Review

FAT EMBOLISM SYNDROME

Michael J. Johnson, MD
George L. Lucas, MD

ABSTRACT

Since it was initially described, fat embolism syndrome (FES) has remained one of the least clearly understood complications of trauma. This article is a review of the classic and current literature on FES with regard to its causes, pathophysiology, clinical presentation, diagnosis, and treatment. FES is associated with many traumatic and nontraumatic conditions, but is most commonly associated with fractures of long bones of the lower extremity. The pathophysiology is thought to be a cascade of events which can lead to adult respiratory distress syndrome (ARDS). Signs and symptoms of clinical FES usually begin within 24 to 48 hours after trauma. The classic triad involves pulmonary changes, cerebral dysfunction, and petechial rash. Clinical diagnosis is key because laboratory and roentgenographic diagnosis is not specific. Treatment consists of careful initial handling, early stabilization of fractures, careful volume replacement, analgesia, respiratory support, and perhaps steroids. The vast majority of patients today survive FES without sequelae.

Since 1873, when Von Bergmann first diagnosed fat embolism syndrome (FES) in a man with a fractured femur, FES has remained one of the least clearly understood complications of trauma. FES and fat embolism are not synonymous; FES is a phenomenon secondary to fat embolism.

Fat embolism syndrome is a cascade of events that is the infrequent complication of fat emboli primarily from fractures of long bones and the pelvis. Clinical manifestations are primarily various degrees of respiratory insufficiency, cerebral changes, and petechiae. Although the incidence of FES is controversial, it still is considered to have a significant incidence in the trauma setting. As a clinical syndrome, FES is thought to occur in 0.5% to 3.5% of isolated long-bone fractures and in 5% to 10% of patients with multiple skeletal trauma. The incidence of fat embolism is unknown but is undoubtedly much higher. The overall mortality rate for clinical FES is still significant at 5% to 15%.

This article is a review of the classic and current literature of FES with regard to its causes, pathophysiology, clinical presentation, diagnosis, and treatment.

CLINICAL SETTINGS

FES has been associated with many traumatic and nontraumatic conditions; it is associated most commonly with fractures of the long bones of the lower extremity, particularly the shaft of the femur or tibia and the pelvis. As mentioned, the incidence of clinically apparent FES is 0.5% to 3.5% for isolated long-bone fractures and 5% to 10% for multiple trauma. As a subclinical syndrome, fat embolism is thought to occur in almost all pelvic and lower extremity skeletal trauma.

Surgery on the lower extremity also predisposes the patient to FES. Based on the literature, the most common operations to predispose to FES are total hip arthroplasty, total knee
Table 1

**CLINICAL SETTINGS FOR FAT EMBOLISM SYNDROME**

<table>
<thead>
<tr>
<th>Setting</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trauma</strong></td>
</tr>
<tr>
<td>Lower extremity long-bone fractures</td>
</tr>
<tr>
<td>Pelvic fractures</td>
</tr>
</tbody>
</table>
| Child abuse without fractures
| Blunt concussion             |
| Liver trauma                 |
| Severe burns                 |
| Massive soft tissue injury   |
| **Surgery**                  |
| Total joint replacement      |
| Total hip arthroplasty       |
| Total knee arthroplasty      |
| Intramedullary nailing of femoral shaft |
| Closed femoral osteotomy    |
| Femoral elongation          |
| Spinal fusion               |
| Liposuction                 |
| Bone marrow transplantation |
| Renal transplantation       |
| **Nontrauma**                |
| External cardiac massage    |
| Lipid emulsions in long-term hyperalimentation |
| Intraosseous venography      |
| Acute hemorrhagic pancreatitis |
| Carbon tetrachloride poisoning |
| Prolonged corticosteroid therapy |
| Fatty liver secondary to alcohol |
| Acute osteomyelitis          |
| Bone infarction secondary to sickle-cell disease |
| Epilepsy                     |
| Diabetes mellitus            |
| Extracorporeal circulation  |
| Severe infection (especially *Clostridia* spp. |
| High-altitude flights        |

arthroplasty,16-20 and intramedullary nailing of the femoral shaft.21-23 Other orthopedic procedures have been associated with FES, including femoral elongation,24 closed femoral osteotomy,25 and spinal fusion.26

Other nonorthopedic surgical procedures have been implicated in the development of FES, most notably liposuction27-30 and bone marrow transplantation.31,32 Many nontraumatic, nonsurgical conditions also can lead to FES. Because of the frequency of closed cardiac massage, it may be the most common cause of FES after trauma.33,34 (For a complete list of clinical settings for FES, refer to Table 1.) In addition, Peltier34 noted that predisposing factors include osteopenia and preexisting heart and pulmonary disease.

The incidence of FES in these settings is much more common in adults than in children. According to Gossling and Pellegrini,2 "children develop clinical manifestations almost 100 times less frequently than do adults with comparable injuries, presumably because of a differing marrow fat content with smaller amounts of liquid triolein."

**PATHOPHYSIOLOGY**

Of all the aspects of FES, its pathophysiology remains the most controversial; many theories have been proposed during the past 150 years. Theories in the current literature involve some common mechanisms that are believed to be responsible for fat emboli in the lung microvasculature manifesting as FES (Fig).

It generally is agreed that the source of fat emboli in the trauma setting is the bone marrow of long bones at the site of the injury, and it is thought that nonsusseous fat usually is not the source of emboli.55 Fat droplets gain access to the vasculature immediately by intravasation. As Peltier54 suggests, “intravasation is promoted by repeated manipulation or failure to splint fractures promptly and efficiently.”

The fat droplets then embolize primarily in the lung, with a small amount embolizing to other organs such as the brain. According to Peltier,54 initially the effect of fat emboli on the lung is mechanical. This can cause an increased perfusion pressure, which, if severe enough, could lead to right heart strain or failure secondary to pulmonary hypertension. This concept is considered important in the development of “fulminating” FES.56,57 However, many autopsy studies58,59 have shown that there is not much correlation between the presence and amount of intravascular fat and the severity of clinical manifestations. Therefore, it is theorized that FES is more than just simple mechanical obstruction.

Once the neutral fat emboli are trapped in the lung microvasculature, the lung responds by
secretion of lipase. Lipase then hydrolyzes the non-toxic neutral fat into free fatty acids (FFAs) and glycerol, which have been shown to be chemically toxic to the lung parenchyma.60,61

The free fatty acids then cause a severe inflammatory reaction. The inflammatory reaction causes complement-mediated leukocyte aggregation, which releases chemotoxins from these cells, which causes endothelial damage, alveolar architecture injury, increased capillary permeability, and damaged lung surfactant.62-64

These events lead to adult respiratory distress syndrome (ARDS).65 The time delay between initial fat embolization and the development of FES is thought to be related to the time required for the metabolic conversion of neutral fat to FFAs.66

Some controversy also surrounds the pathophysiology of cerebral dysfunction associated with FES. Again, there is some definite mechanical destruction secondary to small vessel occlusion with hemorrhages primarily throughout the white matter of the cortex. However, as with the lung, the severity of the neurologic deficit does not correlate with the amount of fat in the brain.59 It has been suggested that elevated levels of FFAs in the cerebral circulation also damage the cortex in a process similar to that occurring in the lungs.67

**Clinical Presentation**

Signs and symptoms of clinical FES usually begin within 24 to 48 hours after trauma, with the mean time of onset being 40 hours.68 but may appear almost immediately.43 The classic triad of FES involves pulmonary changes, cerebral dysfunction, and petechial rash. If this triad occurs 1 to 2 days after trauma, it is virtually pathognomonic for FES.33

Pulmonary findings usually are the earliest signs of the syndrome. Pulmonary insufficiency occurs in 75% of patients with FES,69 and presents commonly as tachypnea, dyspnea, and cyanosis. Rales and rhonchi often can be heard. Hypoxemia may be detected hours before the onset of respiratory symptoms.70 Approximately 10% of these patients progress to respiratory failure.71

Cerebral changes occur in up to 86% of patients with FES.57 and they can manifest in a wide range of presentations from headache, lethargy, or irritability to delirium, stupor, convulsions, or coma. Neurologic signs may be the primary presentation, but it is rare for these patients not to develop respiratory signs.72 Focal neurologic findings such as hemiplegia or hemiparesis are rare, but have been seen. Favorable prognostic signs include normal muscle tone with active deep tendon reflexes and retention of appropriate pain response.70 Although normal arterial oxygen level restoration has little effect on central nervous system manifestations,73 if the patient survives the respiratory insufficiency, the neurologic dysfunction is almost always reversible.

Petechia occurs in 50% to 60% of the patients69 and often appears after a delay of 24 to 48 hours.70 Lesions are found most often over the anterior chest, neck, axillae, oral mucous membrane, and conjunctivae and are probably secondary to the embolization of fat within the dermis.74 The distribution is theorized to be related to fat particles floating in the aortic arch—like oil in water—and are embolized to nondependent skin areas via subclavian or carotid arteries.75 The petechia usually resolves within 7 days.76

In addition to the classic signs and symptoms of FES, several other signs may occur but are more nonspecific. Tachycardia and pyrexia are common, but nonspecific features of FES.77 Retinal changes may include exudates, cotton-wool spots, edema, hemorrhage, or intravascular fat globules.78,79 Additionally, two self-limiting signs may include renal change secondary to embolism, which may present as lipuria,80 or hepatic changes, which may present as jaundice.69

As mentioned, FES can present in a variety of signs and symptoms; it also can present in a wide range of severity. Sevitt66 classified FES into three distinguishable clinical presentations. First, a subclinical FES probably occurs in almost all long-bone fractures of the lower extremity.77-10 This is characterized by decreased PaO₂ and minor hematologic changes such as thrombocytopenia and decreased hemoglobin,10 with no clinical signs or symptoms of respiratory insufficiency. The second syndrome is a clinically apparent, nonfulminant FES, in which the manifestations of respiratory insufficiency, cerebral changes, petechial, and typical laboratory and radiographic changes are seen. The third, rarer, form is a fulminant type that can be seen within hours of injury and results in severe physiologic impairment including respiratory failure and altered mental status.81-83 This third form usually occurs in the absence of operative intervention.84

FES is only one of many conditions that can lead to adult respiratory distress syndrome (ARDS). ARDS has a variety of origins, such as sepsis, trauma, aspiration, or infection. Despite these heterogeneous causes, it has a relatively homogeneous clinical picture of noncardiogenic pulmonary edema, hypoxemia, and decreased pulmonary compliance. Fortunately, FES only rarely leads to ARDS.
Table 2

GURD'S Diagnosis of FES

<table>
<thead>
<tr>
<th>Major Features</th>
<th>Minor Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory insufficiency</td>
<td>(at least one)</td>
</tr>
<tr>
<td>Cerebral involvement</td>
<td>Pyrexia</td>
</tr>
<tr>
<td>Potential rash</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>Retinal changes</td>
<td>Retinal changes</td>
</tr>
<tr>
<td>Jaundice</td>
<td>Renal changes</td>
</tr>
<tr>
<td>Laboratory features</td>
<td></td>
</tr>
<tr>
<td>Fat macroglobulinemia</td>
<td>(at least four)</td>
</tr>
<tr>
<td>Anemia</td>
<td>Pyrexia</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>High erythrocyte sedimentation rate</td>
<td>Retinal changes</td>
</tr>
</tbody>
</table>

Reprinted with permission from the British Journal of Hospital Medicine.

Table 3

Schonfeld's Diagnosis of Fat Embolism Using a Fat Embolism Index

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Petechiae</td>
<td>5</td>
</tr>
<tr>
<td>Diffuse alveolar infiltrates</td>
<td>4</td>
</tr>
<tr>
<td>Hypoxemia (P/O2 &lt; 9.3 kPa)</td>
<td>3</td>
</tr>
<tr>
<td>Confusion</td>
<td>1</td>
</tr>
<tr>
<td>Fever &gt; 38°C</td>
<td>1</td>
</tr>
<tr>
<td>Heart rate &gt; 120 beats/min</td>
<td>1</td>
</tr>
<tr>
<td>Respiratory rate &gt; 30/min</td>
<td>1</td>
</tr>
</tbody>
</table>

A score > 5 is considered diagnostic. Reprinted with permission from the British Journal of Hospital Medicine.

Diagnosis

Clinical. Laboratory, radiologic, and electrocardiographic findings in FES are either too sensitive or not specific enough to be pathognomonic for FES. Therefore, clinical findings are the cornerstone for diagnosing FES.

An important component of diagnosis is clinical suspicion in patients who are in a setting for development of FES, such as a patient with a long-bone fracture who develops symptoms 1 to 2 days after trauma. With regard to clinical diagnosis, there is no standard method, although some authors have suggested some aids in clinically diagnosing FES. Gurd and Wilson have divided symptoms of FES into major and minor features (Table 2). They propose that if any one major and four minor features plus fat macroglobulinemia are present, after a latent period after injury, then the diagnosis of FES can be made.

Murray and Raczy have proposed that the presence of tachycardia, tachypnea, pyrexia, and central nervous system manifestations with arterial hypoxemia were the diagnostic indicators. Schonfeld et al proposed a quantitative means of diagnosing FES as shown in Table 3. Seven signs were assigned a score, with a cumulative score greater than five being necessary for a positive diagnosis of FES.

Laboratory. Many laboratory tests have been considered to be diagnostic of FES. Many of these tests, however, tend to be nonspecific and positive results are found in trauma patients with or without FES. Unquestionably, the most useful diagnostic test is arterial blood gas analysis. Development of FES causes the PaO2 to decrease to 50 mm Hg or less within the first 72 hours. Peltier suggested that arterial blood gases be done early and frequently in all patients with significant skeletal trauma. Lindeque et al proposed that the diagnosis of FES can be made on the basis of respiratory status alone. The proposed criteria for a positive diagnosis of FES are: 1) sustained PaO2 less than 60 mm Hg; 2) sustained PaCO2 greater than 55 mm Hg; 3) pH less than 7.3; 4) sustained respiratory rate greater than 35 breaths per minute despite adequate sedation; and 5) increased work of breathing. If any one of these criteria is present in a patient with a long-bone fracture, then a diagnosis of FES can be made.

Hematologically, various abnormalities can occur. Anemia caused by a decrease in hemoglobin of 3 to 5 g/dl can occur. In addition, thrombocytopenia is often associated with FES. Peltier suggested that thrombocytopenia levels of less than 150,000/µl are diagnostic of FES. Despite thrombocytopenia and other abnormal coagulation laboratory parameters, a clinical bleeding disorder is rarely a problem with FES.

Other various laboratory tests for FES have been proposed and include elevated plasma FFA levels secondary to lower serum albumin levels, which is probably a result of increased capillary permeability. Hypocalcemia also is often reported, and some propose that it may be of prognostic value. In a general stress response to injury, elevations in cortisol, glucagon, and catecholamines are also often seen.

Positive test results for such things as fat globules in urine, sputum, cerebrospinal fluid, and blood and elevated serum lipase can occur in significant numbers of patients with FES. However, the tests tend to be too sensitive to be of clinical value.

Recent reports in the literature propose that bronchoalveolar lavage may be of diagnostic
value in FES. Chastre et al. suggested that fat droplets within cells recovered by lavage may be both rapid and specific in diagnosing FES. Nimi et al. suggested that intra-alveolar fat globules and hemorrhage are consistent with the diagnosis of FES.

Radiographic. Radiography represents a nonspecific aspect of the diagnosis of FES. The chest radiographs usually yield normal findings in mild cases of FES; even in severe cases of FES, the chest radiographic findings are normal initially but usually will change during the next 72 hours. Changes usually begin as a diffuse, fluffy, bilateral infiltrate. If FES is severe, these infiltrates often will progress to increasing diffuse opacity consistent with the capillary permeability edema of FES. This may progress to widespread air space consolidation caused by alveolar hemorrhage and edema consistent with ARDS of any cause.

When diagnosing FES by chest radiographs, a differential diagnosis including cardiogenic edema and traumatic contusion must be considered (Table 4).

In addition to routine chest radiographs, some authors have suggested the use of radionuclide scans for the diagnosis of FES. They propose it may be of particular use in equivocal cases of FES with normal or borderline chest radiographic findings or a questionable clinical presentation. They report that ventilation/perfusion images show “matching” defects. They have proposed that these images, although nonspecific, may provide the earliest clue for the diagnosis of FES.

Electrocardiographic. With FES, the electrocardiographic findings are usually normal. When a change is present, signs of right heart strain or failure are likely to be seen. Nonspecific T wave abnormalities have been observed and may represent ischemia or hypoxia.

**TREATMENT**

In the treatment of FES, initially fracture first aid is important. The fractured extremity must be handled gently and splinted properly, and the patient transported carefully. Early immobilization definitely decreases the incidence of FES. Immediate open reduction and internal fixation within 24 to 48 hours decreases the development of FES over conservative treatments such as traction or casting. The incidence of FES is increased when internal fixation is delayed. Bone et al. strongly recommended open reduction and internal fixation within 24 hours of injury to significantly reduce the incidence of FES.

Prevention of hypovolemic shock is important and restoration of adequate blood volume must be performed immediately, because shock has been shown to increase the incidence of FES. Human albumin has been advocated for fluid replacement, because it decreases the incidence of FES in patients with multiple trauma by binding free fatty acids.

Careful monitoring of blood pressure, urinary output, and possibly pulmonary wedge pressure is helpful for shock. Overly aggressive fluid resuscitation may worsen the patient's pulmonary status; if a perfusion can be maintained, judicious use of a diuretic may be helpful. Analgesia is important to limit the sympotomimetic response to injury. Care must be taken not to oversedate the patient and thus depress respiratory drive.

Respiratory support is the mainstay of the treatment of FES. Early and frequent monitoring of respiratory function, either by pulse oximetry or arterial blood gases, is advocated for patients at risk for FES. Respiratory support can range from the use of nasal cannula to mechanical ventilation. Peltier suggested that immediate administration of oxygen (40%) via face mask or nasal cannula be applied to all patients with significant fractures. A face mask or nasal cannula may be all that is necessary. However, if persistent or worsening hypoxemia (paO2 < 60 mm Hg) and increasing respiratory distress are present despite supplemental oxygenation, then mechanical ventilation may be necessary. Continuous positive airway pressure may be helpful in avoiding mechanical ventilation. Positive end-expiratory pressure is advocated in conjunction with mechanical ventilation to avoid oxygen toxicity.

With regard to drug therapy, many agents have been proposed and recommended by their supporters, but most are not considered as cur-

<table>
<thead>
<tr>
<th>Table 4</th>
<th>RADIOGRAPHIC DIFFERENTIAL DIAGNOSIS OF FES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FES</td>
</tr>
<tr>
<td>Distribution</td>
<td>Peripheral 101</td>
</tr>
<tr>
<td>Cardiac enlargement</td>
<td>No</td>
</tr>
<tr>
<td>Signs of pulmonary hypertension</td>
<td>No</td>
</tr>
<tr>
<td>FES</td>
<td>Traumatic</td>
</tr>
<tr>
<td>Onset of radiographic changes after trauma</td>
<td>1-2 days 99, 100</td>
</tr>
<tr>
<td>Time for radiographic findings to return to normal</td>
<td>7-10 days 102</td>
</tr>
<tr>
<td>Location of radiographic changes</td>
<td>Bilateral &amp; symmetrical 133</td>
</tr>
</tbody>
</table>
rent components of regimens for FES. Alcohol has been proposed because it decreases serum lipase activity, thereby decreasing lipolysis of neutral fat emboli and production of FFAs. Several studies have exhibited equivocal findings and it is not currently recommended for FES. Heparin has been suggested because it increases lipase activity and thereby decreases the amount of circulating fat. Because of the possibility of elevation in FFA levels secondary to increased lipase activity, concerns about anticoagulation side effects, and other studies showing increasing mortality, heparin is not recommended. Dextran 40 was recommended because of its ability to reduce red blood cell aggregation, expand plasma cell volume, and decrease blood viscosity. Because of dextran's anticoagulation effects, risk of inducing renal failure, and other study findings, dextran is no longer used for FES.

The most studied and most promising agents for the treatment of FES are corticosteroids, which have been studied for both prophylaxis and treatment. Many studies have touted the beneficial effects of corticosteroids. The mechanism of action appears to be inhibition of the inflammatory reaction associated with FES, including leukocyte aggregation, and a decrease in the increase of plasma FFA levels. Corticosteroids appear to limit the decrease in $P_{aO_2}$ seen with FES.

It appears that corticosteroids hold promise for a specific drug treatment for FES in conjunction with respiratory support and other supportive measures. As for the particular regimen, it has varied from that recommended by Kallenbach et al., 1.5 mg/kg of methylprednisolone every 8 hours for 4 doses, to that recommended by Lindeke et al., 30 mg/kg of methylprednisolone every 2 hours for 2 doses, to that of Schonfeld et al., 7.5 mg/kg of methylprednisolone every 6 hours for 12 doses.

MORBIDITY AND MORTALITY

Most commonly, FES is self-limiting, and pulmonary function returns to normal if adequate supportive care is given.

Despite increasing knowledge of its pathophysiology and advances in treatment, FES still remains a significant cause of morbidity and mortality in the posttraumatic setting. Defining the mortality rate is difficult because of its wide range of associated injuries, from an isolated fracture to multiple organ trauma. Its mortality has decreased since the 1960s and 1970s, but it is thought that clinical FES still has a mortality of 5% to 15%. Mortality is closely correlated with the degree of respiratory complications, which is the primary cause of death in most patients. Most long-term morbidity is secondary to focal cerebral neurologic deficits. The fulminant form of FES still carries a higher mortality than does most clinically apparent FES.

CONCLUSION

Fat embolism, usually subclinical, still occurs in the majority of patients with long-bone fractures, particularly of the femur and tibia. Despite certain laboratory and radiologic diagnostic aids, clinical diagnosis is still the cornerstone of diagnosis of FES. Respiratory insufficiency, central nervous system changes and petechiae 1 to 2 days after injury is still considered pathognomonic of FES. Treatment consists of aggressive supportive care including fracture immobilization, respiratory support including possible mechanical ventilation, judicious volume replacement, analgesia, and corticosteroids in certain settings. Fortunately, most treated patients today survive FES without sequela.

REFERENCES

20. Orsini EC, Richards RR, Mullen JM. Fat embolism during
fat embolism following total knee arthroplasty. Minn Med.
22. Lachiewicz PF, Ramaswamy CS. Fat embolism syndrome follow-
23. Hugh J, Mikolasek P. Hansen-Street nailing of fractures of
the femoral shaft: a study of 70 consecutive cases. Injury.
1983; 440-446.
24. Talacchi RL, Manning J, Lampsad S, Bach A, Carrico CJ. Early
intramedullary nailing of femoral shaft fractures: a cause of fat
26. Scully RE. Fat embolism in Korean battle casualties: its inci-
1956; 32:379.
27. Haig JS, Burghele LW, Okazaki H. The clinical and patholog-
28. Baker PL, Pezino JA, Peltier LF. Fat embolism, cavitary abdomi-
nal lesions, and renal involvement in fat embolism. Surg Gynec
29. Richards GR, Corey TS, Davis GJ. Non-associate fat emboli-
35:493-499.
30. Schwartz SI, Shires GT, Spencer FC. eds. Principles of
31. Lynch WC, Peltier LF. Seizures following head trauma in
association with long-term hyperalimentation. Hum Pathol.
32. Haber LM, Hawkins EP, Sehleimer DK, Saleem A. Fat over-
load syndromes: an autopsy study with evaluation of the coagula-
33. Estes JP, Millard Y. Fat embolism after liquid emulsion
34. Mudgez, Robison MJ, Duckworth W. Neonatal fat
embolism and agglutination of intralipid. Arch Dis Child. 1984;
59:1098-1109.
35. Young AK, Evans IL, Irving D, Hannin CD. Fat embolism
36. Thomas ML, Tieghe JR. Death from fat embolism as a compo-
37. Lynch MJ. Nephrosis and fat embolism in acute hemorrhagic
38. Mahoney HE, Weiss JS. Carbon tetrachloride poisoning with
39. Petsore L, Kessler S. Pulmonary fat embolism in the immuno-
40. Hill RR. Fatal fat embolism from steroid-induced fatty liver.
41. Rosen JM, Braman SS, Hassam FM, Teplitz C. Nontraumatic
fat embolization: a care cause of new pulmonary infarcts in an
42. Durlacher SR, Meier JR, Fisher RS. Sudden death due to pul-
monary fat embolism in chronic alcoholics with fatty liver. J
43. Broder G, Ruztums L. Systemic fat embolism following
44. Shapiro MP, Hayes JA. Fat embolism in sickle cell disease:
report of a case with brief review of the literature. Arch Int Med.
1984; 144:181-182.
45. Kaufman AD, Finn R, Bourdillon RE. Fat embolism follow-
46. Todd N. Fatal fat embolism during ritual initiation. Can Med
 Assoc J. 1973; 113:133-137.
48. Benetar SR, Ferguson AP, Goldschmidt RB. Fat emboli: some
clinical observations and a review of controversial aspects. J
Med. 1972; 41:85.
50. Evans CM. The fat embolism syndrome: a review. Surg Clin
52. Scully RE. Fat embolism in Korean battle casualties: its inci-
1956; 32:379.
53. Dines DE, Burghele LW, Okazaki H. The clinical and patholog-
54. Baker PL, Pezino JA, Peltier LF. Fat emboli, cavitary abdomi-
nal lesions, and renal involvement in fat embolism. Surg Gynec
55. Richards GR, Corey TS, Davis GJ. Non-associate fat emboli-
35:493-499.
56. Schwartz SI, Shires GT, Spencer FC. eds. Principles of
57. Lynch WC, Peltier LF. Seizures following head trauma in
association with long-term hyperalimentation. Hum Pathol.
58. Haber LM, Hawkins EP, Sehleimer DK, Saleem A. Fat over-
load syndromes: an autopsy study with evaluation of the coagula-
59. Estes JP, Millard Y. Fat embolism after liquid emulsion
60. Mudgez, Robison MJ, Duckworth W. Neonatal fat
embolism and agglutination of intralipid. Arch Dis Child. 1984;
59:1098-1099.
61. Young AK, Evans IL, Irving D, Hannin CD. Fat embolism
62. Thomas ML, Tieghe JR. Death from fat embolism as a compo-
63. Lynch MJ. Nephrosis and fat embolism in acute hemorrhagic
64. Mahoney HE, Weiss JS. Carbon tetrachloride poisoning with
65. Petsore L, Kessler S. Pulmonary fat embolism in the immuno-
66. Hill RR. Fatal fat embolism from steroid-induced fatty liver.
67. Rosen JM, Braman SS, Hassam FM, Teplitz C. Nontraumatic
fat embolization: a care cause of new pulmonary infarcts in an
68. Durlacher SR, Meier JR, Fisher RS. Sudden death due to pul-
monary fat embolism in chronic alcoholics with fatty liver. J
69. Broder G, Ruztums L. Systemic fat embolism following
70. Shapiro MP, Hayes JA. Fat embolism in sickle cell disease:
report of a case with brief review of the literature. Arch Int Med.
1984; 144:181-182.
71. Kaufman AD, Finn R, Bourdillon RE. Fat embolism follow-
72. Todd N. Fatal fat embolism during ritual initiation. Can Med
Assoc J. 1973; 113:133-137.
74. Benetar SR, Ferguson AP, Goldschmidt RB. Fat emboli: some
clinical observations and a review of controversial aspects. J
Med. 1972; 41:85.
90. Evarts CM, Diagnosis and treatment of fat embolism. JAMA. 1965; 194:899-901.

EDITORIAL DISCUSSION

ORTHOPEDICS: Some European literature states that early fixation of long-bone fractures in a patient with compromised pulmonary status may worsen the pulmonary function. What is the authors' opinion on the matter?

John and Lucas: It has been well-documented that immediate open reduction internal fixation (ORIF) of long-bone fractures, particularly of the femur, within 24 to 48 hours, decreases the development of fat embolism syndrome (FES) as compared with conservative nonoperative treatment such as traction or casting. This has been well documented in Scandinavian, European, and American literature. This aggressive trauma management appears to be accepted almost universally as the standard care of long-bone fractures.

In response to your question concerning a "patient with compromised pulmonary status," it is still our opinion that early ORIF is indicated. We believe that this may help in a number of ways. First, if the compromised pulmonary status is from a non-fat embolism cause, such as
pulmonary contusion or pneumothorax, preventing the additional insult of FES is vital to preventing additional pulmonary compromise.

Second, by ORIF of lower extremity long-bone fractures, the patient may be mobile sooner and begin earlier ambulation. This will aid in pulmonary cleansing needed to prevent or aid in treatment of various pulmonary insults including both FES and non-FES origins.

Third, if adult respiratory distress syndrome (ARDS) should develop in the trauma patient, causing compromised pulmonary status, aggressive medical management should be instituted. The tenants of ARDS management are oxygen supplementation, fluid management, and the removal of the underlying cause of the problem. If ARDS should develop in the trauma patient with long-bone fractures, the FES is a possible cause, and therefore, ORIF is essential to maximize other medical management of this syndrome. If the underlying cause persists in ARDS, it may progress to an irreversible state.12

Because of the these reasons, it is our belief that early ORIF of long-bone fractures in the trauma patient is necessary both to prevent the development of FES and to aid in the treatment of pulmonary compromise in both the FES and non-FES patient.

REFERENCES

(EDITORIAL DISCUSSION)