

month-old child,¹⁴ who was successfully treated with dantrolene. Because MH occurred in the mother in our case, the possibility of MHS has to be considered in the infant due to the hereditary nature of this condition. Therefore, all precautions were taken, except for the preoperative administration of dantrolene. The authors thought that, because of the muscle relaxant properties of this drug, it should be avoided in the presence of hypotonia. However, dantrolene was immediately available for intravenous use in the event of MH occurring.

REFERENCES

1. Liebenschutz F, Mai C, Pickerodt VWA: Increased carbon dioxide production in two patients with malignant hyperthermia and its control by dantrolene. *Br J Anaesth* 51:899-903, 1979
2. Willatts SM: Malignant hyperthermia susceptibility—management during pregnancy and labour. *Anaesthesia* 34:41-46, 1979
3. Wadhwa RK: Obstetric anesthesia for a patient with malignant hyperthermia susceptibility. *ANESTHESIOLOGY* 46:63-64, 1977
4. Crawford JS: Hyperpyrexia during pregnancy. *Lancet* 1:1244, 1972
5. Harrison GG, Saunders SJ, Biebuyck JF, et al: Anaesthetic in-

- duced malignant hyperpyrexia and a method for its prediction. *Br J Anaesth* 41:844-854, 1969
6. Britt BA, Gordon RA: Three cases of malignant hyperthermia with special consideration of management. *Can Anaesth Soc J* 16:99-105, 1969
7. Britt BA, Kalow W: Malignant hyperthermia: a statistical review. *Can Anaesth Soc J* 17:293-315, 1970
8. Gronert GA, Thompson RL, Onofrio BM: Human malignant hyperthermia: awake episodes and correction by dantrolene. *Anesth Analg (Cleve)* 59:377-378, 1980
9. Fitzgibbons DC: Malignant hyperthermia following preoperative oral administration of dantrolene. *ANESTHESIOLOGY* 54:73-75, 1981
10. Wingard DW: Malignant hyperthermia—acute stress syndrome in man. *Malignant Hyperthermia*. Edited by Henschel ED. New York, Appleton Century Crofts, 1977, pp 79-85
11. Mathieu A, Bogosian AJ, Ryan JF, et al: Recrudescence after survival of an initial episode of malignant hyperthermia. *ANESTHESIOLOGY* 51:454-455, 1979
12. Beldavs J, Small V, Cooper DA, et al: Post-operative malignant hyperthermia: a case report. *Can Anaesth Soc J* 18:202-212, 1971
13. Sewall K, Flowerdew RMM, Bromberger P: Severe muscular rigidity at birth: Malignant hyperthermia syndrome? *Can Anaesth Soc J* 27:279-282, 1980
14. Mayhew JF, Rudolph J, Tobey RE: Malignant hyperthermia in a six month old infant: a case report. *Anesth Analg (Cleve)* 57:262-264, 1978

Anesthesiology
56:146-147, 1982

Intracranial Hypertension Following Cross-clamping of the Thoracic Aorta

CHARLES B. HANTLER, M.D.,* AND PAUL R. KNIGHT, M.D., PH.D.†

Management of intracranial pressure is of prime concern during anesthesia in a patient with closed head injury. Many intraoperative manipulations may adversely affect control of intracranial pressure. In this report, a case is described in which thoracic aorta cross-clamping resulted in a sudden increase in both arterial and intracranial pressure.

REPORT OF A CASE

An otherwise healthy 18-year-old man presented to the emergency room with multiple injuries following a motor vehicle accident. Neu-

rologic examination revealed an unresponsive patient without localizing signs. An EMI scan showed diffuse cerebral edema. Aspiration of the abdominal cavity revealed free blood in the peritoneum. An uneventful splenectomy was performed following exploratory laparotomy. After removal of the spleen, a subarachnoid intracranial pressure monitor was inserted. Ventilation was controlled to maintain the P_{aCO_2} in the range of 25-30 mmHg. Intravenous medications included 4 mg dexamethasone every 6 h, and 12.5 g mannitol every 6 hours. With systolic blood pressure in the ranges of 120-130, 130-140, and 140-150 mmHg, the corresponding mean intracranial pressures (ICP) were 9.3, 11.5, and 10 mmHg, respectively.

An aortogram, performed because of a widening mediastinal shadow on the chest roentgenogram, revealed dissection of the aortic arch distal to the left subclavian artery. Eight hours after the initial operation, the patient returned to the operating room where a left thoracotomy was performed. Anesthesia was maintained with intermittently administered intravenous thiamylal and inhalational enflurane. Enflurane was chosen to prevent intraoperative hypertension during one lung anesthesia. If enflurane had caused an elevation of ICP despite hyperventilation, substitution of narcotics and additional barbituates was planned; however, ICP did not increase after induction. Ventilation was controlled to maintain a P_{aCO_2} between 25 and 30 mmHg. A Gott bypass shunt was inserted from the area of the left ventricular apex to the descending aorta. Prior to thoracic aorta cross-clamping, there were only minor fluctuations in arterial or intracranial pressures. Following cross-clamping of the aorta, the systolic blood pressure immediately increased from 100 to 130 mmHg, CVP was unchanged, while ICP increased from 18 to 32 mmHg. An intracranial pressure wave of 32 over 18 mmHg was also present. Sodium nitroprusside (SNP) was initiated to lower blood pressure in an attempt to reduce

* Research Fellow, Department of Anesthesiology, University of Michigan Medical Center, Ann Arbor, Michigan.

† Assistant Professor, Department of Anesthesiology, University of Michigan Medical Center, Ann Arbor, Michigan.

Supported by Research Career Development Award, GMA00477, United States Department of Health, Education, and Welfare, Public Health Service.

Received from the University of Michigan Medical Center, Department of Anesthesiology, Ann Arbor, Michigan 48109. Accepted for publication August 11, 1981.

Address reprint requests to Dr. Hantler: University of Michigan Medical Center Department of Anesthesiology Ann Arbor, Michigan 48109.

Key words: Brain: intracranial pressure. Arteries: aorta. Surgery: neurologic.

ICP. Systolic blood pressure was successfully reduced to 100 mmHg while ICP decreased to only 28 mmHg. Thiarnylal, 200 mg, was given intravenously over a 5-min period. The ICP then decreased to 20 mmHg, while systolic blood pressure remained at 100 mmHg. The sodium nitroprusside was discontinued and the systolic blood pressure returned to 120 mmHg while the ICP remained below 20 mmHg. The operation was completed without further incidence and the patient recovered without neurologic sequelae.

DISCUSSION

This case represents an unusual cause of intracranial hypertension. The patient showed evidence of decreased intracranial compliance as evidenced by edema on an EMI scan. Preoperatively ICP showed only minor fluctuations with changes in blood pressure, suggesting either functioning cerebral autoregulation or that the patient had not yet reached the steep part of the intracranial compliance curve.

Intraoperatively, prior to thoracic aortic cross-clamping, the P_{aCO_2} was between 25 and 30 mmHg, and intracranial pressure remained below 15 mmHg. This would suggest that the expected rises in cerebral blood flow (CBF) secondary to enflurane were prevented successfully with hyperventilation; this has been reported previously for halothane.¹ At the time of aortic cross-clamping, there was an immediate increase in arterial blood pressure and in ICP. Since the CVP was unchanged, the changes suggest an increase in CBF secondary to clamping of the thoracic aorta. The changes in ICP could be predicted based on the rapid increase in blood pressure² and/or the loss of autoregulation secondary to enflurane anesthesia.‡

Although SNP returned systemic blood pressure to preclamp levels, there was no significant reduction in ICP. Cotrell *et al.*³ found an increase in ICP on exposure to SNP in patients with intracranial tumors. Turner *et al.*⁴ found a similar increase in ICP in patients with either intracranial tumor or aneurysm. In that study, prior hyperventilation reduced the mean increase in ICP following administration of SNP; however, there were large individual variations. Indeed, several patients had large increases in ICP despite hyperventilation. Ivanovitch, *et al.*⁵ have shown SNP to be a direct cerebral vasodilator in the goat. On the other hand, Stoyka *et al.*⁶ have demonstrated that in the dog, CBF is maintained despite a decrease in cerebral perfusion pressure caused by SNP. In the presence of low intracranial compliance,

SNP may produce an elevation in ICP by a vasodilation-induced increase in cerebral blood volume.

Intravenously administered thiarnylal successfully reduced ICP following failure of SNP. Barbiturates reduce acute intraoperative ICP elevation.⁷ This reduction may be secondary to decreased cerebral blood flow and possibly represents a direct vasoconstrictor effect on cerebral blood vessels.⁸

In the present case, if barbiturates had failed to reduce ICP, trimethaphan would have been considered. In neurosurgical patients undergoing induced hypotension, where trimethaphan was compared to SNP, trimethaphan caused no significant increase in ICP, although two individual patients had moderate (9.3 mmHg and 5.7 mmHg) increases in ICP.⁴ Stoyka *et al.*⁶ showed, in the dog, that trimethaphan-induced hypotension caused a decrease in CBF with no apparent change in cerebral vascular resistance; however, when cerebral perfusion pressure (CPP) fell below 50 mmHg there was evidence of borderline brain hypoxia. It was of interest that there was no evidence of cerebral ischemia in dogs receiving SNP with CPP equal to 30 mmHg. Increases in CVP have been reported with clamping of the thoracic aorta.⁹ These changes may lead to increases in cerebral blood volume without an increase in CBF. This did not occur in our case; therefore, the most likely cause of the acute elevation of ICP was an increase of CBF. In this setting and based on the available evidence, trimethaphan could be postulated as superior to SNP.

REFERENCES

1. Adams RW, Gronert GA, Sundt TM, et al: Halothane, hypocapnia, and cerebrospinal fluid pressure in neurosurgery. *ANESTHESIOLOGY* 37:510-517, 1972
2. Shapiro HM: Intracranial hypertension: therapeutic and anesthetic considerations. *ANESTHESIOLOGY* 43:445-470, 1975
3. Cottrell JE, Patel K, Turndorf H, et al: Intracranial pressure changes induced by sodium nitroprusside in patients with intracranial mass lesions. *J Neurosurg* 48:329-331, 1978
4. Turner JM, Powell D, Gibson RM, et al: Intracranial pressure changes in neurosurgical patients during hypotension induced with sodium nitroprusside or trimethaphan. *Br J Anaesth* 49:419-425, 1977
5. Ivankovich AD, Miletič DJ, Albrecht RF, et al: Sodium nitroprusside and cerebral blood flow in the anesthetized and unanesthetized goat. *ANESTHESIOLOGY* 44:21-26, 1976
6. Stoyka WW, Schutz H: The cerebral response to sodium nitroprusside and trimethaphan controlled hypotension. *Can Anaesth Soc J* 22:275-283, 1975
7. Shapiro HM, Galindo A, Wyte SR, et al: Rapid intraoperative reduction of intracranial pressure with thiopentone. *Br J Anaesth* 45:1057-1062, 1973
8. Smith AL: Barbiturate protection in cerebral hypoxia. *ANESTHESIOLOGY* 47:285-293, 1977
9. Kouchoukos NT, Lell WA, Karp RB, et al: Hemodynamic effects of aortic clamping and decompression with a temporary shunt for resection of the descending thoracic aorta. *Surgery* 85:25-30, 1979.

‡ Murphy FL, Kennell EM, Johnstone RE, et al: The effects of enflurane, isoflurane and halothane on cerebral blood flow and metabolism in man. (Abstract) American Society of Anesthesiologists' annual meeting, October 14, 1974.