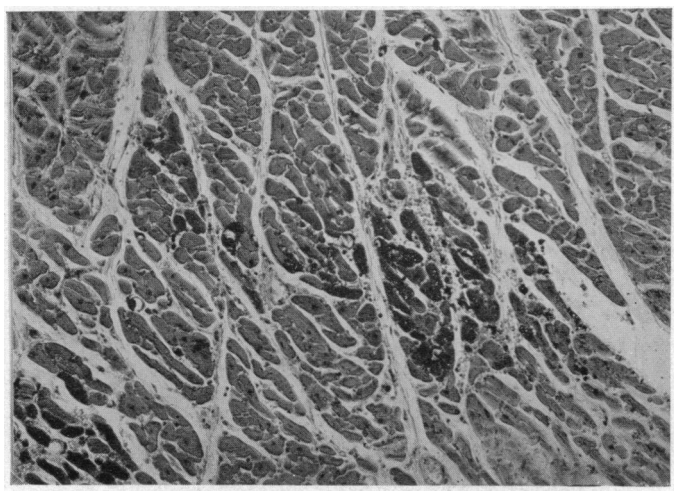

FIG. 13.—Case II. Heart. Scharlach R. and hæmatoxylin. Area in centre undergoing fatty degeneration, several emboli, œdema and congestion.

The latter finding is, of course, not new and only serves to bear out what has long been known in lipæmia, as of diabetes, where there is an extremely fine emulsion of fat and blood and in which fat embolism does not occur. So it is that from these experiments we believe that while the progress of fat emboli through a capillary may be slow or even halted, it is due to the greater viscosity of the fat alone, rather than to the increased

viscosity of the whole fluid. These experiments were performed both with olive oil and human fat. Olive oil has a somewhat greater viscosity than human fat.

The characteristic lesions of fat embolism are widespread throughout the body, and a knowledge of the pathology is absolutely essential for a proper interpretation of the clinical phenomena. At the site of the injury or operation liquid fat is found often mixed with blood or pus. This fat enters the more or less patent veins, especially those of injured bones, aided by the increased tension at the site of the injury. In addition to the fat which enters the blood-vessels, fat may also enter the lymphatics, pass through the regional lymph-glands, and reach the venous circulation through thoracic duct.

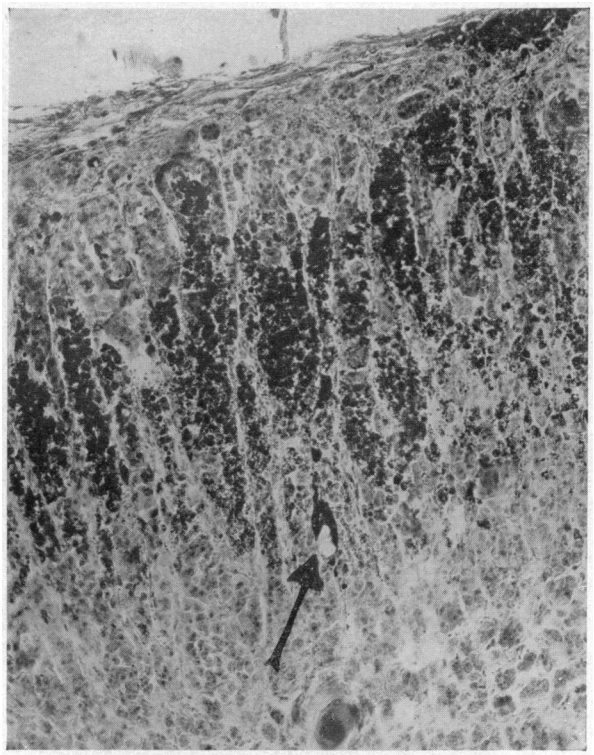

FIG. 14.—Case II. Adrenal. Scharlach R. and hæmatoxylin. Extensive fatty degeneration of the cortical cells. The arrow points to a large embolus.

The lesions which occur depend upon the amount and the distribution of the fat which enters the circulation. The fat appears to be first arrested chiefly in the pulmonary capillaries, and in many instances it may go no further, but as the fat increases in pulmonary capillaries it passes into general circulation, then it is that a widespread distribution takes place; and there are no organs or tissues which may not show fatty embolism.

It is probable that a considerable amount of fat can be tolerated in the capillaries without necessarily producing serious lesions. If the fat enters the heart in large amounts, the result may be very similar to that observed in air embolism. The heart action may not be sufficient to drive the fat into the lungs, and serious embarrassment of the heart and death may ensue. The heart action and function are also seriously and even irreparably impaired by myriads of capillary fat emboli in the terminals of the coronary arteries, causing large numbers of small cardiac infarcts, and it is probable that most of the cardiac symptoms are due to this condition. Liquid fat may be observed in the right heart, and numerous petechial hemorrhages occur

beneath the epicardium and endocardium, surrounded in some instances by a small rim of light yellow discoloration. Microscopically, the emboli seem to group themselves in colonies, and around these emboli the heart muscle shows fatty degeneration and anæmic infarcts.

The pulmonary capillaries receive by far the largest number of fat emboli. The lungs at autopsy are rather firm, and a thin blood-stained fluid drips from the cut surfaces, while fat droplets may be seen in the blood-vessels. Œdema, congestion, hemorrhage and rarely hemorrhagic infarction are seen. Subpleural hemorrhages are often observed. Microscopically the lungs present in addition, when stained with Sharlach R or osmic acid, capillaries greatly dilated and filled with fat, which may be in a globular or confluent form.

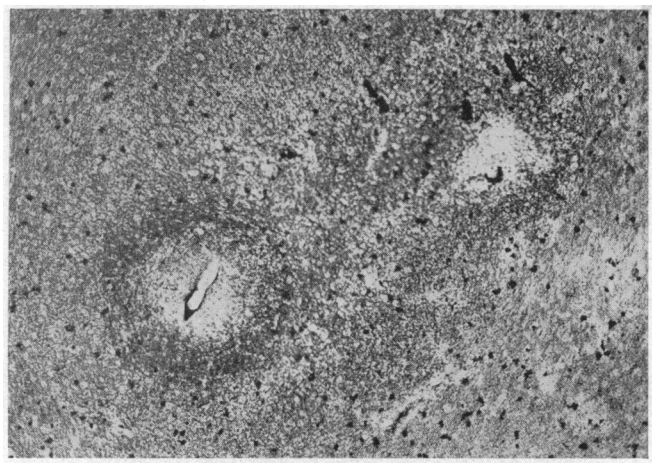

FIG. 15.—Case II. Cerebral cortex. Scharlach R. and hæmatoxylin. Two areas of focal necrosis, each containing a fat embolus.

The brain in the gross usually is the picture of cerebral œdema. Scattered miliary hemorrhages are evident on the surface, as well as throughout the substance of the brain. Microscopically, minute hemorrhages are found around small vessels filled with fat. The surrounding brain tissue shows more or less pronounced degeneration characteristic of focal necrosis or infarction.

These lesions adequately explain the early cerebral irritation followed by the paralysis so characteristic of the cerebral syndrome.

The spinal cord presents similar, though less striking lesions.

The liver shows passive congestion due to the heart impairment, and there are minute areas of focal necrosis. Fat emboli may be observed in the capillaries surrounded by areas of cell degeneration.

The spleen is usually swollen and congested. The fat emboli are particularly abundant and tend to group themselves around the Malpighian bodies.

The kidneys are congested. The fat emboli are found especially in the glomerular capillaries, and are much less evident in the vessels about the tubules. There are often fatty casts in the tubules. Small hemorrhages may be present, but degeneration of the renal cells is usually slight.

The petechiæ observed in the skin are the result of minute hemorrhages around capillaries filled with fat.

The pancreas shows numerous fat emboli, chiefly in the capillaries of the islands of Langerhans.

The adrenals show many emboli in the capillaries of the cortex, with marked degeneration of the surrounding cells.

The thyroid, striated muscles, and in fact all the other tissues show more or less pronounced capillary fat embolism.

The excretion of the fat is largely through the kidneys, and numerous clinicians have noted its presence in the urine. Fat is also excreted through the activity of the phagocytes, and as Warthin first emphasized, many of these fat-laden phagocytes appear in the sputum, a diagnostic point to which he called especial attention. Through the intestine, bile and other channels the fat-laden phagocytes may make their escape. A very small amount of the fat may be taken up by certain of the fixed cells of the body, and it is probable that a small portion of it is also disposed of through the processes of oxidation and saponification.

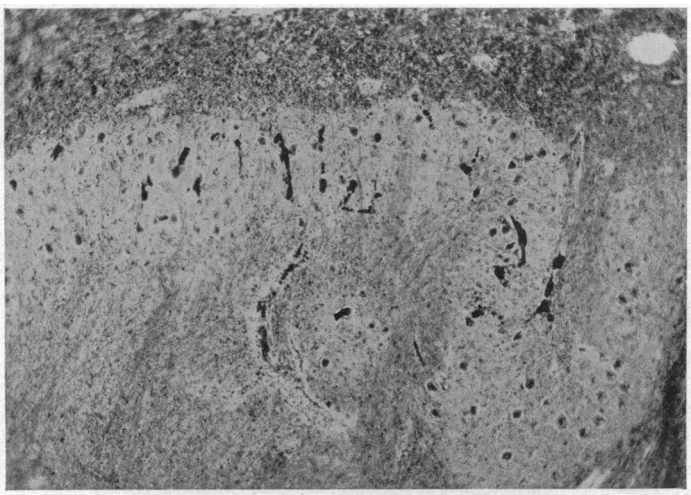

FIG. 16.—Case II. Inferior olivary body. Scharlach R. and hæmatoxylin. Capillaries of the convolutions containing numerous emboli.

A study of the clinical features of fatal cases of fat embolism shows the striking uniformity with which certain symptoms appear. There seems to be a fairly well defined clinical picture and one which should be recognized. Following a trauma most commonly associated with fracture of a long bone, or an operation upon the bones or fatty tissues, there is usually a period of well-being during which the patient's condition is fairly satisfactory. This euphoria may last from a few hours to a week or more.

As a general rule the symptoms due to fat embolism are first observed on the second or third day following the trauma or operation, although they may occur within a few hours or be deferred for several days. As a rule the earlier the symptoms present, the more serious the case, and the more certain the fatal termination.

The symptoms may be classified into two general clinical syndromes, although these merge into one as the fatal termination is approached: (1) The cardio-respiratory syndrome, and (2) the cerebral syndrome. Some writers attempt to differentiate a cardiac from a pulmonary syndrome, but we believe from a study of the clinical phenomena and the pathological findings that they should be classed as one symptom complex in which, unlike

most writers, we regard the cardiac phenomena of more importance as a rule than the pulmonary. The chief symptoms are dyspnœa, cough, cyanosis, pulmonary œdema, occasional pulmonary hemorrhage, rapid and irregular pulse with a low arterial and high venous pressure. There may be cardiac dilatation and precordial distress. The temperature is at first as a rule not elevated, but as the case progresses, it may reach 103° or more and usually continues high until death. Cheyne-Stokes respiration is often present. Moist râles are often heard over the lungs from evident pulmonary œdema, and at times the sputum is very profuse. As a rule there is little impairment of the pulmonary resonance associated with pure fat embolism.

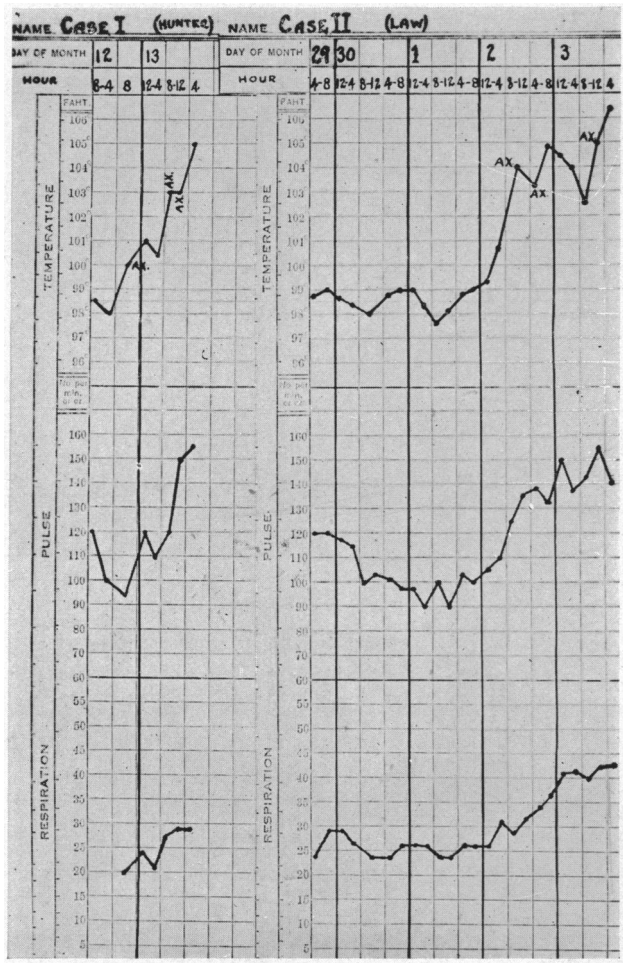

FIG. 17.—Charts of the two fatal cases.

The cardio-respiratory syndrome is the one which usually develops first, but if the case progresses, the second or cerebral syndrome comes strikingly into evidence, although it may appear within a few hours after the trauma or operation. The important features of the syndrome are at first restlessness, often headache, more or less delirium, sometimes hallucinations, then drowsiness, stupor, and finally coma, which persists until death. The reflexes may or may not be abolished, and there may be spasms, tremors or convulsions. The earlier cerebral symptoms are those of cerebral irritation coincident with the occurrence of the showers of emboli, while the later symptoms are those of paralysis, due to the rapid cerebral degeneration. There are no signs of increased intracranial pressure, and the eye grounds are usually normal. Embolism of the respiratory centre accounts for the Cheyne-Stokes respiration. Embolism of the heat regulating mechanism

may explain much of the elevated temperature, while embolism of the vasomotor centre may cause many of the apparent symptoms of shock. The pupils are usually rather small but equal. Petechiæ may be observed in the skin but are more or less obscured by the cyanosis and are at any rate of relatively late appearance.

The symptoms described characterize the fatal cases, but they may be seen in milder form in cases that recover, and it is quite evident that this picture closely resembles what is usually regarded as shock. It is probable that careful urinary examination will always reveal some fat in the urine in severe cases of fat embolism, and it may be present in mild ones. Warthin and others have also emphasized the importance of fat in the sputum as an aid in diagnosis. In cases associated with head injury, the diagnosis may be very difficult during life, but it should not be at autopsy, as it appears to have been in the past.

The course of fat embolism may be rapid or slow. Some cases are so rapid as to be described as apoplectiform, with death in a few hours, while others may persist for days or weeks to end in death or recovery. As a rule the longer the patient lives, the better the prognosis. In well-developed cases of fat embolism, especially those of the cerebral type, the prognosis is almost invariably bad, and the great majority of all severe cases die. One cannot judge the mortality by the literature, for most of the cases reported have been fatal ones, while it is probable that a much larger proportion recover but are not recognized as such and are classified under various headings. Post-operative or post-traumatic diagnosis of bronchitis and pneumonia may not infrequently be rather fat embolism.

The treatment of fat embolism is chiefly prophylactic. All cases of trauma, especially fractures, should have absolute rest, and all unnecessary movement or manipulation should be avoided. Transportation of such a case should be with the greatest care. Tight bandages should not be used. All unnecessary trauma should be most carefully avoided in operations, whether of orthopædic or other character. None of the methods of treatment so far proposed seems to be of any especial value, the reason for which becomes apparent when one studies the pathology of the condition. Venesection, saline injection and thoracic duct fistula are the chief measures hitherto recommended, but the results of their employment are by no means encouraging. Symptomatic treatment is in general about all that can be proposed. It is hoped, however, that as surgical attention is directed more and more to the importance of this condition, effective methods of treatment may be devised.

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