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Fat embolism and postoperative coagulopathy

To the Editor:

I was interested to read the article¹ and accompanying editorial² regarding fat embolism and postoperative coagulopathy.

Byrick points out that "even after cardiac arrest and resuscitation, such an impressive coagulopathy, leading to shock and death is exceedingly rare".

I recently reported a case of the fat embolism syndrome³ in an 18-yr-old girl who had sustained a minimally displaced oblique fracture through the mid-shaft of her left tibia and fibula. In this case only very gentle, closed manipulation (performed within 24 hr of the accident) was required to bring the bones to a suitable alignment. Nonetheless, she developed disseminated intravascular coagulation, multi-organ dysfunction syndrome, and refractory hypoxemia, dying approximately six days and a half after the initial injury.

I agree with Byrick's comment that 'intravascular fat may only be a marker for the intravasation of marrow products during cemented arthroplasty': this then begs the question why did such a common event⁴ have such devastating consequences in these two particular patients? Does it have anything to do with an abnormal and overwhelming inflammatory response? If so, who (and why) are certain people liable to such reactions?

In the case I described the only medical history of note was that of resolved but, at the time, debilitating myalgic encephalomyelitis. Was this another manifestation of an abnormally reactive immune/inflammatory system? If the immune system is involved how can

we identify such patients, and such responses?
As always "more research is needed".

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Thoracoabdominal retroperitoneal lymph node dissection (RPLND) and hemodynamic instability in the postoperative period.

To the Editor:

Thoracoabdominal retroperitoneal lymph node dissection is the most common and accepted method of treatment for patients with testicular cancer. This procedure involves extensive retroperitoneal dissection performed via a thoracoabdominal incision. All the lymph nodes, metastatic masses and lymphatic tissues are removed. In approximately 10% of cases the aorta and vena cava are removed and replaced with grafts. Coincidental removal of sympathetic ganglia and fibres during dissection is inevitable and may result in signs and symptoms of autonomic dysfunction in these patients, such as postural hypotension and tachycardia.^{1,2} The signs and symptoms of autonomic dysfunction depend on the extent of the dissection, and such an extensive dissection is performed in 20-25% of the patients.

At the Norris Cancer Hospital we have done over 400 such cases and also managed them postoperatively in the intensive care unit.³ Our observation has been that the degree of hemodynamic instability (i.e., hypotension, tachycardia) directly correlates with the extent of the dissection. One-sided dissection results in ipsilateral signs of sympathectomy such as venous