case with the patient in the sitting position. In Artru's case, an unknown amount of cerebrospinal fluid was removed to treat a rising ICP immediately following dural closure.<sup>2</sup> This created a possibility for fresh air entry into the superficial intracranial space, which then certainly could have expanded during N<sub>2</sub>O administration and elevated ICP. A portion of the intracranial gas compartment in his patient was clearly not equilibrated under conditions that prevailed in our experimental model, or in neurosurgical patients not subjected to acute CSF removal at the end of the surgical procedure.

Artru does not understand the experimental conditions established in group III animals. After promoting conditions leading to brain shrinkage and CSF loss, we slowly pumped air, under pressure, into the supracortical space in amounts sufficient to elevate the baseline ICP to 15 mmHg during N<sub>2</sub>O breathing. Absent the pump effect and in the presence of a CSF drainage pathway, the ICP would not increase at all and an isobaric pneumocephalus would occur. Removal of N<sub>2</sub>O under group II conditions decreased ICP and would reduce the volume of pneumocephalus that slowly occurred and was equilibrated, or at near equilibrium, with N<sub>2</sub>O when that anesthetic gas had already been administered for a period of time.

Thus, Skahen *et al.*'s study was prompted by Artru's misinterpretation of the factors contributing to the ICP elevation in his patient.<sup>2</sup>

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## Succinylcholine-induced Hyperkalemia in Near-drowning

To the Editor:—Succinylcholine has been widely reported to lead to hyperkalemia in a number of preexisting disease states, including multiple trauma, thermal injuries, neuromuscular disorders, ruptured cerebral aneurysms, and increased intracranial pressure.

Succinylcholine can also lead to hyperkalemia in near-drowning patients. A 14-kg 2-yr-old near-drowning patient sustained post-injury hypoxic-ischemic encephalopathy. Two weeks later, while receiving succinylcholine (1 mg/kg) during anesthesia for a feeding gastrostomy, she became rigid and converted from a sinus rhythm to ventricular tachycardia. Her serum potassium rose from a pre-succinylcholine administration level of 3.4 meq/l to 7.47 meq/l and fell to 3.1 meq/l after treatment for hyperkalemia and electrical cardioversion.

With the availability of other neuromuscular blocking

agents, it is probably safest to avoid succinylcholine and other depolarizing neuromuscular blocking drugs in the post-acute near-drowning patient.

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