REFERENCES


2 Wilcox PA, Potgieter PD, Bateman ED, Benatar SR. Rapid diagnosis of sputum negative miliary tuberculosis using flexible fiberoptic bronchoscope. Thorax 1986; 41:681-84


Fat Emulsion and ARDS

To the Editor:

In the June, 1989 issue of the CHEST (95:1278-81), Venus et al reported the effects on hemodynamics and gas exchange after administration of fat emulsion infused intravenously (3.0 ± 0.3 mg/kg/min) in 19 patients with ARDS. The authors described a significant reduction in PaO2/FiO2, and an increase in MPAP (mean pulmonary artery pressure), PVR (pulmonary vascular resistance) and Qva/Qt (pulmonary venous admixture). Furthermore, they found that Qva/Qt increased to a greater extent in septic vs non-septic ARDS patients, while the magnitude of increased MPAP was not influenced by the presence or absence of septicemia.

We would like to report our findings obtained after intravenous infusion of 20 percent Intralipid emulsion in six patients suffering from ARDS (Table 1) in which, in contrast to the Venus et al report, we did not observe any significant change in hemodynamic and gas exchange measurements.

We maintain that the discrepancies between our results and that of Venus et al are due to different protocols involved in the two studies. 1) We administered the same amount of 20 percent Intralipid (500 ml) twice as fast (4 hours instead of 6). 2) Our patients had less severe ARDS (mean value of Qva/Qt was 13.9 kg vs 20.6 percent). 3) We measured hemodynamics and gas exchange in our patients more often than Venus et al did. We believe that intervals of 8 and 3 to 4 h between measurements (immediately prior to and following completion of Intralipid infusion, respectively, in the report) are too long for such critically ill and unstable patients.

In brief, it is likely that the conflicting results could be attributed mainly to the infusion rate. Skie et al have suggested that during slow lipid infusion (eg, 3 mg/kg/min for eight hours, similar to Venus et al’s report) there may be a net increase in vasodilatory and anti-inflammatory prostaglandins (resulting in a release of HPV and hence an increase of Qva/Qt). In addition, after the administration of a bolus or rapid infusion of lipids (eg, 8 to 10 mg/kg/min), fatty acid substrate may overwhelm the effect of vasodilatory prostaglandin (PGF2α and PGL1) production, resulting in increased production of vasopressor and inflammatory prostaglandin metabolites (eg, thromboxane A2).

An intermediate fat emulsion infusion rate, like in our experiments (500 ml of 20 percent Intralipid in 4 h, about 6.0 mg/kg/min), may result in an equilibrium between vasodilating and vasoconstricting prostaglandin production without an effect on hemodynamics or gas exchange in patients with ARDS. Hence our findings might suggest that fat emulsion administration to critically ill patients should be done relatively fast, if we wish to prevent effects on gas exchange.

Kausal Patel, M.D.
Vallabhbhai Patel Chest Institute
Delhi, India

REFERENCES


3 Wilcox PA, Potgieter PD, Bateman ED, Benatar SR. Rapid diagnosis of sputum negative miliary tuberculosis using flexible fiberoptic bronchoscope. Thorax 1986; 41:681-84


S. Zakynthinos, M.D.;
G. Baltopoulos, M.D., and
Ch. Roussos, M.D.,
Department of Critical Care Medicine,
University of Athens Medical School,
Athens, Greece