Cardiopulmonary Effects of Lipid Emulsions in Patients With ARDS*

Marion Faucher, MD; Fabienne Bregeon, MD; Marc Gainnier, MD; Xavier Thirion, MD; Jean-Pierre Auffray, MD; and Laurent Papazian, MD

Study objectives: Lipid emulsions have been suspected of inducing certain modifications in gas exchange and pulmonary hemodynamics. The aim of this prospective study was to evaluate the hemodynamic and pulmonary effects of two lipid emulsions.

Design: Prospective, randomized, double-blind, crossover study.

Setting: Medical and surgical ICU in a French university hospital.

Patients: Eighteen patients presenting ARDS.

Interventions: Each patient received a 6-h infusion of a 20% fat emulsion containing 100% long-chain triglycerides (LCTs) and a 6-h infusion of 50% LCTs/50% medium-chain triglycerides (MCTs) 20% lipid emulsion at the rate of 1.0 mL/kg/h. An 18-h period with no lipids separated the two periods. An additional 18-h period after the end of the second lipid emulsion administration was observed prior to the final measurements.

Measurements and results: The MCT/LCT emulsion increased the PaO2/fraction of inspired oxygen (FIO2) ratio (p < 0.005) compared with LCT emulsion alone. The mean (± SD) PaO2/FIO2 ratio increased from 165 ± 55 to 191 ± 64 mm Hg after 1 h of LCT/MCT administration (p < 0.03), and to 175 ± 46 mm Hg after 6 h. Moreover, there was an increase in oxygen delivery after 6 h of LCT/MCT administration (p < 0.001 vs baseline). While a time-related increase in mean pulmonary artery pressure (p = 0.012) during lipid administration was found, no effect of the kind of lipid emulsion was observed. The time-related increase in cardiac index (p = 0.002) was more marked when the patients received the LCT/MCT emulsion (p = 0.002). Pulmonary vascular resistances were not affected by the kind of lipid emulsion.

Conclusions: The present work showed that while the LCT emulsion induced no deleterious effects on oxygenation in ARDS patients, the LCT/MCT emulsion improved the PaO2/FIO2 ratio and had a further beneficial effect on oxygen delivery.

Key words: ARDS; emulsion; hemodynamic; lipid; oxygenation

Abbreviations: ANOVA = analysis of variance; CI = cardiac index; DO2I = oxygen delivery index; FIO2 = fraction of inspired oxygen; LCT = long-chain triglyceride; MCT = medium-chain triglyceride; MPAP = mean pulmonary arterial pressure; PAO2 = pulmonary artery occluded pressure; PEEP = positive end-expiratory pressure; PVRI = pulmonary vascular resistances indexed; QVA/QT = venous admixture

Although lipid emulsions are routinely used as a caloric source for critically ill patients, these emulsions have been suspected of inducing certain modifications in gas exchange and pulmonary hemodynamics. However, there are conflicting data in the literature about the effects of lipid emulsions on pulmonary diffusion capacity and arterial oxygen tension (Table 1).1–10 The composition in fatty acids of the available lipid emulsions is considered to be the main factor responsible for the cardiopulmonary effects of such lipids. Long-chain polyunsaturated fatty acids are precursors of substances such as prostaglandins, leukotrienes, and thromboxanes. These substances are able to modify the ventilation/perfusion ratio, causing hypoxemia and the modification of pulmonary arterial pressure. Currently available commercial lipid emulsions are derived

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from soybeans and/or safflower oils, and they contain long-chain ω-6 and ω-3 polyunsaturated fatty acids that have been esterified with glycerol to form long-chain triglycerides (LCTs). A new lipid emulsion was characterized by the partial replacement of LCT by medium-chain triglycerides (MCTs). MCTs are oxidized faster than LCTs without acting as precursors of prostanoids. Therefore, while MCTs do not interfere with eicosanoid synthesis, it has been hypothesized that the use of LCT/MCT emulsions could be associated with fewer pulmonary effects.

Nutritional support could influence the prognosis of patients presenting with ARDS. Moreover, the impairment in oxygenation and the modifications of pulmonary hemodynamics found during ARDS could be altered by agents that are supposed to interfere with inflammatory mediators. Therefore, the effects of lipids on gas exchange were studied in patients presenting with acute respiratory failure. However, the results have been contradictory. The aim of this prospective study therefore was to evaluate the hemodynamic and pulmonary effects of two lipid emulsions (ie, one LCT emulsion, and one LCT/MCT emulsion) in patients presenting with ARDS.

### Materials and Methods

#### Study Population

During a 12-month period, we performed a prospective, randomized, double-blind, crossover study that included 18 patients (15 men and 3 women; mean [± SD] age, 52 ± 14 years; simplified acute physiology II score on hospital admission, 36 ± 10) who had been admitted to the medical and surgical ICUs of Sainte-Marguerite University Hospital in Marseille, France. These patients were prospectively investigated 3 ± 2 days after the onset of ARDS. They were included after written informed consent was obtained from each patient’s next of kin. When the study was started, no patient had evidence of dyslipidemia, diabetes mellitus, or renal, cardiovascular, or hepatic dysfunction. No anti-inflammatory drugs were administered during the week prior to the beginning of the study or during the study. The study was approved by our ethics committee (Comité Consultatif de Protection des Personnes dans la Recherche Biomédicale de Marseille) and was supported by l’Association Régionale d’Assistance Respiratoire à Domicile. ARDS was defined according to the recommendations of the American-European Consensus Conference. Among the 18 patients enrolled in the study, 9 had been admitted to the hospital for an acute medical illness, 4 had been admitted for postoperative complications following major surgery, and 5 had been admitted to the ICU after experiencing multiple trauma. ARDS was related to pulmonary causes in 72% of the patients (infectious pneumonia, six patients; lung contusion, one patient; aspiration pneumonia, six patients), and to extrapulmonary causes in 28% of the patients (extrapulmonary sepsis, four patients; acute pancreatitis, one patient).

#### Instrumentation and Measurements

**Blood Gas Analyses:** Systemic and pulmonary arterial blood samples were simultaneously withdrawn within 3 min before the measurement of cardiac output. Arterial pH, P<sub>O2</sub>, mixed venous partial pressure of oxygen, and P<sub>CO2</sub> were measured using a blood gas analyzer (model 278-blood gas system; Ciba Corning; Medfield, MA). Hemoglobin concentration, arterial oxygen saturation, and mixed venous oxygen saturation were measured using a calibrated hemoximeter (model 270-CO-oxymeter; Ciba Corning).

**Determination of Blood Concentration of Lipids:** In order to verify that baseline conditions were identical before each lipid emulsion infusion, blood samples were drawn before the beginning of each lipid emulsion infusion period and 18 h after the end

### Table 1—Lipid Emulsions in ICU Patients

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Lipid</th>
<th>Patients, No.</th>
<th>Change in Oxygenation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rademacher et al¹</td>
<td>Noncomparative</td>
<td>LCT/MCT, 0.15 g/kg/h for 4 h</td>
<td>Sepsis, 9</td>
<td>No</td>
</tr>
<tr>
<td>Masclans et al²</td>
<td>Comparative</td>
<td>LCT, 2 mg/kg/min for 12 h</td>
<td>ARDS, 7</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCT/MCT, 2 mg/kg/min for 12 h</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smirniotis et al³</td>
<td>Comparative</td>
<td>LCT, 12 g/h for 8 h</td>
<td>Surgical septic ARDS, 10</td>
<td>Decrease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCT/MCT, 12 g/h for 8 h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hwang et al⁴</td>
<td>Comparative</td>
<td>LCT, 6 mg/kg/min for 4 h</td>
<td>ARDS, 5</td>
<td>Decrease</td>
</tr>
<tr>
<td>Fiaccadori et al⁵</td>
<td>Noncomparative</td>
<td>LCT/MCT, 3.3 mg/kg/min for 2 h</td>
<td>Open-heart surgery, 12</td>
<td>No</td>
</tr>
<tr>
<td>Mathru et al⁶</td>
<td>Comparative</td>
<td>LCT, 5 g/h for 10 h</td>
<td>Septic ARDS, 8</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCT, 10 g/h for 5 h</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Venus et al⁷</td>
<td>Noncomparative</td>
<td>LCT 5 g/h for 10 h</td>
<td>ICU, 20</td>
<td>Decrease</td>
</tr>
<tr>
<td>Chassard et al⁸</td>
<td>Comparative</td>
<td>LCT, 3 mg/kg/min for 8 h</td>
<td>Sepsis, 6</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCT/MCT, 3 mg/kg/min for 8 h</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Ball and White⁹</td>
<td>Noncomparative</td>
<td>LCT/MCT, 3.5 mg/kg/min for 3.5 h</td>
<td>Trauma or surgery, 17</td>
<td>No</td>
</tr>
<tr>
<td>Venus et al¹⁰</td>
<td>Noncomparative</td>
<td>LCT, 3 mg/kg/min for 8 h</td>
<td>ARDS, 19</td>
<td>Decrease</td>
</tr>
</tbody>
</table>

*Note: LCT = long-chain triglycerides; MCT = medium-chain triglycerides.*
of the second emulsion infusion period. They were analyzed for triglycerides, nonesterified fatty acids, and cholesterol (measured by enzymatic methods).

Hemodynamic Parameters: All of the patients had a radial artery catheter (Seldicath; Plastimed; Saint Leu la Forêt, France) and a pulmonary artery catheter (model 93 A-434H-7.5F; Baxter Healthcare Corporation; Irvine, CA), which were inserted percutaneously through the right jugular or the left axillary vein and were positioned so that the distal port was in the pulmonary artery and the proximal port was in the right atrium, just above the tricuspid valve.

Systolic arterial pressure, diastolic arterial pressure, systolic pulmonary arterial pressure, diastolic pulmonary arterial pressure, pulmonary artery occluded pressure (PAOP), and right atrial pressure were measured at end-expiration. Cardiac index (CI), oxygen delivery index (DO₂I), oxygen consumption index, and venous admixture (QVA/QT) were calculated using standard formulas. Pulmonary vascular resistances indexed (PVRI) were calculated using the following standard formula: PVRI = (mean pulmonary arterial pressure [MPAP] − PAOP) × 79.9/CI.

Procedure
The study was performed after the optimization of the treatment of hypoxemia in order to obtain at least 12 h of stability of FIO₂/fraction of inspired oxygen (FIO₂) without any change in ventilator settings (fluctuation, < 20%). Selection of the appropriate level of positive end-expiratory pressure (PEEP) was performed by increasing PEEP in steps of 2 cm H₂O. A blood gas analysis was performed when pulse oximetric saturation was stable during a 30-min period after PEEP level adjustment. Finally, the lower level of PEEP giving the greater improvement in oxygenation was chosen. When no improvement was found while increasing PEEP, the level was set at 8 cm H₂O. Nitric oxide and almitrine bismesylate were used when the PaO₂/FIO₂ ratio remained at < 150 mm Hg despite PEEP adjustment. For the purpose of the study, prone position was not permitted until completion of the protocol. For each patient, tidal volume and PEEP levels were kept constant throughout the study period.

No patient received lipid emulsion prior to inclusion in the study. On days 1 and 2, each patient received, in random order, a 6-h infusion of a 20% fat emulsion containing LCTs (Endolipide [20%]; B. Braun Medical; Boulogne, France) or an infusion of 50% LCT/50% MCTs (Medialipide [20%]; B. Braun Medical) at the rate of 1.0 mL/kg/h. An 18-h period without lipid administration separated the two periods. An additional 18-h period after the end of the second lipid emulsion administration was observed prior to the final measurements (control 2) [Fig 1]. Both lipid emulsions contained LCTs derived from soybean oil. The LCT emulsion contained 200 g soybean oil per liter, while the LCT/MCT emulsions contained 100 g soybean oil. Soybean oil provides 54% of linoleic acid and 8% of α-linoleic acid. The LCT/MCT emulsion also contained 100 g MCTs (capric acid, 54%; caprylic acid, 40%; lauric acid, 4%; and caproic acid, 2%). One liter of the 20% LCT emulsion and 1 L LCT/MCT emulsion contained 25 g glycerol and 12 g lecithin. The study was performed in patients whose only caloric source was a 5% glucose infusion (infusion rate, 1.5 mg/kg/min).

Measurements were obtained before starting the first lipid emulsion infusion, after 1 h of administration, at the end of the 6-h period of administration, and, finally, 18 h after the end of the administration of each lipid emulsion.

Statistical Analysis
The data were expressed as the mean ± SD. Statistical calculations were performed using a statistical software package (SPSS, version 8.0; SPSS Inc; Chicago, IL). Statistically significant differences were analyzed by parametric or nonparametric tests when required. The effects of the two lipid emulsions were compared by using a two-way, repeated-measures analysis of variance (ANOVA) [factors considered were time and type of lipid emulsion]. When appropriate, a post hoc analysis was performed using a pairwise multicomparison procedure (ie, Tukey test). A p value of < 0.05 indicated significance.

Results
Characteristics of the Study Population
The duration of mechanical ventilation preceding the study was 4.4 ± 2.8 days. All of the patients were sedated with a continuous infusion of sufentanil and midazolam, and lungs were ventilated using conventional volume-controlled mechanical ventilation (7200 series; Mallinckrodt Puritan Bennett; Carlsbad, CA). On the day of the study, the mean lung injury score was 2.7 ± 0.2. On inclusion in the study, the mean respiratory parameters were as follows: tidal volume, 7.1 ± 1.6 mL/kg; FIO₂, 0.63 ± 0.11; PEEP, 10.0 ± 2.5 cm H₂O. Only four patients received inhaled nitric oxide and/or a continuous infusion of almitrine bismesylate during the study period. There was no modification in core temperature between the two groups (data not shown).

Effects of Lipid Administration on Oxygenation
The LCT emulsion had no effect on oxygenation, whereas the MCT/LCT emulsion improved the PaO₂/FIO₂ ratio (p = 0.04 [by one-way, repeated-measures ANOVA]). When the two lipid emulsions were compared, two-way, repeated-measures ANOVA showed that the MCT/LCT emulsion improved the PaO₂/FIO₂ ratio (p = 0.005) compared with the LCT emulsion (Fig 2). The order of lipid emulsions did

![Figure 1. Study design.](image-url)
not influence their effects (ANOVA, not significant), and the baseline value after the first period was equivalent to the initial baseline value for each patient group (ANOVA, not significant). A pairwise multicomparison procedure (ie, Tukey test) showed that the 16% increase in the \( \text{Pa}_2/\text{Fi}_2 \) ratio 1 h after the beginning of the MCT/LCT emulsion infusion was significant \( (p < 0.03) \), while the \( \text{Pa}_2/\text{Fi}_2 \) ratio at the end of the 6-h infusion was not different from the baseline value (Fig 2 and Table 2). Using ANOVA, we verified that inhaled nitric oxide and almitrine bismesylate did not influence the effects of these lipid emulsions.

**Effects of Lipid Administration on Gas Exchange and Respiratory Parameters**

No modification in \( \text{PaCO}_2 \) was observed during the study period. We did not observe any modification in respiratory parameters related to lipid administration.

**Effects of Lipid Administration on Hemodynamics**

We did not observe a significant effect of the kind of lipid emulsion on MPAP (Table 2). However, a time-related increase in MPAP during the administration of lipid emulsion \( (p < 0.012 \text{ by ANOVA}) \)

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**Table 2—Respiratory and Hemodynamic Parameters**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control 1</th>
<th>LCT Emulsion Before</th>
<th>LCT Emulsion 1 h</th>
<th>LCT Emulsion 6 h</th>
<th>LCT/MCT Emulsion Before</th>
<th>LCT/MCT Emulsion 1 h</th>
<th>LCT/MCT Emulsion 6 h</th>
<th>Control 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{PaO}_2/\text{Fi}_2 ), mm Hg</td>
<td>164 ± 45</td>
<td>156 ± 44</td>
<td>161 ± 43</td>
<td>156 ± 42</td>
<td>165 ± 55</td>
<td>191 ± 64</td>
<td>175 ± 46</td>
<td>177 ± 65</td>
</tr>
<tr>
<td>( \text{PaCO}_2 ), mm Hg</td>
<td>44 ± 8</td>
<td>45 ± 9</td>
<td>44 ± 8</td>
<td>45 ± 9</td>
<td>43 ± 8</td>
<td>43 ± 7</td>
<td>44 ± 7</td>
<td>43 ± 7</td>
</tr>
<tr>
<td>MPAP, mm Hg</td>
<td>24 ± 6</td>
<td>24 ± 7</td>
<td>25 ± 7</td>
<td>26 ± 7</td>
<td>23 ± 6</td>
<td>24 ± 7</td>
<td>25 ± 6</td>
<td>25 ± 5</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>87 ± 14</td>
<td>90 ± 11</td>
<td>86 ± 11</td>
<td>95 ± 17</td>
<td>88 ± 14</td>
<td>86 ± 12</td>
<td>99 ± 19†</td>
<td>90 ± 15</td>
</tr>
<tr>
<td>CI, L/min( \cdot )</td>
<td>4.6 ± 1.3</td>
<td>4.8 ± 1.3</td>
<td>4.4 ± 1.2</td>
<td>4.9 ± 1.5</td>
<td>4.3 ± 1.0</td>
<td>4.4 ± 1.0</td>
<td>5.4 ± 1.6†</td>
<td>4.7 ± 0.9</td>
</tr>
<tr>
<td>PVRI, dyne( \cdot )cm( ^{-5} )( \cdot )m( ^2 )</td>
<td>236 ± 128</td>
<td>225 ± 123</td>
<td>263 ± 127</td>
<td>251 ± 117</td>
<td>231 ± 99</td>
<td>244 ± 109</td>
<td>206 ± 105</td>
<td>213 ± 68</td>
</tr>
<tr>
<td>QVA/QTE, %</td>
<td>37 ± 8</td>
<td>40 ± 8</td>
<td>37 ± 8</td>
<td>37 ± 9</td>
<td>37 ± 10</td>
<td>35 ± 8</td>
<td>36 ± 11</td>
<td>33 ± 6</td>
</tr>
<tr>
<td>( \text{DO}_{2i} ), mL/min( \cdot )m( ^2 )</td>
<td>564 ± 145</td>
<td>597 ± 161</td>
<td>547 ± 153</td>
<td>608 ± 197</td>
<td>532 ± 131</td>
<td>551 ± 133</td>
<td>672 ± 181†</td>
<td>573 ± 116</td>
</tr>
<tr>
<td>( \text{VO}_{2i} ), mL/min( \cdot )m( ^2 )</td>
<td>119 ± 42</td>
<td>114 ± 38</td>
<td>116 ± 33</td>
<td>133 ± 37</td>
<td>116 ± 40</td>
<td>123 ± 34</td>
<td>136 ± 53</td>
<td>142 ± 39</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. HR = heart rate; \( \text{VO}_{2i} \) = oxygen consumption index.*

\( p < 0.03 \) vs before LCT/MCT administration.

\( p < 0.02 \) vs before and 1 h after LCT/MCT administration.

\( p < 0.001 \) vs before and 1 h after LCT/MCT administration.

\( p < 0.01 \) vs 6 h after LCT administration.
was observed as well as a time-related increase in heart rate and CI (p = 0.002 by ANOVA). An interaction effect on CI also was noted between the kind of lipid emulsion and the duration of its administration (p = 0.002 [by ANOVA]), indicating that the increase in cardiac output during lipid infusion was more marked when the patients received the LCT/MCT emulsion. For example, a 27% increase in CI was noted at the sixth hour of administration of the LCT/MCT emulsion (p = 0.001 vs before and at 1 h) [Fig 3], while no significant change in CI was related to LCT emulsion administration (Fig 3). A time-related increase in oxygen delivery (p = 0.003 [by ANOVA]) and in oxygen consumption (p = 0.029 [by ANOVA]) was observed during lipid emulsion administration. An interaction effect on oxygen delivery also was found between the duration of lipid administration and the kind of lipid emulsion (p = 0.002 [by ANOVA]) [Table 2]. The increase in oxygen delivery related to LCT/MCT emulsion, which reached 4% after 1 h of infusion and 26% after 6 h of infusion, illustrates this action (Fig 4). Pulmonary vascular resistances were not affected by the kind of lipid emulsion (p = 0.067). No statistically significant alterations in mean arterial pressure, right atrial pressure, or PAOP were noted during the entire period of investigation. No significant change in the rate of fluid administration was observed for a given patient throughout the study period.

Effect of Lipid Emulsion Administration on Plasma Levels of Triglycerides, Cholesterol, and Nonesterified Fatty Acids

As shown in Table 3, triglycerides, cholesterol, and nonesterified fatty acid levels were not different before and 18 h after the administration of both lipid emulsions.

Discussion

The results of the present study suggested that neither lipid emulsion (ie, LCT or LCT/MCT lipid emulsion) decreased PaO₂. Moreover, no deleterious effects on pulmonary hemodynamics were noted. In the literature, lipid emulsions have been associated with changes in oxygenation and pulmonary hemodynamics. Increased oxygen consumption and increased CO₂ production have been reported after MCT administration.\(^{12,13}\)

A review of the literature to date showed no clear evidence that major clinical changes in oxygenation occurred with lipid administration in ARDS patients. Discrepancies between studies may be a consequence of alterations in lipid metabolite concentrations due to differences either in metabolic clearance or lipid administration rates and durations.\(^{5,7,14,15}\) For example, Hunt et al\(^{16}\) reported that slow infusion rates of lipid emulsion resulted in a relative predominance of vasodilating prostaglandins, while rapid
infusion induced the predominance of vasoconstricting prostaglandins. It has been hypothesized that rapid infusion could increase the production of vasoconstrictive eicosanoids.\textsuperscript{17–19} In the present work, we found a time-related increase in MPAP only during lipid administration, with no significant difference between the two kinds of lipid emulsions. This increase in MPAP could be related to the concomitantly observed increase in CI.

Preexisting lung status with the degree of lung injury also could explain certain discrepancies between studies.\textsuperscript{4,6} Indeed, it was demonstrated in an animal study\textsuperscript{19} that while lipid emulsion administration has a negligible effect on healthy lungs, it induced hypoxemia in animals with injured lungs. In a rabbit model,\textsuperscript{15} there were no significant blood gas or prostaglandins changes in the saline solution control groups or in the normal lung groups that were infused with an LCT emulsion. However, in the lung-damaged groups (\textit{i.e.}, by oleic acid), there was a small but significant decrease in PaO\textsubscript{2}, which reached 12 mm Hg.\textsuperscript{15} There was also a significant increase in arterial vasodilating prostaglandin levels.\textsuperscript{15} A vasodilatory response to prostaglandins precursors was noted in the presence of an increased pulmonary vascular tone.\textsuperscript{10} Finally, controversy remains as to whether and which prostaglandins are involved in these sometimes-observed manifestations.

The mechanisms by which fat emulsions could influence oxygenation and pulmonary hemodynamics are poorly understood. The prevention of the decrease in PaO\textsubscript{2} by indomethacin has suggested that these changes are at least in part mediated by prostaglandins.\textsuperscript{19,20} Nevertheless, one can argue that the beginning of a rapid lipid emulsion infusion (as during the first hour in the present work) could provoke an increase in vasoconstrictive products. These substances could enhance selective regional hypoxic pulmonary vasoconstriction in lung areas with low ventilation/perfusion ratio, resulting in a decrease in blood flow in such areas. However, Planas et al\textsuperscript{21} have shown that the administration of

![Figure 4. Evolution of DO\textsubscript{2}I before, 1 h after, and 6 h after the beginning of the administration of both lipid emulsions. Values are given as the mean ± SD. * = p < 0.001 vs before and 1 h after the beginning of LCT/MCT emulsion, and p < 0.01 vs 6 h after the beginning of LCT administration.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21996/)

| Table 3—Plasma Levels of Triglycerides, Cholesterol, and Nonesterified Fatty Acids* |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Variables       | Control 1       | LCT/MCT Emulsion Before Infusion | 18 h Postinfusion | LCT/MCT Emulsion Before Infusion | 18 h Postinfusion | Control 2       |
| Triglycerides, mmol/L | 1.7 ± 1.2 | 1.6 ± 1.2 | 1.5 ± 0.8 | 1.5 ± 1.3 | 1.8 ± 1.3 | 1.6 ± 0.9 |
| Cholesterol, mmol/L    | 2.7 ± 0.8 | 2.7 ± 0.8 | 2.7 ± 1.1 | 2.7 ± 1.1 | 2.7 ± 1.0 | 2.7 ± 1.1 |
| Nonesterified fatty acids, mmol/L | 0.5 ± 0.3 | 0.4 ± 0.2 | 0.3 ± 0.1 | 0.4 ± 0.3 | 0.3 ± 0.2 | 0.2 ± 0.1 |

*Values given as mean ± SD.
lipid emulsion (LCT or LCT/MCT emulsion) in ARDS patients did not alter the plasma levels of prostanoids compared with those who did not receive infusion of a lipid emulsion. Smirniotis et al found that LCT infusion induced an elevation of MPAP and QVA/QT while the $\text{PaO}_2/\text{FIO}_2$ ratio declined, whereas LCT/MCT emulsion infusion induced no significant changes either in oxygenation or in pulmonary hemodynamics. However, when changes in oxygenation and pulmonary hemodynamics have been reported, they have generally not been clinically significant. As in the present work, Masclans et al found that LCT administration did not modify oxygenation. Furthermore, they also reported increased cardiac output and increased oxygen delivery to peripheral tissues. Radermacher et al found that pulmonary hemodynamics and gas exchange were not modified by LCT/MCT administration. Moreover, no modification of low ventilation/perfusion ratio in the lung regions was observed after LCT/MCT administration.1 In 19 patients presenting with moderate respiratory failure (ie, $\text{PaO}_2/\text{FIO}_2$ ratio, 241 ± 50 mm Hg), Venus et al reported that LCT administration induced a 24% decrease in $\text{PaO}_2/\text{FIO}_2$ ratio that was associated with a significant increase in MPAP and QVA/QT. The same group also reported an increase in MPAP related to LCT administration in non-ARDS patients.7 However, patient baseline physiologic data are difficult to estimate in a great number of these studies.

In the present study, we chose to give the highest amount of fat emulsion recommended per day at the maximal infusion rate, as recommended by the manufacturer. Indeed, a dose-dependent effect of the fat infusion rate has been shown in patients presenting with septic respiratory failure.6

CONCLUSIONS

The present work showed that lipid emulsion infusion induced no deleterious effects on oxygenation and hemodynamics. However, it should be noted that the composition of LCT emulsions varies from formulation to formulation. Finally, the unexpected transient increase in the $\text{PaO}_2/\text{FIO}_2$ ratio 1 h after the beginning of the MCT/LCT lipid emulsion requires further investigations, for example, by measuring the plasma concentrations of the lipid components.

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