CHAPTER 5
Fat Embolism

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► INTRODUCTION

Fat embolism (FE) is the partial obstruction of multiple blood vessels by fat globules. This is usually a temporary phenomenon. The fat embolism syndrome (FES) is the clinical manifestation of FE.

► HISTORY

In initial experiments, milk injected into the circulation of dogs produced FE. This was followed by oil-injection studies in the nineteenth century. However, the pathology and clinical picture were appreciated only in 1862, when fat was detected in the lungs of trauma patients. Zenker postulated that the fat in the lung was aspirated, but Wagner showed that fractures of bones released fat into the circulation.

The clinical manifestation of FE, that is, FES, was described in 1873 by Bergman, and 350 cases were described in 1913. Lehman and Moore in 1927 postulated a metabolic mechanism to explain the FES, while Sevitt drew a distinction between systemic and pulmonary fat emboli. Peltier concluded that the morbidity associated with FE was caused by fatty acids and associated inflammation.

► INCIDENCE

Fat emboli in the lungs of patients with skeletal trauma are common. Analysis of blood taken from the pulmonary artery of patients with long bone or pelvic fractures demonstrated that 7 percent of patients had fat emboli. Long bone fractures, of the lower extremities in particular, seem to cause fat emboli.

However, the presence of fat emboli is not synonymous with FES and, because of under reporting, differences in diagnostic criteria, and studies in different populations, the reported incidence of FES varies from 0.25 to 29 percent. Perioperative FES, after immediate fixation of fractures, has a reported incidence of 0 to 5 percent. A report on more than 900 cases of surgery for lower extremity fracture gave an incidence of 0.2 percent.

► PATHOLOGY

The etiology of FE and FES is classified as traumatic and nontraumatic.

Traumatic

The essential pathology is the entrance of marrow fat into veins. This process is enhanced by

- Multiple fractures
- Unstable fractures
- Increased pressure in the medullary cavity
- Shock, hypoxia, and stress, although this will not cause FES by itself

The attachment of veins to bone prevents their collapse; even during episodes of low venous pressure, such as circulatory shock. Fractures of bones with a higher density of vessels, such as the isthmus of the proximal femoral shaft, are more likely to cause the release of fat into the circulation.

Mechanics dictate that if the medullary pressure is raised when veins are torn, more fat will enter the
circulation. Hence closed fractures result in more fat emboli. Reaming of medullary spaces during surgery and nailing of prostheses (which raises the medullary pressure to 500 mmHg) tend to cause more fat emboli. Venting of the medullary cavity before inserting instruments therefore decreases the incidence of fat emboli. Young men are more prone than others to develop FES.

Burns and soft tissue injury can also result in FES, but it is less common, even though fat emboli are common.

Nontraumatic

These are rare causes of FES and they are classified as follows:

Procedure-Related

This group includes intraosseous fluid administration, intraoperative autotransfusion, and lipid-soluble radiocontrast.

Disease-Related

Sickle cell hemoglobin C, pancreatitis, fat necrosis of the omentum, and immunosuppressive disease are typical examples.

Drug-Related

Intravenous hyperalimentation, intraarterial cisplatinum, and long-term steroid administration are included here.

PATHOPHYSIOLOGY

Three main hypotheses have been put forward to explain FES:

Mechanical hypothesis

Biochemical hypothesis

A combination of mechanical and biochemical mechanisms

Mechanical Hypothesis

This hypothesis proposes that the embolized fat causes mechanical obstruction of the pulmonary and systemic capillaries. Fat has been demonstrated in the circulation soon after injury, and fat in the lungs of animals has been shown to originate from the bone marrow. Experiments in rabbits have demonstrated that raising the intramedullary pressure at a fracture site increased the extravasation of marrow fat. In humans, significant amounts of fat have been found in the ipsilateral femoral vein of a fractured femur during surgery. The lung capillaries are prominently affected in FE, and there appears to be a relationship between the decrease in arterial PaO₂ in FES and compressed closed fractures.

Neutral fat (not free fatty acids) can obstruct the pulmonary arterial system, causing acute pulmonary artery hypertension and acute right ventricular failure. The resultant decrease in cardiac output together with inflammatory changes in the lung will worsen hypoxia. Obstruction of capillaries is not restricted to the lung but occurs in the myocardium, brain, kidney, and other organs when fat enters the systemic circulation. The mechanisms by which fat enters the systemic circulation are either through a patent foramen ovale, which opens once the right atrial pressure is raised (because of acute pulmonary hypertension associated with acute lung injury or mechanical obstruction), or through the pulmonary capillaries. Patent foramina ovale were, however, not found in many patients with FES, and this supports the capillary transit theory. Another way in which fat may enter the systemic circulation is via pulmonary-bronchial anastomoses if the obstruction of the pulmonary bed results in reverse flow. Chylomicrons are small (1 µm in diameter) but, under the influence of mediators, coalesce into 10- to 40-µm fat globules, which are thought to obstruct the capillaries in the lung and the systemic capillary bed. One study demonstrated a significant increase in cholesterol in fat obtained from the pulmonary capillaries of patients with FES, but this observation has not been confirmed.

Biochemical Hypothesis

This hypothesis proposes that the fat globules in the systemic and pulmonary circulation originate from fat normally present in the blood, presumably altered by physical and more probably biochemical factors, resulting in either toxic or obstructive pathology.

The toxic hypothesis suggests that fat is broken down to free fatty acids (FFA), which cause injury to capillaries and pneumocytes. The resultant clinical picture is similar to that of acute lung injury with hemorrhagic lung injury to the pulmonary interstitium, edema, and pneumonitis. The effects of FFA on the pulmonary tissue have been confirmed and there is a correlation between serum FFA levels and hypoxia. However, this association does not necessarily imply causation. In addition, there is uncertainty about the origin of the FFA, with one group postulating that it is formed in the lung by the activity of pulmonary lipase on fat, while others postulate that it originates from the body fat. Trauma and catecholamines do result in FFA release, but whether this is important in the development of FES is uncertain. If one considers the number of surgical procedures performed and/or trauma cases managed annually, the relatively low incidence of FES argues against this theory as a sole explanation for FES.
studies have suggested that the serum albumin neutralizes the effect of FFA on the systemic circulation.\textsuperscript{41–43} There is also a suggestion that FFA is raised more in patients with decreased liver function. This hypothesis has some credibility, since shock, hypoxia, and anemia decrease liver blood flow. Under these circumstances, together with raised catecholamines, FE may well become a clinical entity.

**Combined Mechanical and Biochemical Hypothesis**

Because mechanical and biochemical hypotheses separately do not appear to completely explain the FES, a combination of the two has been proposed.\textsuperscript{5,34} The fat embolization of the capillaries by marrow is the initiating event and thereafter biochemical factors come into play.

**Other Theories**

Neutral fat has a thromboplastic effect, which activates the clotting cascade and could lead to disseminated intravascular coagulation (DIC) and eventually depletion of clotting factors. The end result is spontaneous bleeding.\textsuperscript{44} This tendency, coupled with the suppression of the fibrinolytic system by trauma, results in the accumulation of red blood cells, platelets, leukocytes, and fibrin in the microvascular bed, including the lung.\textsuperscript{55–47} The aggregates and clots will be filtered in part by the lung, and this contributes to the clinical picture of pulmonary parenchymal dysfunction often associated with FES. An inverse relationship has been demonstrated between platelet count and the degree of pulmonary dysfunction in these patients.\textsuperscript{48}

DIC is not always associated with FES; in the author’s practice, however, it does seem to occur regularly, albeit more often as subtle DIC patterns.

Another hypothesis is that trauma depresses the immune system, but the evidence for this as a cause of the FES is scanty.\textsuperscript{49}

**CLINICAL PRESENTATION**

FES usually presents in one of three degrees of severity:\textsuperscript{3}

- **Subclinical**
- Nonfulminant subacute
- Fulminant

**Subclinical Form**

The clinical signs are nonspecific and often confused with postoperative symptoms ascribed to pain, discomfort, or the postoperative inflammatory process. Tachypnea (defined as a breathing rate in excess of 25/min), elevated temperature (>37.8°C; 100.4°F) and tachycardia (>100 beats per minute) occur in the majority of patients with the subacute and mild form of FES.\textsuperscript{50} Approximately 50 to 90 percent of patients with lower extremity fractures suffer from some degree of hypoxia (defined as a PaO\(_2\) <80 mmHg; 10.6 kPa),\textsuperscript{51–52} and FES is viewed as the most likely explanation for the recorded desaturation of the arterial blood. Increased alveolar ventilation, resulting in various degrees of hypocapnia, also occurs in patients with subclinical FES.\textsuperscript{48} The latter study demonstrated a 48 percent incidence of thrombocytopenia (platelet count less than 200,000/µL), which has been regarded as an important indicator for FES\textsuperscript{48} and suggested to be linked to the lung injury in FES. However, some of the conclusions in this regard rest on indirect evidence (AaDo\(_2\)) and are uncertain, since the AaDo\(_2\) is affected by numerous factors and does not correlate well with the calculated shunt (Qs/Qt).\textsuperscript{53}

An echocardiographic study showed marrow particles in the right atrium in 60 percent of patients in whom bones had been manipulated during surgery.\textsuperscript{54}

**Nonfulminant Form (Subacute Form of FES)**

This is the more classic and well-known mode of presentation.

Patients often have *petechial rashes* on the anterior upper portion of the chest, especially in the areas adjacent to the axillae. Other areas commonly affected are the shoulders, mucous membranes (mouth), and conjunctiva.\textsuperscript{35,41,55} These rashes usually appear 12 to 96 h after the injury and may last from hours to a week. It is important that an active search be made for these telltale signs of FES. It has been suggested that the upper areas of the body are more affected because fat is lighter than blood, but this is at best an uncertain explanation. Histology of the skin lesions shows occlusion and distention of the capillaries with fat globules and increased permeability of the capillaries.\textsuperscript{56}

Neurologic signs are present in up to 86 percent of patients with FES.\textsuperscript{55,57} These vary in severity and include confusion, stupor, coma, and even decerebrate rigidity. Confusion is the more common presenting symptom and convulsions have been reported. Signs are nonlateralizing and are usually progressive, starting with confusion and progressing to more severe encephalopathy in many patients. Localizing signs such as hemiplegia, apraxia, visual scotomata, and conjugate eye deviation occur but are rare.\textsuperscript{57} The pathophysiology of neurologic dysfunction is thought to be the direct result not of cerebral hypoxia but rather of embolization. Cerebral edema may be induced by the FFA causing vascular disruption.\textsuperscript{10,58}

The *respiratory system* is often also involved in FES, and this syndrome is among the causes of acute lung
injury (ALI), the severe form of which is called acute respiratory distress syndrome (ARDS). The clinical picture includes tachypnea, cyanosis or some degree of arterial hemoglobin desaturation, and a respiratory alkalosis. The pathophysiology of the acute lung injury is that of poor pulmonary compliance resulting in tachypnea, low ventilation/perfusion ratios, either Qva/Qt or Qs/Qt causing hypoxia, and an increased Vd/Vt. This is accompanied by various degrees of pulmonary hypertension, which jeopardize right ventricular function and cardiac output, resulting in mixed venous blood desaturation. The latter will be aggravated by the raised temperature that commonly occurs in patients with FES. The combination of mixed venous blood desaturation and pulmonary shunt will further depress the arterial oxygenation.

The pulmonary effects of FES usually have a relatively benign course, and by the third day—barring new fat emboli or infection—the lung parenchyma starts to improve rapidly. There are reports of patients who had FES without pulmonary involvement. The retina is affected in approximately 50 percent of patients. In 4 percent of patients with long bone fractures, retinopathy presents subclinically. Eye lesions, consisting of cotton-wool spots and flame-like hemorrhages, are indistinguishable from the Purtscher retinopathy described in extraocular trauma. The cotton-wool areas are microinfarcts in the nerve fiber layer of the eye and the pathophysiology is the same as in other organs—i.e., capillary engorgement and loss of endothelial integrity. This results in hemorrhage, edema, and microinfarction. The lesions disappear in weeks but permanent central scotomata have been described.

**Fulminant FES**

This form of FES contains all the elements of nonfulminant FES but is more severe. It has been described after the release of limb tourniquets, reaming of the femur, insertion of intramedullary prostheses, closed reductions of fractures, and manipulation of a deformed hip. However, it can also occur in the absence of surgery and several hours after surgery. In the anesthetized patient, it may present as sudden hypotension, wheezing, tachycardia or bradycardia, poor lung compliance, pulmonary edema, cyanosis, or unexplained bleeding. Failure to regain consciousness after orthopaedic surgery and anesthesia should raise the possibility of cerebral FES.

In the unanesthetized patient, the signs and symptoms of the nonfulminant type occur, including more severe cerebral symptoms such as agitation, choreoathetosis, seizures, psychosis, or coma. These may precede fever, acute respiratory distress, significant hemodynamic instability, oliguria, renal failure, and clotting disorders. The lungs are affected in various ways: lung edema (even in the upper lung lobes) as well as reduced compliance and poor gas transfer resulting in hypoxia. Involvement of the upper lobe is explained by the hypothesis that fat is lighter than blood and hence will affect the upper zones. What argues against this idea is that the toxic effects of FFA damage the endothelium of the vasculature; hence areas with higher perfusion, such as the dependent parts of the lung, will be affected more. Another explanation for the upper lobe edema is the acute pulmonary hypertension associated with acute lung injury. This will increase perfusion of the upper lobes and may highlight the edema seen in those areas of the lung that are normally less well perfused (because of gravity). The picture can progress to a full-blown ARDS with all the clinical signs and symptoms of severe acute lung injury requiring respiratory support.

The right heart is severely affected by acute increases in the afterload. Acute lung injury is virtually always associated with ARDS, and data show that by the time the patient is admitted to the intensive care unit with a diagnosis of ARDS, acute pulmonary hypertension is already present. The patient will have tachycardia, raised jugular venous pressure, and raised right ventricular end-diastolic pressure. Acute cor pulmonale results in systemic hypotension, intraventricular septal shift, and low cardiac output. The arterial pressure will swing with the ventilator (pulsus paradoxus) because of the low ventricular end-diastolic volume; but because of the septal shift, the end-diastolic pressure in the left ventricle is raised, and this will aggravate the tendency to lung edema. Left ventricular ischemia occurs because of the low arterial pressure, and the risk of ischemic right and left ventricular failure is significant. The resultant low cardiac output, especially in the presence of fever, results in low mixed venous hemoglobin oxygen saturation. This, together with the shunt or low V/Q lung units, aggravates arterial hypoxia, which cannot be corrected with mechanical ventilation or increased inspiratory fraction of oxygen. Angina has been reported, which is presumed to result from emboli in the coronary arteries or cor pulmonale.

Acute renal failure occurs relatively seldom and it is thought that less neutral fat emboli enter the kidneys than other organs. In acute circulatory failure, as in cor pulmonale, the neurohumoral response of the body significantly decreases the renal blood flow, decreasing the number of renal emboli.

We have observed acute and severe diffuse intravascular coagulation and clotting disorder (DIC) during surgery on long bones. This occurred without other prodromata of the FES. The patient started to bleed spontaneously, and special examinations showed raised D-dimers, low fibrinogen and platelets, raised international normalized ratio (INR) and partial thromboplastin
time (PTT), and excessive levels of fibrinogen breakdown products. Some degree of clotting disorder is, in the author’s experience, very common in patients with the fulminant form of FES.

**SPECIAL EXAMINATIONS**

The FES is a syndrome and the diagnosis is therefore primarily a clinical one. Special examinations can help to confirm it and indicate the degree of organ dysfunction.

The arterial blood gases, more often than not, show a low PaO₂ and this is often described as a diagnostic criterion. However, there are many other reasons for a low PaO₂ and the absence of a reduction in arterial oxygen tension does not exclude the diagnosis. Hypoxia is usually accompanied by a respiratory alkalosis, but once mechanical respiratory failure occurs, the PaCO₂ increases because of alveolar hypoventilation, which results from respiratory muscle fatigue.

X-rays of the lungs are nonspecific and lag behind the clinical syndrome. In the presence of FES, the typical x-ray findings are useful and help to confirm the diagnosis. The typical pattern is that of a “snowstorm.” Bilateral patchy infiltrates occur mainly in the perihilar and basilar areas, usually sparing the apices. This classic picture can be modified by pulmonary edema, which can occur because of the septal shift associated with acute pulmonary hypertension and the resultant raised left atrial pressure or primary (usually ischemic) left ventricular failure.

Dilatation of the right heart (acute cor pulmonale) can sometimes be seen.

Computed tomography (CT) scans of the lungs show the subsegmental perfusion defects and ventilation/perfusion scans confirm the ventilation/perfusion mismatch. Indeed, CT and V/Q scan findings can be diagnostic when radiologic findings are negative.

A CT scan of the brain may show the absence of cortical sulci and compressed lateral ventricles due to brain edema. Hemorrhagic brain infarcts have been recorded. Acute cor pulmonale patterns and/or myocardial ischemia may be revealed by an electrocardiogram (ECG), depending on the severity of the FES and its effects on the pulmonary and coronary circulation.

Coagulation disorders, although not specific for FES, require quantification for effective treatment, since the INR, PTT, fibrinogen, d-dimer, fibrinogen breakdown products, and platelet count will identify either accelerated clotting and/or clot lysis. Fibrinogen, an acute-phase protein that should be increased because of the trauma and stress, is often decreased or may be normal because of the activation of the coagulation system. As the patient improves, the fibrinogen usually increases (within 3 to 5 days).

A progressive fall in hemoglobin has been reported, with a 30 percent decrease over 48 h in 75 percent of patients with FES. This is thought to be the result of intralveolar hemorrhage. However, this hypothesis is uncertain, since a similar aggressive decrease in hemoglobin does not regularly occur in patients with other causes of ARDS.

Cytologic examination of urine and sputum could be useful in confirming the clinical diagnosis. Sudan red staining will detect free fat globules in the macrophages. This procedure takes about 2 to 3 h, but its value is questionable, since it has given negative results in a group of patients with a positive diagnosis of FES. This highlights the point that FES is primarily diagnosed clinically, and the value of confirmatory tests is subject to their specificity and sensitivity.

The cryostat test can be used to identify fat globules in blood. Peripheral blood is rapidly frozen and the clot sectioned for microscopic examination. This test has a limitation in that most of the fat will be found in the veins draining the source area and not in the peripheral blood. Because the fat globules are trapped in the pulmonary capillaries, a blood sample from a pulmonary artery catheter in a wedged position would increase positive results for the test. Bronchoalveolar lavage (BAL) may contain cells with fat droplets. This was seen in 31 percent of patients with FES but in only 2 percent of those without FES.

Blood lipids do not correlate with the severity of FES. Activated complement (C5a) increases in FES, but this increase is not specific for this syndrome.

**DIAGNOSIS**

The only pathognomonic sign for fat embolism is the petechiae. Other clinical signs and symptoms and most of the readily available special examinations are nonspecific. However, FES is a syndrome, and the diagnosis rests on a constellation of findings. If all the signs and symptoms are present, the diagnosis is usually not difficult; but if some of them are lacking, the diagnosis may be uncertain.

For patients with lower body skeletal trauma, Gurd and Wilson have compiled the following diagnostic criteria:

- One of the major manifestations; i.e., petechiae and pulmonary or cerebral involvement
- Four of the five minor manifestations; i.e., pyrexia, tachycardia, jaundice, renal or retinal changes, and fat macroglobulinemia

The approach of Gurd and Wilson is limited by the absence of arterial blood gas findings and other lung pathology descriptors. Lindeque therefore expanded
on the Gurd and Wilson approach by including the following:

\[ \text{PaO}_2 < 60 \text{ mmHg (8 kPa)} \text{ on a FiO}_2 \text{ of 0.21} \]
\[ \text{PaCO}_2 > 55 \text{ mmHg (7.3 kPa)} \text{ or a pH < 7.3} \]

- Respiratory rate >35 breaths per minute (even after sedation)
- Increased work of breathing as manifest by dyspnea, use of accessory muscles, and a tachycardia

As can be seen from the above, the criteria for diagnosis are fairly nonspecific except for petechiae. Even the inclusion of the lung descriptors does not make criteria more specific, since the latter could signify lung injury without FES.\(^3\)

Based on individual organ dysfunction, which can occur in FES, there is an extensive list of differential diagnoses that should be considered.\(^3\) However, the key issues in diagnosing the syndrome remain a high degree of suspicion, petechiae and other organ involvement. The timing of the clinical signs may also assist in the diagnosis. For example, the development of petechiae soon after bone manipulation or injury would very likely suggest FES; on the other hand, if it occurs after 3 days and after stabilization of the fractures, the diagnosis is less likely (although not excluded).

The clinical picture may differ in patients under general anesthesia as well as muscle relaxation and mechanical ventilation, since the telltale cerebral and pulmonary signs can obviously not be observed in them.

**TREATMENT**

There is no specific treatment for FES; the only option is to continue life support until the patient recovers.

The management of shock is very important in the outcome of FES. Data indicate that mortality was higher in patients with systolic blood pressure <100 mmHg and a heart rate in excess of 120 beats per minute.\(^5\) Because this was a retrospective study and it is not ethical to do a trial in which hypovolemia and incipient shock is not treated, it is not known whether the increased mortality was due to FES and incipient shock or hypovolemia and shock alone. It was most likely the latter. Hemodynamic shock alone can cause acute lung injury, and this could well be aggravated by FES. In addition, hypovolemia in the presence of acute pulmonary hypertension could have a significant effect on the cardiac output.

Excessive fluid loading is detrimental to the thin-walled right ventricle in the presence of an acute increase in afterload. Intravascular fluid restoration should therefore be done cautiously and monitored closely. A central venous catheter is valuable, since it will give indirect guidance as to the volume status and also indicate whether the right ventricle is under strain. The latter can be gauged from the central venous pressure response to a small fluid load, abnormal a or c waves, and the appearance of acute tricuspid incompetence. In severe FES, a pulmonary artery catheter is necessary to determine right ventricular failure. The latter is diagnosed by critically evaluating the right ventricular stroke work (RVSW) against the pulmonary artery pressure. If RVSW remains normal or does not increase as the pulmonary artery pressure increases, right ventricular failure is occurring. This implies that the function of the right ventricle changes from maximal efficiency to maximum stroke work. This is indicated by lower than expected RVSW in the presence of a raised pulmonary artery pressure. It is mandatory to support right ventricular function with inotropes and to maintain the systemic pressure so as to prevent hypotension and right ventricular ischemic failure. The choice of inotrope depends on the systemic blood pressure: if the perfusion pressure is still normal, dobutamine is useful. If the perfusion pressure is low, an alpha agonist is required and epinephrine or norepinephrine is a more appropriate choice. Pulmonary vasodilators are singularly unsuccessful, and since drugs like isoproterenol will cause systemic hypotension and increase the pulmonary shunt, they should not be used. The same can be said for all of the other generally available nonspecific vasodilators. The role of inhaled nitric oxide has not been established, and in view of the mechanical theory of obstruction, it is doubtful that it would be helpful.\(^3\) If there is significant pulmonary vasoconstriction in addition to the capillary obstruction, then there would be reason to expect that nitric oxide could help in unloading the right ventricle (and improving arterial oxygenation).\(^3\)

Studies indicate that albumin should be used for volume restoration because, in addition to this, it binds the FFA and thereby reduces the potential for lung injury.\(^2,3\) Excessive fluid will, as in all cases of acute lung injury (ALI), increase the amount of interstitial fluid in the lungs and could worsen the venous admixture or pulmonary shunt.

Ventilatory support is often mandatory to restore arterial oxygenation. The decision to support oxygenation and ventilation rests on known criteria. These maneuvers include additional inspired oxygen, noninvasive and invasive ventilation. Given that the basic pulmonary pathology is that of low ventilation/perfusion units resulting from a reduction in the functional residual capacity, the judicious application of positive end-expiratory pressure with or without lung recruitment is valuable. The management of mechanical ventilation and alveolar pressure is guided by the current knowledge of volume trauma, and protective ventilatory strategies should be employed when dealing with these patients.\(^2,3\)

Of all the causes for acute lung injury, fat embolism is one of the more benign etiologies. The acute ventilatory difficulty usually lasts for approximately 3 days, after
which the patient often improves and can be weaned from ventilatory support. Unfortunately associated problems—such as acute renal failure, right ventricular failure, or pulmonary infection—can intervene and will obviously modify the course of the disease.

Although cardiac and pulmonary fat embolectomy has been performed, it is of little value, since the emboli are in the capillaries. High doses of corticosteroids have not been shown to be of value in ALL. Because FES is often an isolated problem and generally has a less malignant course than, for instance, ALI caused by sepsis, it has nevertheless been suggested that steroids, given early in the course of the disease, could be beneficial. Aprotinin has been administered because it decreases platelet aggregation and the release of sero-

tin. A retrospective study demonstrated an increased recovery and decreased mortality when aprotinin was used in FES. Another study confirmed that aprotinin will lessen the reduction in platelet count but does not prevent the pulmonary effects of FES.

For the prevention and treatment of diffuse intravascular clotting disorder, heparin should be used. For prevention 300 U of unfractionated heparin per hour administered intravenously was successful in preventing thrombin-induced DIC in baboons. For treatment of established DIC, it is mandatory to know whether the DIC is in the accelerated clotting phase, clot-lysis phase, or both. This can be achieved by means of a complete coagulation scan. Heparin dosage can then be titrated against the pathology, and once the accelerated clotting is controlled, clotting factors can be given to replace those used in the process.

There is a theoretical argument that heparin may be detrimental because it raises the FFA significantly. However, the need to prevent or treat DIC in these patients, outweighs this possible negative effect, and the author elects to use heparin as described.

► PREVENTION

Early stabilization of fractures is important in an effort to prevent the FES; this will reduce its incidence and decrease the incidence of septic and pulmonary complications (see Chap. 16). Intraoperative drilling of a venting hole before inserting a prosthesis reduces the intramedullary pressure but does not prevent the FES in every patient.

The use of a tourniquet has been proposed as a method of preventing the fat from reaching the systemic circulation. However, once the tourniquet is released, the fat will still reach the systemic circulation. In addition, the accumulated fat could cause severe FES; this method is therefore not recommended.

The only drug that seems to have some (still debatable) value is corticosteroid. There is great variation in the suggested dose, from 9 mg/kg over 2 days to 90 mg/kg over 4 days.

► PROGNOSIS

The general mortality rate associated with FES is 10 to 20 percent. However, it appears that early fixation of fractures reduces the mortality to less than 10 percent (see Chap. 16).

There are, however, publications suggesting higher and lower mortalities. Whereas Burgher reported a mortality of 33 percent, Guenter reported no deaths in 54 patients.

Death is more often than not the result of respiratory insufficiency, but it is difficult to collect accurate data, since there may be many intervening problems that can be fatal.

Permanent cerebral injury can occur, especially in young patients with the fulminant variant of the disease.

REFERENCES


