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Criteria for Adequacy of Reversal of Neuromuscular Blockade

To the Editor:—In his editorial in which concern regarding the problem of residual neuromuscular blockade is expressed, Miller[1] concludes by stating that we should seek more sensitive indices of adequacy of reversal of neuromuscular blockade than those used at present. He lists the current criteria that include a maximum inspiratory force of 25 cm H_2O.

I would like to point out that the values of vital capacity and maximum inspiratory and expiratory mouth pressures quoted in the editorial as "normal" and "slightly depressed" are actually very low.

Normal values for vital capacity are usually quoted according to height, age, and sex, not weight, and the ability to achieve a vital capacity of only 15 ml/kg would give great cause for concern. For example, a 30-yr-old man, 160 cm tall, should have a vital capacity of 4 l, decreasing to 3 l at the age of 70.

Normal ranges for maximum inspiratory and expiratory pressures are broad and depend on the source used.[2-4] Whatever the reference value used, the ability to achieve a maximum inspiratory or expiratory pressure of only 25 cm H_2O would alert a respiratory physician to the presence of severe global respiratory muscle weakness needing investigation and probably intervention. Typical values for maximum inspiratory pressure should be about 100 cm H_2O and for maximum expiratory pressure about 150 cm H_2O.[5]

On closer inspection of the reference quoted in the editorial,[6] the values of 15–20 mg/kg for vital capacity and inspiratory and expiratory forces of 20–25 cm H_2O were described as, "clinically acceptable minimum limits required for adequate respiratory function," Not normal or near-normal values. I would suggest that instead of seeking other more sensitive tests of residual neuromuscular blockade, the adoption of higher acceptable values of vital capacity and respiratory muscle strength may be more sensible.

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In Reply:—I am encouraged that the overall concern expressed in my editorial has attracted the attention and agreement of Dr. Derrington. She is quite correct in her assertion that the vital capacity values listed in that editorial are not "normal." They are values that are frequently used to ascertain whether a patient can safely sustain adequate ventilation after endotracheal extubation.

The point of the editorial was to emphasize the possibility that current tests are not sensitive enough to detect residual neuromuscular blockade. If this conclusion is correct, either currently used tests can be altered to become more sensitive (as suggested by Dr. Derrington) or new tests can be created. Dr. Derrington has correctly pointed out that higher acceptable values of vital capacity and respiratory muscle strength than those currently used would be one approach to increasing the sensitivity of tests. In that regard, we are in complete agreement.

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Intracranial Hypertension Following Graft Reperfusion

To the Editor:—We read with interest the case report by Conaty *et al.*[1] regarding the acute elevation of intracranial pressure (ICP) following the release of a pneumatic tourniquet. We share their concern over intraoperative management of patients with decreased intracranial compliance and wish to report a case of acute increase in ICP during orthotopic liver transplantation (OLT) immediately following reperfusion of the graft liver, suggesting the importance of intraoperative monitoring of ICP in these patients.

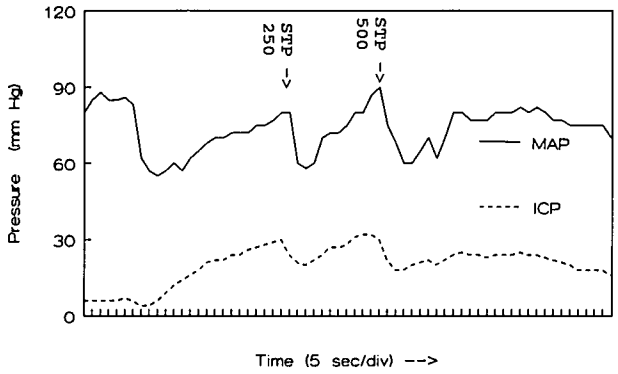

FIG. 1. ICP response to reperfusion and sodium thiopental (STP).

A 22-yr-old man was admitted to our institution in fulminant hepatic failure of unknown etiology. He rapidly developed stage 3–4 hepatic coma, and as ventricular size was small, a modified Richmond subarachnoid bolt was inserted for monitoring of ICP. An initial pressure of 36 mmHg was noted. The following day an orthotopic liver transplant was performed. During the preanhepatic and anhepatic stages of the procedure, the patient's ICP remained below 10 mmHg with only moderate hyperventilation (P_{CO_2} 27). Upon completion of the anastomoses of the graft liver, as the portal vein was unclamped, an immediate decrease in mean arterial pressure (MAP) occurred. As the MAP spontaneously returned to baseline, the patient's ICP began to increase. Manual hyperventilation was instituted with little effect. When the ICP reached 30 mmHg, thiopental was administered intravenously in two bolus doses of 250 mg and 500 mg, respectively (fig. 1). The patient's ICP transiently decreased to 20 mmHg, but rapidly returned to 33 mmHg. Following the second dose of thiopental, the patient's ICP remained below 24 mmHg and continued to slowly decline to 4 mmHg by the end of the procedure. No further treatment of the ICP was instituted other than continued moderate hyperventilation (P_{CO_2} 32 mmHg). No vasopressors were administered during the case. In the ICU the patient's ICP remained 10–13 mmHg and the bolt was removed on the second postoperative day. The trachea was extubated on that same day. No neurologic sequelae were present 1 week postoperatively.

The incidence of cerebral edema in patients with fulminant hepatic failure has been reported to be over 50% with evidence of herniation at the time of death in 12%.[2] Cerebral edema has been considered the most common immediate cause of death in fulminant hepatic failure.[3] Thus, treatment of cerebral edema and its resultant increased intracranial pressure is a critical element in managing cases of hepatic failure. Brajtbord *et al.* have reported a case of elevated intracranial pressure during OLT that was treated by incremental removal of CSF *via* a ventriculostomy.[4] The etiology of this increase was unclear, however, because vasopressors had been given to treat hypotension at the time of the increase in ICP. As our patient did not receive any form of vasopressors, the acute increase in ICP seemed to be the direct result of reperfusion of the graft liver and/or ischemic mesentery and lower extremity tissues. Data concerning a "safe" upper limit for ICP are sparse, but is has been suggested that a persistent level over 30 mmHg should be treated to reduce the risk of herniation.[5]

We have observed this same phenomenon in several, but not all, patients in fulminant hepatic failure undergoing hepatic transplantation. Our experience, along with the other reports cited, suggests that reperfusion of ischemic tissues may have an adverse effect on the intracranial pressure of a patient with reduced intracranial compliance. Early diagnosis and treatment of such may avoid a potentially devastating outcome.

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Pre-Eclamptic and Healthy Term Pregnant Patients Have Different Chronotropic Responses to Isoproterenol

To the Editor:—We have previously demonstrated that healthy term pregnant women have a blunted chronotropic response to isoproterenol compared to nonpregnant women.[1] If pre-eclamptic and healthy term pregnant patients differ in chronotropic responsiveness to isoproterenol, an isoproterenol epidural anesthesia test dose designed for healthy parturients might be unsafe or not efficacious in pre-eclamptic patients. We therefore determined the chronotropic responsiveness of pre-eclamptic patients to isoproterenol and compared these data with those previously obtained from healthy term pregnant and nonpregnant women.[1,2]

With IRB approval, we obtained written informed consent from five nonlaboring, nulliparous, term pregnant patients with mild pre-eclampsia. All patients had new onset proteinuria (≥ 2+ by urine dipstick on two occasions) and a recent (<2 week) diastolic blood pressure (BP) increase of ≥ 15 mmHg and/or systolic BP increase of ≥ 30 mmHg on two occasions at bed rest 6 h apart. Preinjection external