

FAT EMBOLISM STUDIED IN 100 PATIENTS DYING AFTER INJURY

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(RECEIVED FOR PUBLICATION FEBRUARY 9, 1957)

Historical

The occurrence of fat embolism as a complication of traumatic injury was first recognized in the latter half of the nineteenth century. Much work has since been done on the condition and the literature is peculiarly rich in references. Wilson and Salisbury (1944) estimated their number at 600, and Warthin (1913) and Groskloss (1935-36) have reviewed the extensive literature. Discussion has centred around four main points: (1) the type of illness or injury commonly associated with fat embolism; (2) the origins and mode of release of the emboli; (3) the frequency; and (4) the significance of fat embolism.

The first two points have been thoroughly investigated. Various authors have published reports of the frequency of fat embolism in necropsy material (Olbrycht, 1922; Vance, 1931; Whiteley, 1954) and have concluded that, while occasional pulmonary fat emboli may be found in a variety of conditions and a slight degree of fat embolism is commonly found after burns, fat emboli in significant numbers are only found after injury or illness characterized by fits or convulsions. Serious and fatal degrees of fat embolism have been reported after slight injuries (Ziemke, 1922), but are commonly found only in patients with severe injuries, particularly those with fractures of long bones. In the present investigation these findings have been confirmed in a small series.

Aetiology and Pathogenesis

Fat emboli are believed to originate in the marrow of bones injured by fracture, or rarely by general skeletal shaking without fracture ("*Er-schütterung*") by rupture of marrow fat cells. Reports of fat embolism in patients with widespread bruising of the subcutaneous fat have been made (Scully, 1956), but in these patients there

must have been a severe degree of general skeletal shaking and the source of the emboli is still in question. When free fat is released into the marrow cavity of a fractured bone, increased medullary pressure forces it into the venous sinuses which are held open by their attachments to the bony trabeculae. The fat traverses the right side of the heart to impact in the pulmonary vessels, and some may penetrate to the systemic circulation where it again lodges as emboli.

Other views have been advanced as to the origin of the fat, notably that it results from a breakdown of the physiological fat emulsion of the plasma, either from the stimulus of metabolites released by the injury, or "triggered off" by a small amount of free circulating fat (Lehman and Moore, 1927). The experimental evidence advanced in support of this theory is not convincing, and it has not gained wide credence. One of the stimuli to the production of this theory was the statement that there is not enough fat in the marrow of one femur to produce death by fat embolism, even if it all be released as emboli. This has been disproved by Glas, Grekin, Davis, and Musselman (1956) and by Peltier (1956).

Two main clinical types of fat embolism have been described.

Pulmonary Fat Embolism.—This is said to be characterized by dyspnoea, cyanosis, sputum, which may be copious and frothy or slightly blood-stained, and increasing respiratory embarrassment leading in some cases to death. Necropsy examination is said to show characteristic changes in the lungs (Robb-Smith, 1941). They are described as being heavy and voluminous, firm but not solid, with a marbled appearance of the visceral pleura due to alternating areas of haemorrhage and emphysema. Tardieu spots may be present beneath the visceral pleura. Histological examination shows capillary congestion, intra-alveolar haemorrhage, and oedema with zones of emphysema.

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Cerebral Fat Embolism.—The symptoms of cerebral fat embolism may follow those of pulmonary fat embolism or occur without warning. A delay of 24 to 72 hours after injury before the development of symptoms is described as characteristic, but the examination of the records of published cases and personal experience shows the delay to be less than commonly supposed. The patient is generally conscious after injury and loses consciousness later. This loss of consciousness may be sudden or gradual, and may be preceded by a period of restlessness or even mania which has on occasions been diagnosed as delirium tremens. It may also manifest itself as a failure to recover consciousness after anaesthesia. Another initial sign is incontinence in a previously continent patient. Whilst unconscious the patient may show various signs of disturbance of the central nervous system such as paralysis, spasticity, loss of reflexes, or fits. A rise in temperature is often seen, and may reach 105° F. A petechial skin rash may also be present, most marked over the anterior upper thorax and shoulders and in the conjunctivae. At necropsy the macroscopic appearances are of a brain under slightly increased pressure, with congested vessels, in which section shows numerous haemorrhages, ranging in size from petechiae to that of a pea, distributed throughout the white matter of the cerebral hemispheres. Haemorrhages may also be present, but are less common, in the cerebral grey matter and in the brain-stem and cerebellum.

Material and Methods of the Present Investigation

Trauma Series.—Material consisted of clinical records, necropsy reports, and histological preparations from 100 patients who died after injury.

Control Series.—Material from 53 patients who died without suffering trauma was used. Because of the specialized nature of the hospital, a large proportion of the deaths were from burns, and of these a large number occurred in children. This is not a satisfactory control group, as burns are known to give rise to a constant though minor degree of fat embolism (Olbrycht, 1922) and fat embolism is said to be less common in children than in adults (Whiteley, 1954; Wright, 1932). In spite of these objections, there is a qualitative difference between the results in the two series.

Methods

Quantitative Estimation of Pulmonary Fat Embolism.—Various workers have defined mild, moderate, and severe pulmonary fat embolism to their own satisfaction, but it is very difficult to compare various sets

of data with varying, largely subjective criteria. So far as can be ascertained, only one study has been made in which fat emboli per unit area of lung section have been counted, and this was done in experimental animals (Swank and Dugger, 1954). It seemed important that the assessment of pulmonary fat embolism should be made on as strictly a numerical basis as possible in order that comparison between different cases should be easy and accurate. Accordingly, in this investigation the numbers of fat emboli have been counted in standard areas of lung sections. Frozen sections 15 μ thick were used, stained with oil red O and light green, and in each section all the emboli in 20 randomly selected fields were counted. The magnification used was 90. Each field was 0.95 mm. in diameter and 2.985 sq. mm. in area, making the total area counted in each section 59.7 sq. mm. Fat emboli in the lungs are recorded as numbers in this, the unit, area. Emboli were counted individually; there was no difficulty with those obviously intravascular, but some did arise with fat globules in the alveoli. It was considered that where intravascular emboli were numerous, extravascular globules of 10 μ or more in diameter were in all probability extravasated emboli, and they were counted as such. Fat globules smaller than these were ignored, and in lungs with small numbers of intravascular emboli, extravascular fat, which was very small in amount in such lungs, was excluded. The proportion of extravascular to total fat was in no case high, and its inclusion or exclusion would not have altered the group into which a case fell.

Cases were classified as mild, moderate, or severe pulmonary fat embolism. Mild fat embolism included cases with from one to 20 emboli per unit area; moderate fat embolism was from 21 to 60 emboli per unit area, and severe fat embolism more than 60 emboli per unit area. The definition of mild fat embolism adopted approximates to the "mild fat embolism" of Whiteley (1954), the "significant fat embolism" of Denman and Gragg (1948), and the "+ fat embolism" of Robb-Smith (1941). The line between moderate and severe fat embolism has been drawn quite arbitrarily at the level above which systemic fat embolism may be expected to occur.

Distribution of fat emboli in the lungs was not studied, but other workers failed to demonstrate any localization (Olbrycht, 1922). No method of selecting blocks for section was followed, save that, where macroscopic appearances of various areas of the lungs varied, an effort was made to obtain blocks representative of all areas. Each case in both series was examined for pulmonary fat embolism. In these preparations fat emboli were seen as orange-red staining masses in the capillaries and small arterioles, and similar masses often lay free in the alveoli. Fat-containing macrophages were constantly present, but their presence and numbers bore no relationship to the degree of fat embolism (Scott, Kemp, and Robb-Smith, 1942), and they were easily identified. Fat deposits around the larger bronchi and pulmonary vessels were encountered, but the fat could be seen to be extravascular.

The changes in the lung parenchyma accompanying fat embolism were found to be inconstant and non-specific. Transudation of fluid into the alveoli, with infection, diapedesis of polymorphs, and extravasation of red cells are all found in lungs showing no or minimal fat embolism as well as in those with severe degrees of the condition. Conversely, there may be little or no reaction to severe degrees of fat embolism.

Systemic Fat Embolism

Fat emboli originating in the marrow of injured bones traverse the venous system and the right side of the heart and impact in the pulmonary vessels. In the absence of a functioning communication between the pulmonary and systemic circuits emboli appearing in the latter must have traversed the pulmonary capillaries. Possible by-pass routes are:

(1) A patent foramen ovale, which Friedberg (1950) states is present in 20 to 25% of adult hearts but of functional significance in only 6%. In the remainder the opening is covered by a flap valve which is kept closed by the difference in pressure between the atria. Should congestion of the right heart reverse the pressure difference, right to left passage of emboli could occur, and this has been stated to happen (Green and Stoner, 1950).

(2) The bronchopulmonary venous shunt (Marchand, Gilroy, and Wilson, 1950), which is also said to become important if pressure in the right heart rises.

Both these mechanisms depend on a rise in pulmonary arterial and systemic venous pressure. This has been stated to occur in pulmonary fat embolism, but in the present series there has been no clinical evidence to confirm this observation. There is also no evidence that systemic fat embolism is commoner in patients with a patent foramen ovale. It is concluded that when fat emboli gain access to the systemic circulation they commonly do so by passing through the pulmonary capillaries.

The degree of pulmonary fat embolism is one factor determining the occurrence of systemic fat embolism, but other factors remain largely conjectural. Anything changing the diameter of the pulmonary capillaries will affect the passage of emboli; such factors have been described (Swank and Hain, 1952; Swank and Dugger, 1954) and have been suggested to be associated with the production of systemic fat embolism. Systemic embolism has also been stated to be commoner in young people with sound hearts beating forcefully than in the aged (Hämig, 1900). That other factors are involved is seen by the variable occur-

rence of systemic fat embolism in patients with the same degree of pulmonary fat embolism.

Once fat emboli are released into the systemic circulation their distribution is a random matter, depending on the amount of blood each organ receives. The symptoms produced will depend on the vulnerability of the organ to minor vascular occlusions. Most organs have considerable functional reserve and can withstand blockage of a small proportion of their capillaries without disturbance. The brain is the chief exception to this generalization. The vessels of the cerebral white matter in particular are end-arteries and have poor collateral circulation; cerebral tissues are acutely sensitive to anoxia and vascular lesions are promptly followed by disturbances of function. Anatomical lesions follow and are most conspicuous in the white matter, giving the histological picture of an area of haemorrhage surrounded by one of avascular necrosis and forming the "ball" and "ring" haemorrhages and "anaemic infarcts" of cerebral tissue. Swank and Hain (1952) describe the experimental production of such lesions by the injection of emboli of known size, and their studies emphasized the production of micro-infarcts and the abnormal permeability of capillaries in the absence of other visible abnormality. They saw persistent lesions in the brain of an animal 100 days after a single injection of emboli, and emphasized the speed of functional recovery in the presence of anatomical lesions.

The classical description of gross cerebral fat embolism is of a brain under moderate tension, showing on section multiple petechial haemorrhages throughout the white matter of the cerebral hemispheres, and to a lesser degree in the grey matter of the cortex and in the brain-stem and cerebellum. Lesser degrees of fat embolism, while sufficient to cause gross impairment of function, are not always apparent macroscopically and may be diagnosed on histological evidence only. When cerebral fat emboli are present they are seen lying in the cerebral capillaries, in the centre of small areas of haemorrhage, or in association with anaemic infarcts. In numbers they are scanty compared with pulmonary fat emboli, even in patients whose death is attributable to the cerebral fat embolism. No attempt has been made to count cerebral fat emboli, but their numbers were assessed and their importance in causing symptoms and death determined in relation to the other lesions and the clinical history. Any cerebral fat embolism is of grave significance, in contrast to pulmonary fat embolism, which is

an almost constant accompaniment of fractures and of only minor importance.

Non-embolic fat is often found in the brain and has been a source of confusion. Such fat is common in the vessel walls and perivascular tissues of the larger vessels, but no evidence has been found that it is of embolic origin. Swank and Dugger (1954) counted all stainable fat seen in cerebral sections and described it as embolic, but this does not appear to be justified in human material, and only fat seen to be intravascular or in association with infarcts has been accepted as embolic.

It is probable that the effects of cerebral fat embolism are attributable to mechanical occlusion of vessels and tissue anoxia. The possibility of fat having an irritant chemical action has been raised, but histological examination does not suggest that this is significant. Cammermeyer (1953) has described juxta-embolic thrombosis of fibrin complicating fat embolism, and suggests that this is due to anoxic tissue necrosis releasing a thromboplastic substance which diffuses into the vessels. The direct effect of fat on clotting (Fullerton, Davie, and Anastasopoulos, 1953) must also be considered in this connexion, and the effect of increased fat on blood viscosity has been described by Cullen and Swank (1954). They feel that this in itself may be sufficient to cause tissue changes in the absence of actual cerebral embolism.

Sections of kidney were examined for fat emboli in all but one of the trauma series. Systemic fat embolism is generally better seen here than in any other tissue because of the large blood supply and the localization of the capillary bed in the glomeruli. If systemic fat embolism is present it should best be seen in renal sections, and if the emboli are few in number they may be seen nowhere else. Of 24 cases in the trauma series showing systemic fat embolism, only four did not have renal embolism. Renal fat embolism does not produce changes in the renal parenchyma or in renal function. Early attempts were made to implicate renal fat embolism in the production of post-traumatic oliguria and uraemia (Gauss, 1924), but investigation did not sustain this. Flick and Traum (1930) investigated the effects of fat emboli on the kidneys of healthy experimental animals, and showed that only slight changes were produced which resulted in no permanent impairment of renal function.

All sections of brain and kidney examined for fat embolism were cut at 15 μ on the freezing microtome and stained with oil red O and light green.

Systemic fat embolism has been reported to occur in almost every organ, but the only other site in which it has been thought to be of danger to life is the heart. Early descriptions of systemic fat embolism distinguished a coronary type and a fatal syndrome arising therefrom (Gauss, 1924). Sections of myocardium from several patients with severe systemic fat embolism have been examined, but in only one case were emboli found and they did not appear to be of significance. Scully (1956) was unable to identify a syndrome due to myocardial fat embolism.

Results

Control Series.—This consists of 53 patients dying after burns or non-traumatic illnesses. They are analysed in Table I.

TABLE I
CONTROL SERIES

	Age in Decades								Total	Burns	Non-burns
	1	2	3	4	5	6	7	8+			
All cases	21	5	4	3	3	4	5	8	53	43	10
Cases with pulmonary fat embolism	5	2	4	1	1	3	2	3	21 (40%)	16 (37%)	5 (50%)

The incidence of pulmonary fat embolism is high, occurring in 37% of the burns cases and in 50% of the other cases. However, of 16 burns cases showing pulmonary fat embolism, 13 showed a count of 5 or less, and these minimal numbers are not considered significant. The remaining three cases had counts of 13, 16, and 21. Of the 16 cases none showed symptoms of pulmonary or systemic fat embolism, and in those where renal and cerebral sections were examined for fat emboli none were found.

In the "non-burns" series five cases showed pulmonary fat embolism. In three the count was less than 5, and in one it was 6. In the remaining case a man dying of a ruptured cerebral aneurysm had pulmonary fat embolism with a count of 47, but no systemic emboli. The onset of the haemorrhage had caused him to fall 4 ft. from a ladder, and he suffered lacerations but no fractures. The moderate pulmonary fat embolism probably resulted from general skeletal shaking.

Trauma Series.—This consisted of 100 patients dying after injury. In most cases the injury was the immediate or underlying cause of death. The group is analysed in Table II; the preponderance of males reflects the greater liability of men to severe trauma in all but the highest age groups.

TABLE II
TRAUMA SERIES

	Age in Decades								Total
	1	2	3	4	5	6	7	8+	
Males	2	4	12	6	11	12	11	13	71
Females	1				3	3	6	16	29
Both sexes	3	4	12	6	14	15	17	29	100

Only in the over-71 age group is the number of women greater than that of men; of these old ladies, 10 died after a fracture of the neck of the femur.

Classification of Trauma.—Trauma is divided into three grades :

“Mild” denotes fractures of the skull, or a fracture of the neck of the femur.

“Moderate” denotes fracture of one long bone, or two or three fractures of any bones.

“Severe” denotes four or more fractures.

The generally accepted source of fat emboli is the marrow of fractured bones, and in considering the relationship between fat embolism and trauma the number of fractures is of prime importance.

Only very severe soft tissue injuries were taken into account in a few cases in assessing the degree of trauma.

Eighty-nine of the 100 cases showed pulmonary fat embolism. In the 11 cases where none was found, two factors were considered : (1) In all the survival time was long, averaging 40 days after injury and in no case being less than 12 days. (2) In several of the 11 cases the degree of trauma was very slight. In the remaining 89 cases it has been found that in general the degree of pulmonary fat embolism increases with the severity of trauma, severe fat embolism being uncommon with mild and most common with severe trauma (Table III).

TABLE III
RELATIONSHIP BETWEEN DEGREE OF TRAUMA AND OF PULMONARY FAT EMBOLISM

	Degree of Trauma			Total	
	Mild	Moderate	Severe		
Pulmonary fat embolism	Mild . . .	17	6	5	28
	Moderate . . .	7	9	13	29
	Severe . . .	3	4	25	32
Total	27	19	43	89	

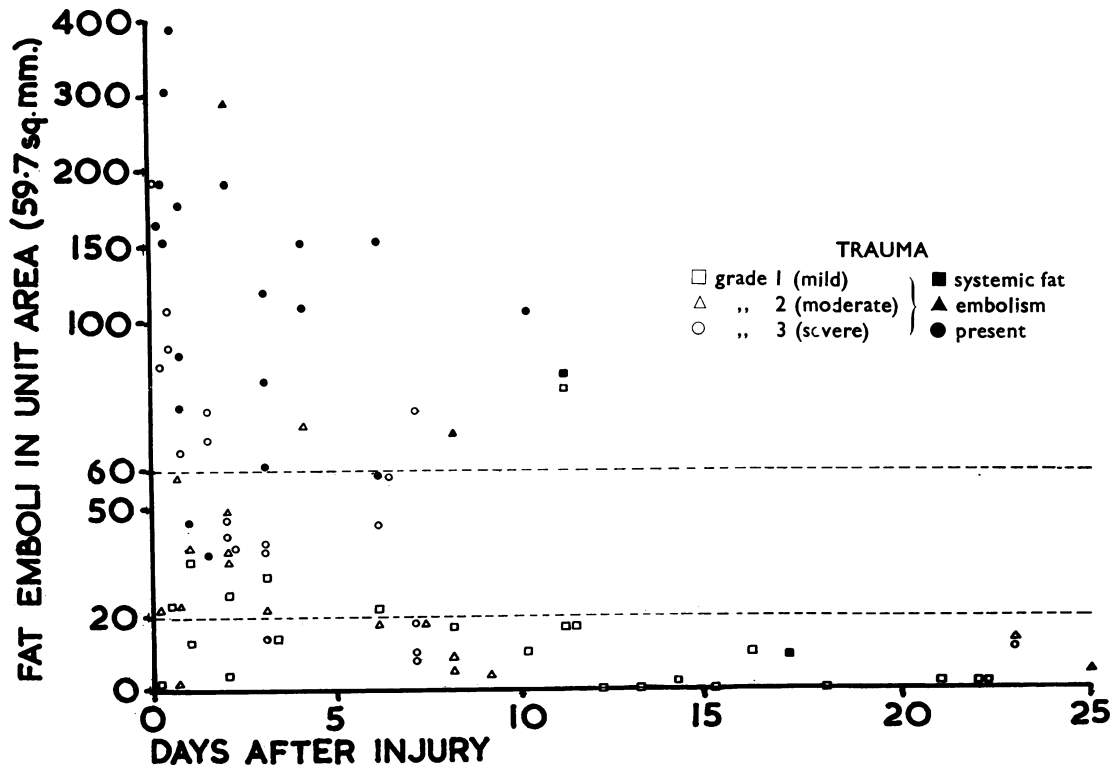


FIG. 1.—Incidence and severity of fat emboli after trauma related to time after injury.

Conversely, mild fat embolism is usually associated with mild trauma, but the correlation is not so close. The longer a patient survived after injury, the less the degree of fat embolism found (Fig. 1).

Incidence of Systemic Fat Embolism.—Each case of the trauma series was investigated for the presence of systemic fat embolism. Whenever possible sections of both kidneys and brain were examined, but both tissues were not available in every case. In no case where pulmonary fat embolism was absent was systemic fat found. Ninety-nine of the 100 cases were examined for renal fat emboli. The one case that was omitted showed neither pulmonary nor cerebral emboli. In 30 cases no sections of brain were available; in three of these renal fat emboli were present, but in the absence of cerebral sections the existence and significance of cerebral fat embolism had to be inferred from the degree of renal fat embolism and the clinical history.

Twenty-four of 100 cases showed systemic fat embolism. Distribution in respect of degree of trauma and of pulmonary fat embolism is analysed in Table IV. Of the 24 cases, 19 (80%) were

TABLE IV
CASES WITH SYSTEMIC FAT EMBOLISM CLASSIFIED BY DEGREE OF TRAUMA AND OF PULMONARY FAT EMBOLISM

	Degree of Trauma			Total	Total as Percentage of Cases in Group
	Mild	Moderate	Severe		
Pulmonary fat embolism	Mild	1	4	5	3.5
	Severe	1	2	16	14 61
Total	2	2	20	24	24
Total as percentage of cases in group	7	10	45	24	

patients with severe pulmonary fat embolism, 20 (83%) had suffered severe trauma, and 16 (66%) fell into both groups. This demonstrates the close relationship between severe trauma, severe pulmonary fat embolism, and systemic fat embolism. There remains one case in which mild trauma and mild pulmonary fat embolism are associated with systemic fat embolism; this has not been explained. Apart from this case, conclusions from this series confirm those of Scully (1956).

Effects of Pulmonary Fat Embolism.—Opinions differ widely on the significance of pulmonary fat embolism, ranging from the belief that it is the cause of grave symptoms and death in a high proportion of accident cases (Warthin, 1913; Robb-

Smith, 1941; Denman and Gragg, 1948; Glas, Grekin, and Musselman, 1953) to the opinion that it is only rarely the cause of symptoms (Wilson and Salisbury, 1944; Scully, 1956). Results of the present series reinforce the latter opinion, and have failed to confirm the existence of a syndrome due to severe pulmonary fat embolism. Characteristic gross findings were not apparent at necropsy, and study of the clinical records for unexplained pulmonary symptoms, e.g., cough, dyspnoea, bronchitis, pneumonia, collapse, or pulmonary oedema, showed that these were at least as common in cases with mild pulmonary fat embolism as in those where the embolism was severe. Of 35 cases of mild pulmonary fat embolism, nine (26%) showed such symptoms before death; of 28 cases of severe pulmonary fat embolism, four (14%) showed such symptoms. It is possible that pulmonary fat embolism might so affect a lung with a reduced reserve from pre-existing disease as to produce acute pulmonary insufficiency, and symptoms thought to be due to this were seen in one case in this investigation.

Effects of Systemic Fat Embolism.—While pulmonary fat embolism is a regular and relatively unimportant finding after fractures, systemic fat embolism was only found in 24% of the trauma series. Four cases showed cerebral fat emboli but no renal fat emboli. In two of these the absence of renal fat emboli is attributed to renal ischaemia in post-traumatic shock, as both were known to have been hypotensive for some hours and in one the kidneys showed ischaemic tubular necrosis. In the other two cases no explanation for the sparing of the kidneys could be found. A complete study of the necropsy findings and clinical records was made in 23 of the 24 patients; in the omitted case the clinical notes were not available.

The cases were separated into three groups: (1) Systemic fat embolism not contributory to symptoms or death (11 cases); (2) systemic fat embolism of doubtful significance in relation to symptoms and death (six cases); (3) systemic fat embolism giving rise to symptoms and death (seven cases). Of these seven cases (a) in four the systemic fat embolism was a contributory cause of death, and (b) in three systemic fat embolism was the main cause of death.

Comment

The frequency of fat embolism will always be found to be higher when assessed on histological as opposed to other criteria, as mild degrees of embolism may not give rise to symptoms. In previously published series the incidence of pul-

monary fat embolism found by histological examination of material from general necropsy series or in cases where injury was not the cause of death has ranged from 2% (Warthin, 1913) to 52% (Wright, 1932). The proportion considered to be of significance in causing symptoms was estimated at 1 to 2% (Warthin, 1913; Denman and Gragg, 1948). The proportion of cases of death after injury showing pulmonary fat embolism has been estimated at from 50% (Denman and Gragg, 1948) to 100% (Wyatt and Khoo, 1950). The number of deaths due to pulmonary fat embolism was estimated as 2% (Vance, 1931), 30% of 70 deaths after fracture (Robb-Smith, 1941), and 37% of 19 deaths from injury (Denman and Gragg, 1948).

When systemic fat embolism is assessed separately only Scully (1956) gives figures based on histological criteria. He found systemic fat embolism in 17% of fatal battle casualties, and considered death to be due to the condition in 1%.

Wilson and Salisbury (1944), using clinical criteria, found an incidence of systemic fat embolism of 0.8% in 1,000 battle casualties with death in 0.6%. Newman (1948), using clinical criteria, found an incidence of 6% of systemic fat embolism, with death in 3%, in a series of 89 injuries to long bones. Glas *et al.* (1953) estimated the incidence of systemic fat embolism in 109 cases of injury as 14% and thought death was due to it in 5.5%.

Not all these results are strictly comparable, as criteria applied vary between them. It is hoped that the strict quantitative histological criteria for the estimation of fat embolism, combined with study of clinical records, may serve as a reference in future investigations of the problem.

Removal of Fat Emboli.—Fat emboli are gradually removed from the tissues where they impact; Fig. 1 shows the steady decline in numbers of pulmonary fat emboli as the survival period after injury increases. Experimentally produced fat emboli are seen in the lungs “within seconds of injury” (Glas *et al.*, 1956). Maximal numbers are found in patients dying within 48 hours of injury, and after this there is a fairly steady decline. The time taken for the emboli to traverse the pulmonary capillaries and impact in the systemic vessels is less certain. The rule generally quoted for the signs of cerebral fat embolism is “fat embolism, 3 days,” but the time lapse after injury is now generally thought to be shorter than this (Allred, 1953) and in three patients in the present series the signs of cerebral fat embolism were apparent within 24 hours of injury.

Loss of fat emboli has been suggested to take place in the sputum, in the urine, by phagocytosis and by lipases. Large numbers of free fat globules can be found in the sputum after injury, and while this is not pathognomonic of fat embolism (Nuessle, 1951) it does show that a considerable amount of fat can be lost in this way. Fat does appear in the urine after injury, but it appears late and is difficult to detect, which suggests that quantitatively this route of elimination is not important.

Disappearance of fat emboli from the lungs can be demonstrated in those cases where an infarct preserves the number of emboli at the time of infarction, while in the remainder of the lung removal proceeds normally. In one such case there was survival for 23 days after injury. A lung infarct found at necropsy could be correlated with an episode of chest pain and haemoptysis seven days after injury. The count of emboli in the uninfarcted lung was 13; in the infarct it was 114, showing that in 16 days 101 emboli per unit area, or 88% of those present, had been removed.

Summary and Conclusions

Clinical records, necropsy findings, and histological preparations stained for fat have been studied in 100 patients who died after suffering injury and in 53 patients who died from burns or causes not due to injury. Pulmonary fat embolism was assessed quantitatively by counting emboli in a unit area of standard section and was classified as mild, moderate, or severe. In all patients of the trauma series and in some of the control series an examination was made for systemic fat embolism in kidney, brain, or both tissues.

Forty per cent. of the control cases showed pulmonary fat embolism, but in all save a few it was minimal and insignificant. In no case of the control series was systemic fat embolism found, nor were there symptoms suspicious of systemic embolism.

Eighty-nine per cent. of patients dying after injury showed pulmonary fat embolism. In the 11% without pulmonary fat embolism the average period of survival after injury was 40 days and the shortest 12 days. In no case was systemic fat embolism found in the absence of pulmonary fat embolism.

Systemic fat embolism was found in 24% of the patients dying after injury. In 11% it was considered to be of no significance, in 6% of doubtful significance, in 4% it was a contributory cause of death, and in 3% the chief cause of death.

Pulmonary fat embolism increases in severity with the degree of trauma. Systemic fat embolism is commonly found only in patients with severe pulmonary fat embolism.

The existence of a syndrome, or of characteristic gross necropsy appearances, due to severe pulmonary fat embolism has not been confirmed. Pulmonary fat embolism is not thought to be of importance as a cause of illness or death. Cerebral fat embolism is more important as a cause of symptoms and death than is commonly realized, but does not always give rise to the syndrome described as characteristic.

I am indebted to Dr. S. Sevitt for access to the pathological records and material, and for advice and encouragement throughout the work. My thanks are due to the surgeons of the Birmingham Accident Hospital for permission to utilize their clinical records, to Mrs. J. Priest and Mr. A. Randle for histological preparations, and to Mr. H. Lilly for Fig. 1.

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