Use of Intralipid[®] in managing refractory hypotension following epidural blockade

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To the Editor,

Systemic hypotension, a common side effect of epidural anesthesia, is commonly treated with intravenous fluids and vasopressors.¹ Nevertheless, under some circumstances, such as Enhanced Recovery After Surgery (ERAS),² fluid restriction is advocated, potentially increasing both the risk and the severity of hypotension. We describe herein the use of intravenous Intralipid[®] to treat a case of refractory hypotension following epidural anesthesia.

After providing consent to publish his case, a 63-yr-old male (body mass index 30.5 kg·m⁻²) with a history of coronary artery disease, along with a previous myocardial infarction (MI) and coronary artery bypass surgery, presented for gastrointestinal surgery under an ERAS protocol. A follow-up transthoracic echocardiography (TTE) after his MI showed mildly impaired left ventricular systolic function with an ejection fraction of 45–50%. No preoperative TTE was performed for the current surgery. Low thoracic (T7/8) epidural anesthesia was chosen for pain management. Surgery was performed under general anesthesia with a pre-induction blood pressure (BP) of 130/75 mmHg. Within the first hour of surgery, lidocaine 70 mg with morphine 3 mg was delivered via the epidural catheter. Following this, the patient's BP fell gradually. To maintain an intraoperative systolic BP of ~100 mmHg, a phenylephrine infusion (1 µg·kg⁻¹·min⁻¹) was required with multiple supplemental intermittent boluses of ephedrine totalling

30 mg. In total, 1.7 L of crystalloid were administered over three hours surgical time. Prior to emergence, an additional bolus dose of epidural ropivacaine 25 mg was given.

Shortly after arrival in the recovery room, the patient's trachea was extubated, and he experienced hypotension with a systolic BP of 68–77 mmHg. He was given phenylephrine 300 µg and ephedrine 40 mg, and a phenylephrine infusion was initiated and increased gradually to 0.7 µg·kg⁻¹·min⁻¹. In the two and a half hours spent in the recovery room, the patient received 4.5 L of crystalloid and colloid (Volulyte 6%, Fresenius Kabi, Germany) fluids, but systolic BP remained <90 mmHg. An intravenous infusion of 20% Intralipid[®] 250 mL (Baxter International, Deerfield, IL, USA) was then administered over five minutes. His heart rate remained unchanged at 75 beats·min⁻¹; however, his systolic BP improved immediately (Figure) and returned steadily to baseline after approximately 20 min post-infusion. Upon commencement of the Intralipid infusion, the phenylephrine infusion was discontinued, and no further vasopressor boluses were required in the recovery room. An epidural infusion at 4 mL·hr⁻¹ was subsequently started, and the patient was discharged to the ward without further vasopressor support.

This is a novel report describing administration of Intralipid to treat refractory hypotension following epidural anesthesia. In this case, the patient experienced prolonged and refractory hypotension despite five hours of intensive management with fluids and vasopressors. Since extended use of vasopressors would have required admission to the intensive care unit, we attempted to wean the patient using a significant fluid load of conventional colloid and crystalloid. Nevertheless, this failed to improve the patient's BP, so we decided to use Intralipid as an alternative colloid.

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Figure Systolic and diastolic blood pressure values and mean arterial pressure (MAP) following infusion of 250 mL Intralipid

Intralipid, a fatty emulsion colloid consisting primarily of soybean oil, is a component of total parenteral nutrition. Intralipid administration is also a well-known rescue measure for cardiovascular collapse due to systemic toxicity from local anesthetics and other toxins.³ The mechanism underlying lipid rescue is still unknown, although it is commonly thought that lipophilic drugs are sequestered from tissue receptors and captured by an extended lipid compartment in the blood (the “lipid sink” theory). Since the local anesthetic dose used in this case was too low to cause any significant systemic toxicity, Intralipid administration may instead have effected cardiovascular improvement by other mechanisms,⁴ potentially overcoming epidural-associated vasodilation. Indeed, Intralipid has previously been shown to affect BP directly by increasing systemic vascular resistance and cardiac output.⁵

While our successful use of Intralipid to treat this patient is encouraging, more information on the cause of hypotension would have been obtained if a bedside TTE and 12-lead electrocardiogram were performed in recovery. Regardless, further clinical and basic research studies are needed to investigate this potential clinical application and the required dosage of Intralipid.

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Conflict of interest/other associations The authors declare no conflict of interest.

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