In summary, although bilateral weakness of the lower extremities following an epidural anesthetic raises the suspicion of neurologic complication of the epidural anesthesia, lower-extremity weakness following vaginal delivery may result from iliopsoas muscle strain without associated neurologic injury as in our case. Accurate diagnosis of this complication, which can be made primarily on the basis of physical examination alone, may obviate the need for unnecessary and often costly diagnostic studies and can assure the patient of a benign prognosis and eventual recovery.

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Isoflurane for Neuroanaesthesia: Risk Factors for Increases in Intracranial Pressure

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Isoflurane often is used for neuroanaesthesia because it has the least effect on cerebral blood flow (CBF)† and cerebrospinal fluid pressure of the currently available volatile anesthetics. While animal studies have tended to reinforce this clinical impression, some clinicians have questioned whether isoflurane should be labeled as the agent of choice for neuroanaesthesia. We have been impressed that an occasional patient with an intracranial mass lesion will have increased intracranial pressure (ICP) when isoflurane is administered, despite prior institution of modest hyperventilation (Fig. 1). We undertook this study to identify the risk factors that make intracranial hypertension a likely event during isoflurane anesthesia.

MATERIALS AND METHODS

Fourteen unpremedicated patients (ages 42–73 years) were scheduled for elective craniotomy for excision of malignant supratentorial neoplasms. The protocol was approved by our institution’s human studies committee. Computed tomographic (CT) scans of patients’ heads were obtained within 3 days before operation and were interpreted by a neuroradiologist with regard to size, type, and location of lesion; the presence of midline shift; the degree of effacement of the lateral ventricles; and the extent of cerebral edema surrounding the tumor. Peritumor edema was quantitated on a visual scale ranging from 0 (no edema) to 3+ (edema present throughout the cerebral hemisphere).

General anesthesia was induced with thiopental (3 mg/ kg iv) and maintained with nitrous oxide, 70% in oxygen. Endotracheal intubation was facilitated with pancuronium, 0.1 mg/kg iv and was performed after a second dose of thiopental, 2 mg/kg, iv, plus lidocaine, 1.5 mg/kg, iv. Ventilation was controlled to maintain end-tidal carbon dioxide tension (PETCO₂) at approximately 26 mmHg (Beckman LB2). A radial arterial catheter was inserted either before or immediately after induction of general anesthesia.
Immediately following induction of anesthesia, a subarachnoid bolt was inserted contralateral to the side of the supratentorial tumor, and ICP and arterial pressure were referenced to the level of the external auditory meatus and continuously transduced (Bentley Model 500\textsuperscript{a} transducers) and recorded (Brush model 440\textsuperscript{a} strip-chart recorder). Pa\textsubscript{CO\textsubscript{2}} and intracranial elastance (ΔP/ΔV) near the subarachnoid bolt were determined the latter of which by injecting 1 ml mock cerebrospinal fluid (CSF) through the bolt while the corresponding change in ICP was recorded.\textsuperscript{6} Mean arterial pressure (MAP) was calculated from the formula

\[
\text{systolic pressure} - \text{diastolic pressure} \div 3 + \text{diastolic pressure},
\]

and cerebral perfusion pressure (CPP) was computed as MAP-ICP.

After stable intracranial and arterial blood pressures were observed, the scalp incision for craniotomy was performed, and isoflurane, 1.1% ± 0.3 SD was added to the inspired anesthetic gas mixture. Changes in ICP and MAP were recorded continuously until the cranium was opened (mean duration = 10 min ± 5.8 SD). Data obtained were subjected to statistical analysis using Student’s \( t \) test for nonpaired data. \( P < 0.05 \) was regarded as significant.

### RESULTS

Before scalp incision and administration of isoflurane, the mean ICP in the 14 patients was 13.9 mmHg ± 1.9 SE and MAP was 101 mmHg ± 4.0 SE. During isoflurane anesthesia and surgical dissection, six of the patients sustained increases in ICP that ranged from 5 to 13 mmHg, resulting in ICPs between 15 and 27 mmHg. These patients were designated as Group 1 (table 1). The remaining eight patients did not develop any increase in ICP and for purposes of comparison they were designated as Group 2. As expected, MAP decreased in both groups in response to isoflurane, but cerebral perfusion pressure was most adversely affected in Group 2. No change in ICP or MAP was so extreme as to require discontinuation of isoflurane or administration of other pharmacologic agents before the cranium was opened and the study was terminated.

The potential risk factors for isoflurane-induced increases in ICP that were examined are summarized in table 2. No significant difference between the two groups of patients was found with regard to measured intracranial elastance, size, or location of tumor; amount of edema surrounding the tumor; Pa\textsubscript{CO\textsubscript{2}}; or inspired isoflurane concentration. We did note, however, a striking difference between the two groups with regard to the presence of shift of midline intracranial structures seen with preoperative CT scans. All of the patients in Group 1 had some degree of midline shift, whereas only one of the patients in Group 2 demonstrated any degree of midline shift (\( P < 0.01 \) by Fisher’s Exact Test).

**TABLE 1. Changes during Isoflurane**

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Intracranial Pressure (mmHg)</th>
<th>Mean Arterial Pressure (mmHg)</th>
<th>Cerebral Perfusion Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6</td>
<td>8.0 ± 1.9 *</td>
<td>-30 ± 6</td>
<td>-37 ± 5 *</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
<td>-1.8 ± 0.9</td>
<td>-23 ± 8</td>
<td>-21 ± 4</td>
</tr>
</tbody>
</table>

All values = mean ± SE.

\* \( P < 0.05 \) versus Group 2.
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<table>
<thead>
<tr>
<th>Group</th>
<th>Measured Intracranial Distance (mmHg/ml)</th>
<th>Tumor Diameter (cm)</th>
<th>Midline Shift (mm)</th>
<th>Tumor Edema (0-5+)</th>
<th>Paco₂ (mmHg)</th>
<th>Inspired Isoflurane Concentration (vol%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.2 ± 0.5</td>
<td>3.8 ± 0.2</td>
<td>9 ± 0.5*</td>
<td>2.0 ± 0.5</td>
<td>29 ± 0.8</td>
<td>1.2 ± 0.2</td>
</tr>
<tr>
<td>2</td>
<td>3.7 ± 0.7</td>
<td>3.4 ± 0.2</td>
<td>1 ± 1.0</td>
<td>1.8 ± 0.6</td>
<td>30 ± 0.9</td>
<td>1.0 ± 0.1</td>
</tr>
</tbody>
</table>

All values = mean ± SE.

\*P < 0.05 versus Group 2.

DISCUSSION

The proper place of the volatile anesthetic agents in the practice of neurosurgical anesthetics has remained controversial ever since Jennett et al. first demonstrated that halothane could cause increases in cerebrospinal fluid pressure in patients with intracranial mass lesions. While Adams et al. showed that establishment of hypocapnia before inhalation of halothane could prevent increases in lumbar CSF pressure in many neurosurgical patients, it is accepted that halothane increases cerebral blood flow (CBF) in excess of the brain's metabolic requirements, that it causes an increase in resistance to CSF outflow, and that it causes more protrusion of brain through a cranietomy than either enflurane or isoflurane. Enflurane also causes a dose-dependent increase in CBF when arterial blood pressure remains normal, and, furthermore, results in both increased production of CSF and increased resistance to CSF outflow. Since ICP can rise in response to an increase in either cerebral blood volume or CSF volume, it is not unexpected that intracranial hypertension results when patients with intracranial mass lesions receive enflurane.

In comparison with halothane and enflurane, isoflurane is considered to be the superior agent for neurosurgical anesthesia. CBF is lower at clinically relevant concentrations, and neither CSF production nor resistance to CSF outflow is increased. When Adams et al. administered isoflurane to patients with intracranial mass lesions, they found that lumbar CSF pressure was not increased as long as Paco₂ was maintained below 30 mmHg, even when hypocarbia was instituted after isoflurane was begun. Further, Campkin studied two patients who had a mean ICP of 12.5 mmHg before receiving isoflurane and concluded that isoflurane is "a suitable agent even when intracranial hypertension is present because the ICP of both patients decreased during inhalation of 1.0-1.5% isoflurane.

Isoflurane, however, does cause dose-dependent increases in CBF both in humans (when arterial blood pressure is held relatively constant) and in dogs (when arterial blood pressure is allowed to decrease). Adams et al. observed that lumbar CSF pressure increased in normocarbic patients with intracranial mass lesions who received isoflurane, and Todd and Drummond likewise found dose-dependent increases in ICP in cats receiving isoflurane. It is not surprising, then, that the role of isoflurane in neurosurgical anesthesia remains unsettled.

The present study was undertaken because occasionally we have observed patients with acute head trauma who have sustained marked increases in ICP when given isoflurane, despite Paco₂ tensions below 25 mmHg. The present study indicates that patients with brain tumors, who may have less compromised intracranial compliance than those with head injuries, may be similarly at risk. While we found no difference in measured intracranial elastance between the patients whose ICPs increased and those who did not, we believe this reflects the limitations of compliance testing through a subarachnoid bolt. Preoperative head CT scans that show a lateral shift of midline structures indicate a deterioration in intracranial compliance as expanding mass impinges on contralateral normal brain. In this situation it appears that isoflurane is not necessarily a safe anesthetic, despite modest hyperventilation, although it is probable that enflurane or halothane would prove to be even more deleterious.

In summary, we found that low-dose isoflurane may cause increases in ICP in patients with malignant brain tumors, despite prior institution of modest hyperventilation. We conclude that isoflurane may not be a benign anesthetic in patients known to be at risk for increases in ICP, particularly those with midline shift of brain structures apparent on preoperative CT scan.

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Additional Inspiratory Work in Intubated Patients Breathing with Continuous Positive Airway Pressure Systems

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Spontaneous breathing with continuous positive airway pressure (CPAP) increases arterial oxygenation in patients with adult respiratory distress syndrome (ARDS).1 Two types of CPAP systems are currently used, continuous flow and demand valve systems. Clinically, the latter has the advantage of being more economical with fresh gas but is often poorly tolerated by patients. Studies in normal volunteers who were sitting have shown the total work of breathing to be increased more with demand valve systems than with a high-flow CPAP circuit without valves.2,3 However, no data were available in supine patients whose tracheas were intubated.

Under these conditions, both the changes in pleural pressure and the values of pleural pressure relative to atmospheric pressure are required to distinguish the part of work done by the patient and that done by the CPAP device.3 The use of esophageal pressure for this determination is questionable in supine subjects.4,5 However, the increase in external inspiratory work added by a CPAP system can be evaluated. This superimposed inspiratory work (SIW) can be determined from simultaneous recordings of the flow and pressure difference across the CPAP device.5 We measured SIW in supine intensive care patients spontaneously breathing with CPAP and compared three demand valve systems with a continuous flow circuit.

Materials and Methods

Patients. Four studies were carried out in each of 12 supine patients (11 male patients and one female patient) undergoing ventilatory treatment with CPAP according to a protocol approved by our institution’s ethical committee on human research. Informed consent concerning the nature and purpose of the study was obtained from each patient. CPAP ventilation was being used in the weaning process in four patients recovering from ARDS secondary to severe trauma and in eight patients recovering from major surgical procedures. All 12 patients had arterial blood gases within normal limits at the time of the study, with an inspired oxygen concentration less than