Clinical Reports
BURNELL R. BROWN, JR., M.D., PH.D., Editor

Anesthesiology

Changes in Neurologic Status and Intracranial Pressure Associated with Sodium Nitroprusside Administration

M. L. Marsh, M.D.,* H. M. Shapiro, M.D.,† R. W. Smith, M.D.,‡ L. F. Marshall, M.D.§

In neurosurgical patients with intracranial mass lesions, laryngoscopy and endotracheal intubation are often associated with abrupt increases in arterial blood pressure (BP) and intracranial pressure (ICP). In an effort to control this untoward response, we rely upon adequate anesthetic depth, muscle relaxants, and occasionally, antihypertensive drugs. Until recently, we employed small bolus doses of trimethaphan (3–4 mg/70 kg), a ganglionic blocking drug, as the antihypertensive agent to offset transient increases in BP. Then, studies by Stulken and Sokoll prompted a change to sodium nitroprusside (SNP) for BP control in the induction period. They found that trimethaphan increased ICP in cats, an effect not seen when SNP was used to decrease BP. Because ICP monitoring is often established prior to induction in our high-risk patients, we had the opportunity to observe the effects of preinduction infusions of SNP in awake man.

METHODS

Four conscious patients who had hydrocephalus secondary to a posterior fossa tumor (acoustic neuroma) were observed during SNP administration. Informed consent for monitoring of ICP and its regulation as needed was obtained. Each patient was brought to the operating room either unpremedicated or lightly sedated (diazepam, 5–10 mg, given orally). The first two had ventricular cannulas inserted in the surgical suite with the use of local anesthesia, while Patients 3 and 4 had previously installed intraventricular catheters. Routine peripheral and central venous catheters were placed, and a radial-artery catheter was positioned for continuous BP and intermittent arterial blood-gas monitoring. Lead II of the ECG was continuously recorded. The patients were supine, and all pressures were zero-referenced to the mid-thoracic level. These pressures were recorded via Statham P23Db transducers and displayed on a Hewlett-Packard 7788A oscilloscope.

Once baseline values were established for the BP, ICP and PaCO₂, a test dose of SNP was administered through the central venous catheter as a small bolus (20 μg) or a slow infusion (0.02 per cent solution). An arbitrary end point consisted of a clinically significant increase in ICP (>10 torr) or a decrease in BP (>20 torr). The PaCO₂ was measured before and immediately after each SNP infusion. Throughout the trial, the patient’s level of consciousness was assessed, and room air was the inspired gas.

RESULTS

All four patients experienced increases in ICP (X = 14 ± 4 torr), with three of four actually doubling their initial pressures (table 1). Patient 1, who received the only bolus SNP injection, had the greatest ICP increment (20 torr) without any change in BP (fig. 1). Patients 2 and 3, who had the lowest initial ICP values (9 and 11 torr, respectively), experienced no change in their states of consciousness, although their mean BP values decreased to less than those of the others. Patients 1 and 4, who had mild to moderate intracranial hypertension prior to administration of SNP, experienced significantly greater ICP peaks, marked by decreased level of consciousness and complaints of headache and dizziness, respectively. Patient 1, who responded to vocal stimuli prior to SNP administration did not respond to moderately
painful stimuli after receiving the drug. With the appearance of clinical signs of intracranial decompensation, anesthesia was induced in each patient with thiopental. In no case were there any signs of brainstem compression or focal deficits. No significant difference in $P_{aCO_2}$ before and after the SNP infusion was recorded for any patient.

**DISCUSSION**

Although it was introduced into clinical practice by Johnson in 1929, SNP has only recently been considered a drug of choice for elective, predictable controlled hypotension during anesthesia. While its systemic and cardiovascular effects were appraised very early, its specific influences on human cerebral blood flow (CBF) and metabolism were not reported until 1974. Griffith et al. used SNP to modulate the BPs of 20 patients undergoing surgical procedures for cerebral aneurysms. They concluded that SNP decreased cerebral vascular resistance (CVR) significantly while preserving CBF and cerebral metabolic rate for oxygen ($CMR_O_2$) during the hypotensive period. ICP was not specifically monitored, and it is not clear whether any or all patients had had prior intracranial hemorrhage.

In 1975, Stulken and Sokoll first addressed the question of the ICP effects of two antihypertensive agents, SNP and trimethaphan. In their experiment, cats without intracranial masses were employed, and they reported that trimethaphan-induced hypotension was associated with significant ICP increases when compared with hypotension from SNP or hemorrhagic shock. Most studies of the CBF effects of SNP have shown a primary decrease in cerebrovascular resistance. The resultant alteration in CBF depends upon the absolute BP level associated with SNP administration; for instance, when the decrease is relatively great, CBF will decrease, whereas modest decreases in BP will result in either no change or an actual increase in CBF. ICP changes generally parallel CBF alterations when intracranial compliance is low.

As we prepared this report, Turner et al. and Cottrell et al. described the effects of SNP in anesthetized patients with increased ICP. Turner et al. found that SNP administered to normocarbic subjects could effect significant increases in ICP with only slight decreases in BP, and that the ICP effect was attenuated, but not obliterated, by hyperventilation. In contrast, trimethaphan administered to normocarbic subjects did not precipitate any ICP increase except in two cases of "severe brain compression." Cottrell's study demonstrated that SNP decreases the systemic BP and CVR reciprocally, augmenting CBF and intracranial hypertension in a stepwise fashion.

Our findings are consistent with those obtained by Turner and Cottrell. However, by administering SNP to unanesthetized patients, we have obviated the known cerebral vascular influences of anesthetic drugs and $P_{aCO_2}$ manipulation. We have demonstrated that increased ICP and neurologic dysfunction can be aggravated by a systemic vasodilator drug. Further, we agree that the mode of SNP infusion plays

**TABLE 1. Effects of SNP on Blood Pressure (BP), Intracranial Pressure (ICP), and Neurologic Status**

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Pre-SNP Infusion</th>
<th>Post-SNP Infusion (BP Nadir)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BP (torr)</td>
<td>ICP (torr)</td>
</tr>
<tr>
<td>Patient 1</td>
<td>43</td>
<td>85</td>
</tr>
<tr>
<td>Patient 2</td>
<td>55</td>
<td>100</td>
</tr>
<tr>
<td>Patient 3</td>
<td>63</td>
<td>85</td>
</tr>
<tr>
<td>Patient 4</td>
<td>42</td>
<td>110</td>
</tr>
</tbody>
</table>

* Clinical data for four patients with large acoustic neuromas who showed marked ICP increases and mild to moderate BP decreases in response to small doses of SNP. Patient 1 experienced a concurrent decrease in his level of consciousness, progressing from lethargic to stuporous. Patient 4 complained of headaches and dizziness following the infusion. No significant change in $P_{aCO_2}$ was recorded.

† CPP = cerebral perfusion pressure.

‡ Lethargic = responsive to verbal stimuli; stuporous = responsive to painful stimuli only.

§ Complained of headache and dizziness.
some role in determining the rate and extent of the ICP response, as seen in Patient 1. The implications of using a small bolus dose are relevant, in that initial SNP infusion rates must be established empirically and mechanical infusion pumps often function in a pulsatile fashion.

While the precise mechanism for intracranial hypertension associated with SNP remains unknown, this possibility should be considered when deliberate hypotension or blood pressure control is contemplated. When elective hypotension is needed in such patients, SNP administration should be carefully titrated, and probably deferred until the skull is opened.

REFERENCES

Acute Subdural Hematoma—An Unusual Sequela to Lumbar Puncture


Intracranial hemorrhage following lumbar puncture or subarachnoid block in the absence of under-

* Assistant Professor, Department of Anesthesiology, University of Washington.
† Professor and Assistant Chairman, Department of Anesthesiology; Professor, Obstetrics and Gynecology, University of Colorado. Current address: Professor and Chairman, Department of Anesthesiology, Ohio State University, 410 West 10th Avenue, Columbus, Ohio 43210.
‡ Resident, Department of Anesthesiology, University of Washington.
§ Resident, Department of Surgery, University of Washington.

Reaching the Departments of Anesthesiology, University of Washington School of Medicine, Seattle, Washington, and University of Colorado Medical Center, Denver, Colorado. Accepted for publication February 20, 1979.

Address reprint requests to Dr. Pavlin: Department of Anesthesiology, RN-10, University of Washington School of Medicine, Seattle, Washington 98195.


Lying intracranial disease is not often recorded.1-4 The following two cases, in which large subdural hematomas occurred within a week of lumbar puncture, are reported to draw attention to the possible relationship of intracranial hemorrhage to previous dural puncture.

REPORT OF TWO CASES

Patient 1. A 23-year-old pregnant woman was admitted to hospital at 40 weeks of gestation for induction of labor. Pregnancy had been uneventful, and there was no significant history of medical illness. Effective labor was established three hours following artificial rupture of the amnion and commencement of an intravenous infusion of oxytocin. Lumbar epidural analgesia was then requested for relief of labor pains; an inadvertent dural puncture with an 18-gauge Crawford needle tip occurred during the attempted epidural. No further effort was made to establish regional analgesia. The infant was subsequently delivered.

0003-3027/79/1000/0338 $00.65 © The American Society of Anesthesiologists, Inc.