

their use be undertaken with extreme care. Happily, in the anesthetic management of our patient, no airway device was necessary.

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Intracranial Hypertension Caused by Nitroglycerin

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Nitroglycerin has gained widespread acceptance as a drug for control of myocardial ischemia, as well as for induction of hypotension during anesthesia and operation.^{1,2} We observed a brisk increase in intracranial pressure (ICP) following the use of nitroglycerin for cardiac therapy in a patient with intracranial hypertension. This observation suggests a need for caution when using nitroglycerin in management of patients who have intracranial hypertension.

REPORT OF A CASE

A 67-year-old white man (height 183 cm, weight 88 kg) who had a preoperative diagnosis of posterior-fossa brain tumor, obstructive hydrocephalus, and intracranial hypertension was scheduled for craniotomy. The medical history included essential hypertension, well controlled with Diazide® (50 mg triamterene, 25 mg hydrochlorothiazide), one tablet daily, biweekly episodes of angina pectoris relieved by sublingually administered nitroglycerin, and residual paresis of the right leg following a cerebrovascular accident, which had occurred six months prior to admission. Dexamethasone (4 mg, four times a day) was started two days preoperatively to control symptoms of intracranial hypertension.

With informed consent, the patient agreed to participate in a double-blind study of the effects on ICP of an intravenously administered benzodiazepine premedicant. The protocol included administration of this drug or a placebo 45 min prior to induction of anesthesia. ICP (via ventricular catheter), arterial pressure (BP),

central venous pressure (CVP), and electrocardiogram (ECG) were monitored during the 45-min observation period and intraoperatively. All monitored physiologic indices were continuously recorded on a Hewlett-Packard polygraph.

Forty-five minutes after receiving either the premedicant or placebo, iv, BP was 150/95 torr; ICP, 12 torr. A borderline-normal cerebrospinal fluid volume-pressure response was elicited at this time; 20 sec after the injection of 1 ml sterile saline solution into the ventriculostomy, an increase in ICP to 4 torr above baseline occurred (abnormal response, ICP gain >5 torr).³ Anesthesia was then induced with thiopental, 250 mg. Pancuronium bromide, 8 mg, was given iv and hyperventilation initiated. Anesthesia was maintained with incremental doses of thiopental (460 mg total) and an inspired gas mixture of 60 per cent N₂O in O₂. When PaCO₂ was 21 torr, halothane 1 per cent was added to the inspired mixture for 10 min and, thereafter, the concentration was increased to 2 per cent.

After 18 min (10 min at 1 per cent, 8 min at 2 per cent) of exposure to halothane, a transient tachyarrhythmia developed; it spontaneously reverted to a sinus pattern. However, there appeared marked ST-segment depression and QRS widening. Since myocardial ischemia was suspected, 1 ml 0.08 per cent solution of nitroglycerin (equivalent to two tablets administered sublingually) was given intranasally on two separate occasions. Figure 1 shows the responses of BP and ICP to both doses. The ICP prior to the first dose of nitroglycerin was 18 torr. This climbed to a peak of 40 torr within 2½ minutes of nitroglycerin administration. Removal of 5 ml cerebrospinal fluid (CSF) curtailed a further increase in ICP. Following the first dose of nitroglycerin, blood pressure increased from 120/80 to 135/85 torr within 30 sec, but then decreased to 100/60 torr after 2 min.

Since the ECG was unchanged, a second dose of nitroglycerin was given 8 min after the first. After the second dose, ICP increased from 22 to 48 torr within 2½ min. Blood pressure initially increased from 122/80 to 175/100 torr within the first minute, but then decreased to 120/60 torr after 2½ min. To offset intracranial hypertension, 200 mg thiopental were given iv when ICP was highest. This produced reductions in both ICP and BP to 16 and 80/45 torr, respectively. The ECG returned to its preanesthetic configuration. With laryngoscopy and intubation, blood pressure increased without the appearance of ischemic ECG changes.

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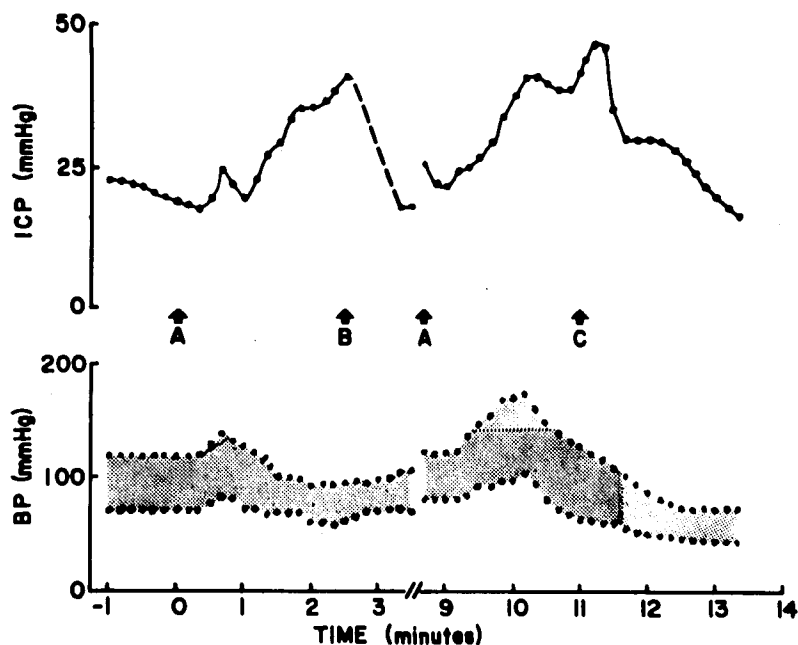


FIG. 1. Blood pressure (BP) and intracranial pressure (ICP) responses to two doses of nitroglycerin. A, 1 ml 0.08 per cent nitroglycerin intranasally; B, withdrawal of 5 ml cerebrospinal fluid; C, 200 mg thiopental, iv. The dashed line indicates a period of cerebrospinal fluid withdrawal when ICP recording was not possible.

The anesthetic course was marked by two more episodes of ST-segment depression and QRS widening, diagnosed (with a 12-lead ECG) as a rate-related intermittent left-bundle-branch block. No further nitroglycerin was given. A large acoustic neuroma and associated cyst were removed surgically. The postoperative course was uneventful, except for transient paralysis of the right facial nerve. Serial cardiac isoenzymes were within normal limits.

DISCUSSION

The large increase in ICP associated with both doses of nitroglycerin occurred within 30 sec. Prior to this, halothane had produced a gradual elevation of ICP, resetting a stable baseline from 12 to 18 torr after 18 min of anesthetic exposure. No other drug was given immediately prior to either dosage of nitroglycerin.

That nitroglycerin produces headache in the awake patient suggests that it has a cerebral as well as a systemic vasodilating effect.

Cerebral vasodilation can increase cerebral blood volume (CBV) and elevate ICP, particularly in the circumstance of reduced intracranial compliance. ICP-elevating effects have been reported to occur with other systemic vasodilators, such as nitroprusside and hydralazine.⁴ Whether cerebral vasodilation produced by nitroglycerin is additive or synergistic with the vasodilating effect of halothane cannot be determined from our data. Certainly the increase in CBV due to halothane could accentuate the ICP-elevating effect of a subsequently applied vasodilator.

This ICP-elevating effect of nitroglycerin may be to some extent ameliorated by the improved venous

return associated with the sitting position. However, depending on the state of intracranial compliance, a significant increase in ICP and/or decrease in cerebral perfusion pressure might still occur.

The increase in BP observed in this patient is interesting but difficult to explain. It may have reflected compression of the brainstem, resulting in a reflexive blood pressure increase similar to that seen during ICP plateau waves.⁵ The peripheral vasodilating effect of the drug may have resulted in the subsequent decrease in blood pressure.

Our observations are clinically significant since nitroglycerin has recently been suggested as an agent of choice for inducing hypotension during neurosurgical procedures.⁶ These findings suggest, also, that nitroglycerin should be used with caution for the treatment of myocardial ischemia in patients who have intracranial hypertension. An alternative, such as propranolol, may be a more appropriate choice for control of angina in these patients.

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