Excellent correlation was found between ultrasound predicted distance and measured needle distance to the epidural space. The mean needle distance measured in this study (4.5 cm) is greater than the mean (4.2 cm) of needle measurements obtained in 3,199 cases by Gutierrez[9] ($P < 0.05$). After each epidural puncture, measurement of the distance to the epidural space was made. Although he did not report an average distance, mean calculated distance from his reported data was 4.2 cm. The difference between the mean distances in the two studies may be due to either population difference or sampling error.

Advance knowledge of distance to the epidural space might have been very helpful in our four unsuccessful procedures. The ultrasound examination in each of these cases measured a greater ultrasound distance to the epidural space than that measured on the needle. This finding suggests insufficient advancement of the needle as the cause of the failure of the technique.

The "double shadow" in the three inadequate ultrasound examinations may have been due to thickness of the ligamentum flavin, causing reflection of ultrasound from both the epidural surface and the surface closer to the transducer. Other possibilities are beam-width error and reverberations of ultrasound. An investigation into this problem is currently proceeding.

Since most regional anesthesia is performed in areas of the body that are ultrasonically accessible and associated with soft tissue or skeletal landmarks that are ultrasonically detectable, this new imaging modality could have an expanded role in anesthetic practice. Certain advantages commend its use. The technique is noninvasive and has no harmful effect at the energy levels and time frames utilized. Obtaining a distance measurement in advance may avoid re-probing and subarachnoid puncture, resulting in less patient discomfort and greater safety. Ultrasonic examination of relevant anatomic landmarks may also be used for training residents in regional anesthesia. Some disadvantages also exist. The equipment is sophisticated, and scan performance and interpretation can be difficult at times. Although the use of ultrasound scanning in anesthesiology may not yet be clinically feasible, further developments in image-processing and transducer design may have significant impact on regional anesthetic techniques in the near future.

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Intravenously Administered Lidocaine Prevents Intracranial Hypertension during Endotracheal Suctioning

Martha F. Donegan, M.D.,* and Robert F. Bedford, M.D.†

One of the primary goals of intensive care for comatose patients with head trauma is normalization of intracranial pressure (ICP). Some necessary procedures, such as endotracheal tube suctioning, however, can result in marked increases in intracranial pressure despite the presence of paralysis and hypocarbia.[1] Intravenously administered lidocaine, 1.5 mg/kg, is effective in depressing the cough response of awake unmedicated subjects,[2] and is also effective in blocking the increases in ICP that often occur when the tracheas of anesthetized patients with space-occupying lesions are intubated.[3] This study was undertaken to

* Resident Fellow.
† Assistant Professor.
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Address reprint requests to Dr. Bedford.

determine whether pretreatment with intravenously administered lidocaine prevents increase of ICP associated with suctioning of the endotracheal tube in comatose patients.

Methods

The subjects were ten comatose patients who had closed head injuries. All had endotracheal tubes in place and were receiving controlled mechanical ventilation. In all, ICPs of more than 20 torr developed in response to endotracheal suctioning. The protocol was approved by the Medical Center's Human Investigation Committee, and informed consent was obtained from each patient's closest relative. Patients were monitored with an EKG, a radial arterial cannula and a central venous cannula, and a subarachnoid pressure screw. Pressures were transduced (Bentley Model 800 transducers) and recorded continuously (Brush Model 440 polygraph). All patients were receiving moderate hyperventilation ($Pa_{CO_2} \approx 30$), mannitol, and dexamethasone. Five patients received pentobarbital hourly to lower abnormal ICPs. Each study was performed in duplicate, once after intravenous administration of 2 per cent lidocaine (1.5 mg/kg) and once after administration of an equal volume of a saline placebo. The medications were given in randomized order and in double-blind fashion.

Baseline values for intracranial and arterial pressures and a sample for determination of arterial blood-gas tensions were obtained. The study medication was given and each patient was manually ventilated with 100 per cent oxygen for 2 min. Sterile endotracheal suctioning then was performed by the same ICU nurse in the same fashion for each pair of observations. Intracranial and arterial pressures were recorded for 5 min after completion of suctioning, and peak changes in these variables were tabulated. Paired studies were performed at least an hour apart on the same day and at the same time interval after doses of pentobarbital and/or mannitol. Statistical comparisons were performed using the Students t test for paired data. $P < 0.05$ was considered significant.

Results

Figure 1 summarizes our observations. Lidocaine, in combination with manually assisted ventilation, caused a significant reduction in ICP, which was not seen following saline pretreatment. After endotracheal suctioning ICP increased regardless of whether the patients received lidocaine or the placebo. Peak ICP after lidocaine pretreatment was not different from control ICP, whereas significant intracranial hypertension was observed when endotracheal suctioning was preceded by the placebo.

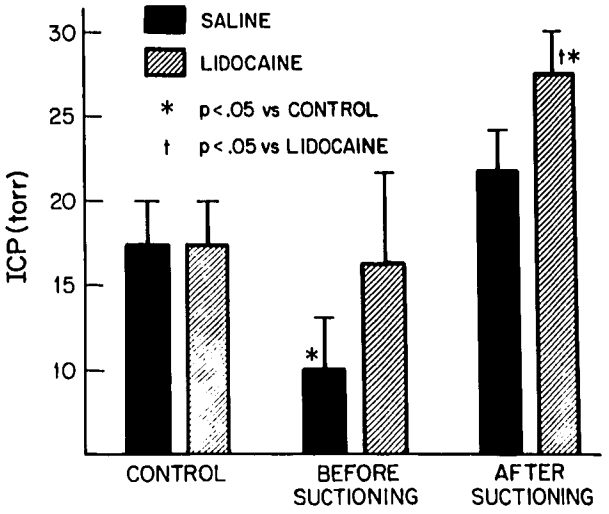

FIG. 1. Responses of intracranial pressure to endotracheal suctioning after pretreatment with intravenously administered lidocaine or a saline placebo. All values are means ± SE.

Control values for Pa_{CO_2} were 28 torr ± 2 (SEM) before lidocaine treatment and 31 torr ± 1 before the saline placebo was given. Corresponding values for mean arterial pressure (MAP) were 87 torr ± 4 before lidocaine and 87 torr ± 3 before the saline placebo. There was no change in MAP associated with a bolus injection of lidocaine, 1.5 mg/kg, but endotracheal suctioning did cause a significant increase in MAP after pretreatment with either lidocaine (12 torr ± 4, $P < .02$) or the saline placebo (12 torr ± 5, $P < .05$).

Lidocaine proved to be particularly effective in preventing intracranial hypertension among patients receiving pentobarbital therapy for increased ICP. After placebo pretreatment, ICP increased 19 torr (±4.7) over control values in the patients receiving pentobarbital, versus only a 5.7 torr (±3.2) increase among those not receiving barbiturate ($P < 0.05$ by t test for non-paired data). In contrast, lidocaine pretreatment limited the mean ICP increase to only 3.4 torr (±6.2) in pentobarbital-treated patients, versus 1.8 torr (±2.6) for those not receiving barbiturate ($P > 0.80$).

Discussion

Patients with intracranial disorders frequently have either elevated intracranial pressure or an intracranial pressure–volume relationship that predisposes them to precipitous increases in ICP.[1] Abrupt increases in ICP may result in herniation of the brain or impairment of cerebral perfusion, thus causing cerebral ischemia.

Drugs and techniques currently used to reduce ICP include corticosteroids, osmotic diuretics, barbiturates, and hypocarbia. As the patients in this study demon-

strated, however, these measures are not always effective in preventing increases in ICP associated with stimuli such as endotracheal suctioning. Pretreatment with intravenously administered lidocaine prevents these potentially harmful increases in ICP without causing major changes in either cardiorespiratory function or neurologic findings. Furthermore, since arterial pressure was not affected by lidocaine treatment, whereas ICP was reduced significantly, cerebral perfusion pressure (MAP − ICP) was always improved. Similarly, we have found lidocaine to be particularly useful in improving cerebral perfusion pressure in clinical situations where elevated intracranial pressure was complicated by arterial hypotension (Bedford and Donegan, unpublished data).

Intravenous administration of lidocaine has been used to blunt the cough reflex in both awake[2] and anesthetized[4] patients. When given before endotracheal intubation, it prevents arterial hypertension and tachycardia in patients with coronary-artery disease[5] and also prevents intracranial hypertension in patients with brain tumors.[3] These actions appear to be mediated primarily through depression of brainstem neuronal activity.[6]

Intravenously administered lidocaine is known to act as a general anesthetic, and this may explain its ability to reduce ICP significantly. Although the dose of lidocaine used in the present study does not usually cause somnolence in human volunteers, it does potentiate the action of inhalational anesthetics,[7] and the present study indicates that it also improves ICP control during barbiturate therapy. Lidocaine reduces the cerebral metabolic rate for oxygen and increases cerebrovascular resistance.[8] Thus, the acute lowering of ICP after lidocaine probably reflects reductions in both cerebral blood flow and cerebral blood volume, the same mechanism by which barbiturate infusion is thought to reduce ICP.[9]

In summary, intravenously administered lidocaine appears to be a potentially valuable adjunct in neurointensive care. When given to patients with closed head trauma, in whom increased ICP is likely to develop, lidocaine, 1.5 mg/kg, effectively reduces ICP and also prevents intracranial hypertension associated with endotracheal suctioning.

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Epidural Block in Obstetrics Followed by Aseptic Meningoencephalitis

Julius Neumark, M.D.,* Wilfried Feichtinger, M.D.,† Alfred Gassner, M.D.‡

Neurologic sequelae of spinal and epidural blocks are very rare,[1,2] but the use of these blocks is somewhat controversial because of the dreadful damage caused by such sequelae. On the other hand, spinal and epidural blocks are routinely used all over the world. The rare neurological sequelae of these blocks are likely to be coincidental rather than due to the blocks. Aseptic meningitis itself forms a small part of these rare complications, and is said to occur mainly after spinal block. The incidence of aseptic meningitis following epidural block is unknown. Usabiaga reviewed 780,000 epidural blocks and found only two cases among 69 neurologic complications or coincidences.[2]

* Obstetric Anesthesiologist, Department of Anesthesiology.
† Resident, 2nd Department of Obstetrics and Gynecology.
‡ Resident, 1st Department of Internal Medicine.

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Address reprint requests to Dr. Neumark: Allgemeines Krankenhaus der Stadt Wien, Klinic Für Anaesthesie Und Allgemeine Intensivmedizin der Universität Wien, A-1090 Wien, Spitalgasse 23, Austria.