FATAL FAT EMBOLISM: A CASE REPORT* †

ERNEST A. DOUD, M.D., AND E. A. ROVENSTONE, M.D.

New York, N. Y.

Fat embolism, the most frequent type of embolism, is one of the serious catastrophes that may befall the anesthetized patient. Taylor (1) reported 2 fatal cases which constitute the only reference to deaths during anesthesia (one patient having received spinal and the other ether anesthesia) from this cause found in the literature. Since fat embolism does not usually cause death until the third postoperative day or later, a report of this proven death from fat embolism during cyclopropane anesthesia is of interest.

Fat embolism may be defined as the circumstance in which there are present in the circulation globules of fat sufficiently large to cause blockage of the capillaries of different organs. The capillary circulation is slowed but usually not permanently occluded by the globules because they are gradually forced through. However, the time required may be longer than vital tissues will tolerate without loss of function.

Normally, fat is stored in cells in the fat depots of the body such as the bone marrow. When trauma ruptures these cells, fluid fat is available to enter the Haversian veins which are kept open by their attachments in the bony canaliculi. Ordinarily, blood flows out of open vessels, but the pressure of a hematoma may force the freed fat into the circulation. The physiologic forces that maintain the venous circulation may also act to draw fluid fat into the open veins. The volume of fat and the rate at which it enters the vascular system determine the extent of the pathologicophysiologic response of the patient.

Lehman and Moore (2) calculated that the amount of fat in the bone marrow of the femur of an adult is approximately 65 cc. Assuming that cotton seed oil does not materially differ from marrow fat and that man, as well as dogs, tolerates free fat in the blood, they demonstrated experimentally that 120 cc. would be required to enter the bloodstream of a man weighing 80 Kg. to produce a fatal result. If this were true, all the fat in the marrow of a femur should be well tolerated. Senderi (3) believes, from experimental evidence, that oleic acid, which is one of the split products of the neutral olein of bone marrow, is a probable etiologic factor of fat embolism. This substance is seven

* From the Departments of Anesthesia, Doctors Hospital and Bellevue Hospital, New York City.
† Presented before a meeting of The American Society of Anesthesiologists, Inc., Feb. 14, 1946, New York, N. Y.

291
times as lethal as neutral bone marrow fat and a sufficient quantity could easily be available in the marrow of a femur. Oleic acid is toxic and it unites with the alkaline radicals in the blood stream to form a soap.

The highest incidence of fat embolism is to be found in the fourth decade. These accidents rarely occur in children because up to the age of 14 there is an insufficient amount of fat in the bone marrow, and it consists primarily of viscous palmitin and stearin and a small quantity only of the more fluid olein (4, 5). Males are affected in a ratio of eight to one. Speed believes that those addicted to alcohol are predisposed more than nonalcoholics because of the instability of the fat emulsion in the serum produced by the frequent dilution with the fat solvent. Simonds (6) found that etherized dogs were more susceptible to fat embolism than those anesthetized by nonfat solvents. It was believed that ether in the blood stream took into solution the circulating emulsified droplets of fat and, as the ether vapor passed into the alveoli, the ether tension in the blood was lowered to a point at which the fat came out of solution as free fat, and emboli occurred (2). Bisgard and Baker (7) showed that ether had little or no beneficial influence upon embolization of fat and, if at all, only during anesthesia. The vast majority of embolisms occurs three or more days postoperatively and ether could have no effect upon the fat that enters the blood stream at that time.

The liquid fat, when it enters the circulation, is broken into coarse globules which are carried from the traumatized area to the right heart. Some are forced through the pulmonary capillaries into the general circulation. Locally, there are small areas of congestion with exudation of erythrocytes and serum into the alveoli. In extreme cases, the globules are so numerous that they cause pulmonary edema and dilatation of the right side of the heart, thus interfering with the oxygenation of the blood. It may take several days for the fat to pass through the pulmonary circulation. Warthin (8) stated that no organ or tissue exists in the body that does not have some fat in its capillaries when fat embolism occurs. Although no symptoms may be noted, embolic phenomena, when present, are most prominent in the brain, heart, kidneys, lungs and skin.

Small amounts of fat are disposed of by various methods: (a) they may lodge within the glomeruli of the kidneys where, by glomerular capillary ruptures or intercellular openings, they pass into the urine; (b) some may be excreted by the bile into the intestinal tract; (c) some may be taken up by the phagocytic endothelial cells and digested, and (d) the remainder may be emulsified by the mechanical action of the blood current, aided to some extent by the saponifying action of the blood lipase, and reabsorbed into the tissues (9).

It is impossible to correlate the degree of injury with the resulting lipemia. Grondahl (10) stated that a terminal result occurs in one of
Fatal Fat Embolism: A Case Report

293

every 3000 cases of fracture and that only one of every 100 such deaths is attributed to fat embolism.

Fat embolism is difficult to diagnose because it is practically impossible to distinguish it from the symptoms and signs of the associated trauma and shock in the severely injured. The mild cases are not recognized. In the anesthetized patient, the signs are those of failing peripheral circulation.

Postoperatively, the signs and symptoms are those of pulmonary embolism of any type. The onset is sudden on the third to the sixth day with a sense of constriction in the chest, air hunger, cyanosis, rapid, feeble and perhaps irregular pulse, and hypotension. If the patient recovers, there is a free interval followed by cerebral manifestations of restlessness, delirium, drowsiness and possibly coma. There may be scattered petechial hemorrhages in the skin. Fat may be found in the urine, sputum, spinal fluid, and retinal vessels. The fat content of the blood is elevated (normal content ranges from 0.2 per cent to 2.0 per cent). It should be remembered that the cerebral symptoms which are occasionally seen following orthopedic surgery and which may have been attributed to hypoxia during anesthesia may in reality have resulted from cerebral embolism. Fat embolism is rarely diagnosed clinically, but usually is accidentally discovered at necropsy. Most of the patients recover, but since only the fatal or very sick ones have had a diagnosis and the mild cases have been overlooked, the reverse opinion is commonly held.

Death will occur in the anesthetized patient if the embolism is sufficiently large to occlude pulmonary circulation. It will occur, also, when fat passes through the pulmonary circulation or into the coronary circulation with embolic coronary occlusion. Scuder believes that the toxicity of oleic acid may be the lethal etiologic factor (3). deTakats (11) proposed that pulmonary emboli may cause death by vagal inhibition of the heart and the accompanying coronary constriction and bronchoconstriction. This theory fits in well with the experimental conclusion that there is insufficient fat in the bone marrow of a femur to cause death from embolism.

Case Report

The subject of this report was a 55 year old, well-developed, white woman whose structures were older than her age. An open reduction of an intracapsular fracture of the upper end of the neck of the left femur and insertion of an autogenous bone peg was proposed. The temperature was 101 F.; pulse rate 88, and respirations were at a rate of 20 per minute. The blood pressure was 158 mm. systolic and 100 mm. diastolic; erythrocyte count was 4,100,000 and hemoglobin 80 per cent. There was a three day old fracture of the left femur incurred in a fall when struck by a bicycle. The patient had a questionable cardiac valvular lesion, subsiding pharyngitis, and extensive, untreated varicosities of the extremities.
Morphine sulfate, 0.005 Gm., and scopolamine hydrobromide, 0.0003 Gm., were given hypodermically one hour preoperatively. This premedication was satisfactory and the induction and maintenance of cyclopropane anesthesia were uneventful for forty-three minutes (fig. 1). During this time, the blood pressure remained close to 150 mm. systolic and 100 mm. diastolic, the pulse rate decreased from 116 to between 80 and 90, and the respirations ranged from 18 to 22 per minute. Suddenly the blood pressure and pulse rate could not be elicited and the respiratory rate increased to 36. It was evident that the patient was in profound circulatory collapse. She was placed in a modified Trendelenburg position, and the breathing bag was repeatedly emptied and refilled with oxygen in an attempt to wash the anesthetic agent from the patient’s tissues.

**Fig. 1.** The Anesthesia Study Record. The respiratory rate is charted during the final thirty minutes without accurate count, since artificial respiration was interrupted at intervals only to determine if respiratory efforts were still being made.
Twelve minutes after the onset of circulatory collapse, an oral endotracheal
airway was inserted, and dilution of the agent was continued. No pulse or blood
pressure reading could be obtained, but irregular gasping respirations continued
at a decreasing rate for approximately thirty minutes. During this entire
period, artificial respiration was performed. An infusion of 5 per cent glucose
in normal saline solution was started seventeen minutes after the onset of shock
and epinephrine was injected into the infusion tubing. Thirty minutes after
the onset, all respiratory effort ceased and the patient was pronounced dead on
the table fifteen minutes later. It was interesting to note that the respirations
continued for a long period of time after the circulation apparently had stopped.

Necropsy disclosed pulmonary fat embolism. Branches of the pulmonary
artery in both lungs were occluded. An analysis of the lung secretions and
the urine disclosed the presence of fat. The heart was moderately distended.

There is no known treatment for this catastrophe. Atropine, 0.0006
Gm., and papaverine, 0.30 Gm., may be given intravenously if they are
immediately available to counteract the autonomic reflexes from the
affected lung. The literature is replete with prophylactic measures.
Among those most frequently mentioned are: early application of a
tourniquet when feasible and a slow release after early immobilization
and reduction, avoidance of unnecessary and rough handling, the early
treatment of shock if it occurs, and the use of an electrical saw instead
of a chisel during open reduction.

SUMMARY

Fat embolism, an unusual cause of death during anesthesia, is dis-
cussed and a case report is presented.

419 Sterling Ct.

REFERENCES

1. Taylor, Ivan B.: Case Report: Fatal Pulmonary Embolism During Operation, Anes-
thesiology 3: 689-691 (Nov.) 1942.
2. Lehman, E. P., and Moore, R. M.: Fat Embolism—Including Experimental Production
(Apr.) 1941.
(Feb.) 1940.
171-227, 1913.
11. de Takats, Geza; Beck, Wm. C., and Fenn, Geo. K.: Pulmonary Embolism, Surgery
6: 339-367 (Sept.) 1939.