FAT EMBOLISM

By Robert B. Wright, M.D.

of Baltimore, Maryland

from the department of pathology of the university of maryland school of medicine

Careful examination of autopsy material will frequently disclose the presence of fat within the capillaries of the lungs, kidneys, brain and other organs. In most instances, the fat globules are not sufficiently numerous to have produced clinical symptoms, but in some cases, especially traumatic ones, fat may exist in such quantities in the vascular system as to give definite indications as to the cause of death.

Warthin,[1] in his excellent monograph on this subject, called attention to the inadequacy of American literature on the subject as compared to European literature. He said, "the time has come that more attention should be paid to this sadly neglected branch of surgery and that the occurrence of fatty embolism after an injury to the bones be at least regarded as a possibility and that preventive means be instituted or therapeutic efforts be made whenever there is a suspicion of its occurrence." Indeed, at the present time, the charge may be made that American clinicians and pathologists in general give too little attention to so important a condition.

The author, to stimulate interest in the study of fatty embolism, offers a brief review of the subject together with the reports of two typical cases. For a history of fat embolism, reference is given to Warthin's monograph.

Endogenous fat embolism is almost always the result of physical injury to one or another of the fat depots of the body. Injury, especially fractures of the long bones, is by far the most common cause. There is no definite parallelism between the extent of trauma and the amount of fat liberated into the circulation, since fatalities are sometimes seen as a result of fracture of a single bone, while in other instances fractures of a number of long bones have not produced symptoms of fat embolism.

According to Conner[2] the character of the marrow fat is one of the determining factors in the production of fat embolism, for he points out that fat in older persons is more liquid, since it contains more olein, while in children, according to both Zwerg[3] and Timmer,[4] it is more cellular and contains more palmitin. This may explain why the condition is more commonly observed in persons past the age of fifty. That the condition may be seen in younger persons is shown by Utgenannt[5] who reported a case in a child eight years of age, by Work[6] who reported a case in a person eighteen years old, by Burns[7] who reported a case in a person twenty-two years old, and by Ryerson[8] who reported a case in an infant eight months old.

Orthopœdic operative procedures are a fruitful cause of fat embolism. There is a long list of such references in the literature. Warthin gives

twenty-two references, while Ryerson and Timmer, to mention only a few, have also stressed this fact.

Concussion of atrophied bones has been the cause of severe fat embolism, as is illustrated by cases reported by Fields,[9] Beitzke[10] and others. Timmer states that atrophied bones at any age may contain much fat and in the very young may be the origin of the fat in fat embolism. Bissell[11] and Sutton[12] have reported cases following operations on the soft parts, such as radical breast amputation, umbilical hernioplasty and laminectomy. Many other references could be cited if space permitted.

Milaslavich[13] thinks that contusions may play an important part in the

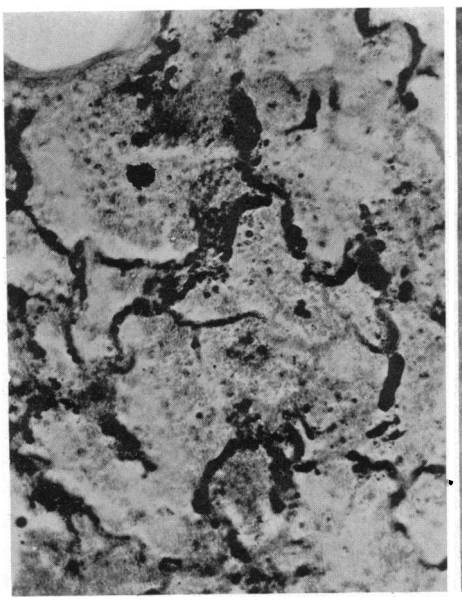


FIG. 1.—Fat emboli in lungs. Herxheimer's method.

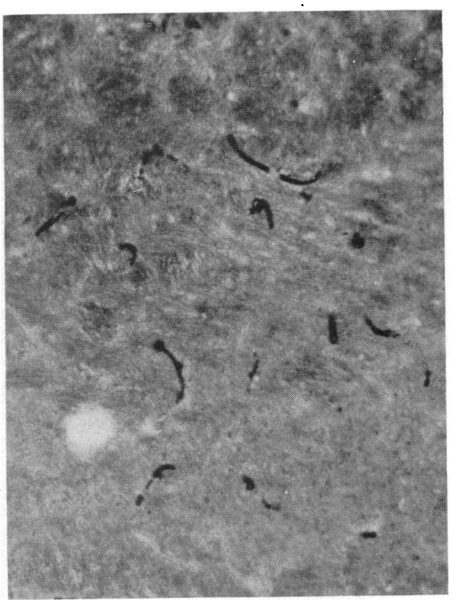


FIG. 2.—Fat emboli in brain. Herxheimer's method.

production of this type of fat embolism. This is not new, since Warthin stressed this possibility in 1913.

Fat embolism of a minor type has been reported in such conditions as pancreatitis, chronic nephritis, diabetes mellitus, chronic tuberculosis, acute and chronic alcoholism. According to Warthin and other workers, fat embolism is seldom, if ever, fatal in these conditions, unless trauma is present.

Burns and Bromberg[14] and Brittingham,[15] in reporting cases of death following therapeutic use of salvarsan and neo-salvarsan, suggest that in some cases, at least, the punctate hæmorrhages in the brain may be the result of the occlusion of the small vessels by fat.

Fat enters the vascular system at the site of injury where vessels are torn. The bones are ideal locations, for marrow of the long bone of the adult is rich in fat and has many vessels. It is possible that by suction fat may be

drawn into the veins, or, on account of the hæmorrhage into the marrow cavity, the fat may be forced into the vessels. Fat, having entered a vein, is carried to the right heart, then to the lung, through which it may filter and pass to the left heart, to be distributed throughout the greater circulation. Instead of passing through the lung, Fromberg[16] suggests that it passes through an open foramen ovale in most cases. That this is not always true is well shown by one case herewith reported.

As to the distribution of fat, much will depend on the myocardium and the capillaries of the lungs. Warthin noted many cases in which there was marked dilatation of the right heart. If the heart is powerful enough to

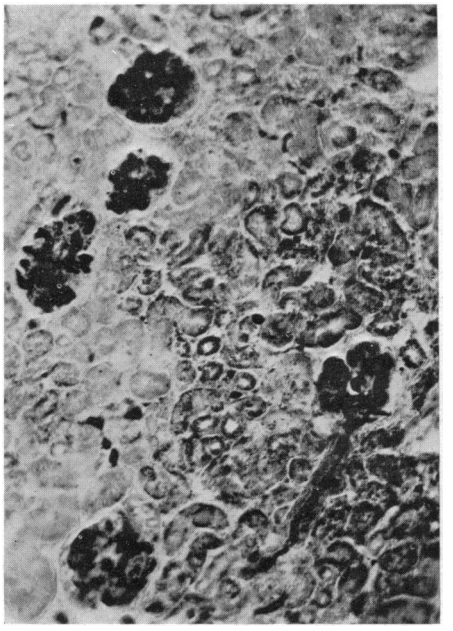


FIG. 3.—Fat emboli in the kidney. Herxheimer's method.

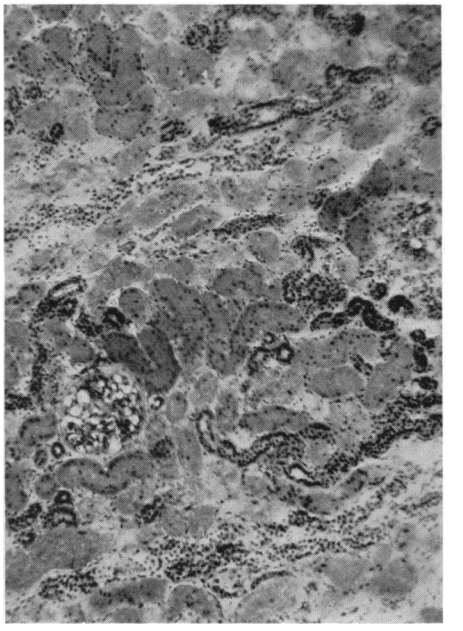


FIG. 4.—Kidney showing tubular degeneration. Hæmatoxylin and eosin.

push the fat through, death may not take place; on the other hand, the heart may be normal and the obstruction too great for it to overcome. It is also possible that changes in the pulmonary capillaries incident to age or disease of the lung may offer increased resistance.

There are two important factors in fat embolism; namely, the quantity of fat and the duration of the discharge of fat into the vascular system. It is easy to understand how these factors may vary and the outcome be changed. If the quantity is large enough it may not pass through the right heart and death may be instantaneous; more finely divided particles may be distributed through the lungs over a short period of time and death take place very quickly from failure of the right heart or from pulmonary œdema.

If the quantity is not too large and the right heart does not fail, much

fat may pass through the lungs and be distributed to all parts of the body. This fat, in the greater circulation, can be found in any tissue, but of course it causes symptoms from its effect on such vital organs as the heart, the brain, kidneys and glands of internal secretion.

In fat embolism there may develop in the lungs, lobular emphysema, small hæmorrhagic areas and pulmonary œdema. According to Lehmann and McNattin[17] slow distribution in experimental animals will cause small collections of wandering cells, fibrosis, endothelial proliferations and sometimes miliary collections of polymorphonuclear leucocytes. In the heart there are apt to be areas of fatty degeneration in the region of fat emboli. In the brain one finds many hæmorrhages in the sub-cortical white substances, especially the corpus callosum (Gauss,[18] Melchoir,[19] and Fromberg[16]). These hæmorrhages are perivascular and at the point where a globule of fat closes the central vessel. In the kidney the glomeruli are apt to be full of fat and degeneration of the tubular epithelium may be present. According to Paul and Windholz[20] there may be evidence of renal disturbances. There may be small hæmorrhages in the skin and in fact in almost any organ in the body. While the above lesions are often definite, yet there are fatal cases of fat embolism with only slight gross changes, so that it is only after properly prepared histological sections are examined that the correct diagnosis can be made.

It is relatively simple to make the diagnosis at post-mortem, but it requires considerable care to determine the significance of the findings. In the laboratories here, blocks are taken from the lung and frozen sections twenty to forty microns in thickness are made and stained by Herxheimer's method for fat. In suspicious cases more than one portion of the lung and also other important organs are examined. Only low-power magnification is required, as the fat is easily seen. Relatively thick sections are made so that the fat may be held in the vessels. Examination at post-mortem should be made for fat embolism in all traumatic cases, especially if there are few or no lesions found in the gross to explain death.

The clinical pathology is quite as definite and should always be borne in mind. Fat may first appear in the sputum and later be found in the urine. Warthin says, "the presence of free fat and fat granule alveolar cells in the sputum is the earliest positive evidence of the condition being detected even before the appearance of free fat in the urine."

Fat embolism is by far the most common type of embolism. Scriba[21] found it in 52 per cent. of all bodies. Lehmann and McNattin found it in thirty-nine out of fifty cases. In routine autopsies as done in general hospitals the best figures show around 50 per cent. of all cases to have more or less evidence of fat embolism. This, of course, does not mean that 50 per cent. die of fat embolism. Milaslavich found fat embolism in all of twenty-two cases of automobile accidents and concluded that it killed two.

DIGEST OF ONE HUNDRED CONSECUTIVE AUTOPSIES

Cases in which fat was not found in the pulmonary vessels

(1)	Cases without history or evidence of trauma		33
(2)	Cases with history of evidence of trauma:		
	Major operations	11	
	Minor contusions	2	
	Pregnancy with delivery	1	
	Old fracture	1	15
	Total		48

Cases in which fat was found in the pulmonary vessels

(1)	Cases without history or evidence of trauma:		
	Very occasional globule	22	
	1 = plus (globules in every 4–6 l.p.f.)	4	
	2 = plus (globules in every 2 l.p.f.)	1	
			27
(2)	Cases with history or evidence of trauma:		
	(*a*) Very occasional globule		
	Major operations	7	
	Cerebral hæmorrhage	3	
	Pregnancy and delivery	2	
	Birth injury	1	
			13
	(*b*) 1–plus (globules in every 4–6 l.p.f.)		
	Fracture of pelvic bones	1	
	Osteomyelitis, chronic	1	
			2
	(*c*) 2–plus (globules in every 2 l.p.f.)		
	Cerebral hæmorrhage	2	
	Convulsions	3	
	Multiple lacerations and contusions	1	
			6
	(*d*) 3–plus (globules in every l.p.f.)		
	Puerperal sepsis	1	
			1
	(*e*) 4–plus (globules in practically every capillary)		
	Convulsions after cerebral apoplexy	1	
	Alcoholic delirium with restraint	1	
	Fracture, contusions and burns	1	
			3
	Total		52

In the table there is an outline of 100 consecutive autopsies performed at University Hospital. It will be noted that fifty-two had some fat in the pulmonary vessels. It can be seen at a glance the rôle played by trauma. Among these 100 cases there is no doubt that fat embolism was of a major importance in at least three, only one of which was traumatic at the onset of the fatal illness. In arranging this table a case was classified under trauma if there was any possible reason for so doing.. Operations, pregnancy, and cerebral hæmorrhages were all considered as traumatic in this study.

The two cases herewith reported were not in the series of autopsies discussed in the table.

CASE I.—Mrs. B. M., Autopsy 1367, Chart No. 57879, white female, aged fifty-nine years, admitted April 18 in coma and died April 19. On the evening of April 17 the patient fell from a street car in front of her home and had to be carried into the house. The family physician found a fracture of the left hip. She was given morphia and made as comfortable as possible until the next morning. When he returned, finding her stuporous and irrational, he sent her to the hospital at once. On admission the temperature was 99° by axilla, pulse 110, respirations 22. The patient was in coma and had labored respiration, the face was drawn and there was no attempt to move. The pupils were contracted, equal, regular, and reacted to light. The right eye was rotated outward and upward, the fundi were negative. The heart and lungs were negative. The blood pressure 110/68. There was some general resistance over the entire abdomen. The extremities were definitely spastic and there were signs of fracture of the left femur. The leg was supported by pillows and she was given intravenous sugar, salt and stimulants. The temperature gradually rose to 107° by axilla, the pulse to 185, and the respirations to 40. She died about forty hours after the accident. (See temperature chart.)

X-ray Report.—Fracture with displacement, neck of femur, left. No evidence of skull fracture.

Laboratory Findings.—Urine on admission normal. Twelve hours later albumin and sugar were found. The urine was never examined for fat. Blood: red blood-cells, 4,370,000; white blood-cells, 12,500; hæmoglobin, 65 per cent.; polymorphonuclears, 86 per cent. Small lymphocytes, 8 per cent. Large mononuclears, 4 per cent. Spinal fluid, negative. Wassermann, negative. Blood chemistry: non-protein nitrogen, 29. Sugar, 93.

Clinical Diagnosis.—Fracture, neck of femur, left. Concussion of the brain.

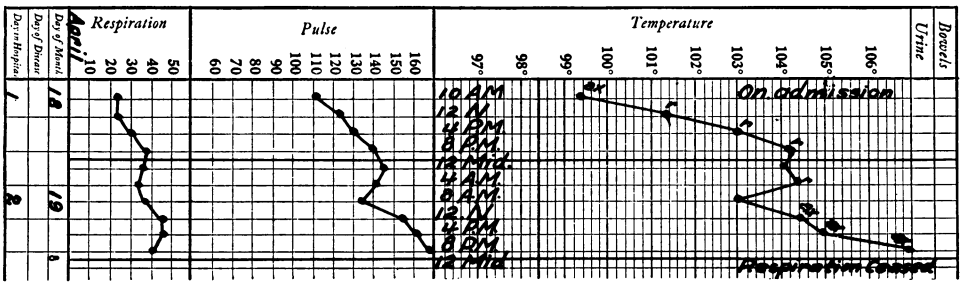
Autopsy performed one hour after death. The only positive findings in the gross were fracture of the left hip, small hæmorrhages in the brain, moderate arteriosclerosis, mild scarring of the kidneys, congestion of the lungs and abdominal viscera, bilateral hydrothorax—right, 300 cubic centimetres, left, 150 cubic centimetres, slight hydroperitoneum, slight œdema of the ankles. The foramen ovale was anatomically closed.

Frozen section stained by Herxheimer's method for fat showed extensive embolism in the lungs, brain and kidneys. Sections stained by hæmotoxylin and eosin showed many perivascular cerebral hæmorrhages and marked degeneration of the epithelium of the convoluted tubules of the kidney. There was no pneumonia.

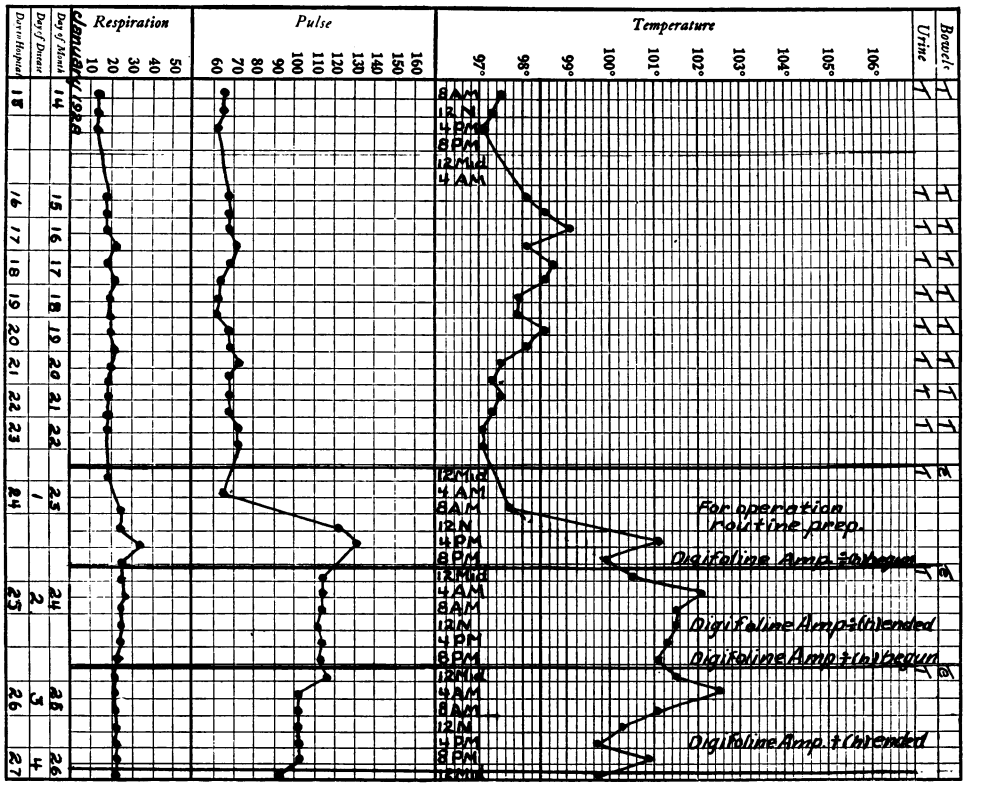
The above is a typical case from several points of view. Clinically, a patient in coma too often suggests only diabetes or nephritis. Not once was fat embolism considered, yet under the circumstances it was by far the most likely thing. The autopsy was performed by a man who had done but 30 or 40 post-mortems and did not suspect this condition. As soon as those who had more experience at post-mortems became aware of the case, the question of fat embolism was introduced and its existence proved a few minutes later by simple frozen section.

CASE II.—Chart No. 61170, white male, aged forty-two years. October 8, 1928, the patient sustained a fracture of the shaft of the right femur and the left humerus. These fractures failed to unite after being immobilized. January 23, 1929, an open reduction of the femur was done. When the smooth ends of the ununited fracture were broken into, a large amount of pus-like material (later found to be fat) escaped. Two hours after operation the patient went into profound shock and remained in this condition for forty-eight hours.

Temperature Chart – Case 1



Temperature Chart – Case 2



Laboratory Findings.—Urine: The urine frequently showed albumin and casts, and on January 24 and 31, large quantities of fat were present. Blood Chemistry: "Non-protein nitrogen," 30 to 39.9; urea nitrogen, 14 to 18.6; creatinin, 1.5 to 1.7; blood sugar, 111 to 118. Blood Wassermann was positive. This case ultimately recovered and left the hospital. The post-operative shock was considered to be due to fat embolism. (See temperature chart.)

From the pathology of the condition it is clear that almost anything can happen. If the greater circulation contains many globules of fat it is obvious that they may lodge in any organ and thus cause a great variety of symptoms. Clinically, in the main, there are two general types of fat embolism, namely, the one with cardiorespiratory symptoms and the other with cerebral symptoms.

The cardiorespiratory group is apt to present dyspnœa, cough, cyanosis, pulmonary œdema, even pulmonary hæmorrhage, low arterial and high venous pressure, cardiac dilatation, precordial distress, elevation of temperature and Cheyne-Stokes respiration. The cerebral type is apt to present restlessness, headache, delirium, drowsiness, stupor, coma and even convulsions. Many cases present a combination of the above.

In making the diagnosis, clinically, the most important point is to keep fat embolism in mind in every traumatic case where the symptoms are not clearly explained by definite findings. Warthin says, "fat embolism resulting from traumatic lipæmia is an important surgical condition which is not of rare occurrence but is probably at the present time, in the absence of infection, the most frequent cause of death after fractures of the long bone." Fat can easily be demonstrated in the sputum and in the urine and, as has been mentioned, it may be found in the vessels in the fundi of the eyes. If one remembers that fat embolism results from many types of trauma, especially fracture of a long bone, and that it is the extent of the embolism and the condition of the involved organ that determines the symptoms, there will be many more cases diagnosed clinically.

It is obvious that treatment of any condition so often not even considered is apt to be far from satisfactory. Diagnosis is, of course, the first step in rational therapy. All agree that shock and pulmonary œdema may demand immediate treatment, but it has occurred to but a few that a tourniquet properly applied in shock following fracture may be of much or more importance than any other procedure (Caldwell and Huber[22]). Some have been so bold as to drain the thoracic duct (Wilms[23]). Others have opened simple fractures, and even amputation (Melchoir) has been performed to prevent fat embolism. Such procedures in selected cases may be of value, for one is dealing with a major condition. Infusions have been used (Schanz,[24]) with some degree of success. Porter[25] thinks that there is a critical diastolic blood-pressure level, below which, if the pressure fails, it does not return unaided.

To bring about a return he advocates carbon-dioxide increase in respiratory air, heat, adrenalin, elevation of lower portion of the body, infusion and

transfusion. It is generally thought that care in handling cases of fracture of long bones will do much to prevent or at least to lessen fat embolism. The Thomas splint probably has done much to reduce fat embolism in those that have to be transported after a fracture of one of the long bones.

This condition is of importance to those interested in medico-legal medicine. In Germany this aspect has received considerable stress, while in this country it is seldom considered. Milaslavich has recently reported a case where the finding of fat embolism in a body recovered from a burning dwelling was admitted as evidence in court and helped to show that the woman's husband murdered her and then burned the house to conceal the crime. There is no doubt that the medico-legal aspect of this malady has by no means been fully developed and will, as time passes, be of more and more interest to the forensic world.

SUMMARY

The clinical and pathological aspects of fat embolism are presented. Two cases are reported, one resulting in death and the other in recovery. A digest of 100 consecutive autopsies as regards this condition is presented. It is felt that fat embolism should be considered in any case where there are grave or unusual symptoms following trauma, whether the trauma be fracture, contusion, concussion, intravenous therapy, delivery or operation.

BIBLIOGRAPHY

[1] Warthin, A. S.: Traumatic Lipæmia and Fatty Embolism. Internat. Clin., series 23, vol. iv, pp. 71–227, Philadelphia, 1913.

[2] Conner, L. A.: Embolism. Nelson's Loose-Leaf Living Medicine, vol. iv, p. 598, 1920.

[3] Zwerg, H. G.: Embolism of Fat. Beitr. z. klin. Chir., vol. cxli, p. 268, 1927.

[4] Timmer, H.: Epileptiform Convulsions and Fat Embolism after Non-surgical Orthopedic Intervention. Nederlandsch Tijdschrift v. Gerreiskunde, vol. ii, No. 3, Amsterdam, July 19, 1919; (Ab. J.A.M.A., vol. lxxiii, 1919).

[5] Utgenannt, L.: Uber Fettembolien und Krampfanfalle nach orthopadischen Operationen. Ztschr. f. Orthop. Chir. Stuttgart, vol. xli, pp. 393–413, 1921.

[6] Dennis, W. S., and Work, P.: Multiple Fat Embolism with Report of Case. Colorado Medical, vol. xxi, pp. 347–352, Denver, November 24, 1924.

[7] Burns, R. E.: Fat Embolism. Case Report. Wisconsin Medical Journal, vol. xxvi, pp. 156–157, Milwaukee, March, 1927; (Ab. J.A.M.A., vol. lxxxviii, 1927).

[8] Ryerson, E. W.: Fat Embolism in Bone Surgery. Incidence and Prevention. J.A.M.A., vol. lxvii, p. 657, 1916.

[9] Field, M.: Fat Embolism from a Chronic Osteomyelitis. J.A.M.A., vol. lix, p. 2065, 1912.

[10] Beitzke: Sur l'embolie graisseuse. Rev. Med. de la Suisse Rom., vol. xxxii, pp. 501–509, Geneve, 1912.

[11] Bissell, W. W.: Pulmonary Fat Embolism. A Frequent Cause of Post-operative Surgical Shock. Surg., Gynec., and Obstet., vol. xxv, No. 1, July, 1917.

[12] Sutton, G. E.: Pulmonary Fat Embolism and Its Relation to Traumatic Shock. British Medical Journal, vol. ii, p. 368, October, 1918.

[13] Milaslavich, E. L.: Fat Embolism. Medico-legal Aspects. Wisconsin Medical Journal, vol. xxix, p. 139, March, 1930.

[14] Burns, E. L., and Bromberg, L.: Fatal Multiple Fat Embolism in a Patient Given Salvarsan. Am. Jour. Syphilis, vol. xiv, p. 43, 1930.

[15] Brittingham, J. W., and Phinizy, T.: Hæmorrhagic Encephalitis after Neo-arsphenamin. Case with Fat Embolism Found at Autopsy. J.A.M.A., vol. xcvi, p. 2021, June 13, 1931.

[16] Fromberg, C.: Die Fettembolie des grossen Blutkreislaufes und ihr Urscechen. Mitteilungen aus den Grenzgebeiten der Medizin und Chir., vol. xxvi, p. 23, Jena, 1913.

[17] Lehmann, E. P., and McNattin, R. F.: Pathology of Lungs in Experimental Embolism. Southern Med. Jour., vol. xxii, p. 201, March, 1929.

[18] Gauss, H.: Pathology of Fat Embolism. Archives of Surgery, vol. ix, p. 593, November, 1924.

[19] Melchoir, E.: Zur Keuntnis der cerebralen Fettembolie. (Fat Embolism in the Brain.) Mitteil. a.d. Grenzgeb. d. Med. und Chir., vol. xxxviii, p. 149, Jena, 1924.

[20] Paul, F., and Windholz, F.: Experimental Studies on Death from Fat Embolism. Mitteil. a.d. Grenzgeb. d. Med. und Chir., vol. xxxviii, p. 614, Jena, 1925.

[21] Scriba, J.: Untersuchungen uber die Fettembolie. Deutsche Zeitschrift Chirurgie, vol. xii, p. 118, Leipzig, 1880.

[22] Caldwell, G. T., and Huber, H. L.: Fat Embolism Following Trauma to Bones. Surg., Gynec., and Obstet., vol. xxv, No. 6, pp. 650–663, 1913.

[23] Wilms, M.: Treatment de l'embolie graisseuse par drainage temporaire du canal thoracique. Semaine Medicale, vol. xxx, p. 138, 1910.

[24] Schanz: (quoted from Warthin.) Zentrbl. f. Chirur., vol. xxxvii, 1910.

[25] Porter, W. T.: Fat Embolism Cause of Shock. Boston Med. and Surg. Jour., vol. clxxvi, No. 6, February 15, 1917; Am. Jour. Physiology, vol. lxxi, 1924.