sensory information in the spinal cord (Kitahata and Collins, 1981), requiring high local concentrations of the drug. A recent editorial in this journal (Hall, 1980) noted that very high doses of fentanyl given i.v. can prevent several of the hormonal and metabolic changes during surgery. The present observations point to the possibility that at least part of this action may be a result of spinal mechanisms; the elucidation of this problem requires further study.

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REFERENCES

Dirksen, R., and Nijhuis, G. M. M. (1980). Epidural opiate and perioperative analgesia. *Acta Anaesthesiol. Scand.*, **24**, 367.
Furness, J. B., and Costa, M. (1974). The adrenergic innervation of the gastrointestinal tract. *Rev. Physiol.*, **69**, 1.
Hall, G. M. (1980). Fentanyl and the metabolic response to surgery. *Br. J. Anaesth.*, **52**, 561.
Kitahata, L. M., and Collins, J. G. (1981). Spinal action of narcotic analgesics. *Anesthesiology*, **54**, 153.

HYPERBARIC NITROUS OXIDE AND MALIGNANT HYPERPYREXIA

Sir,—Nitrous oxide has been suggested as a weak trigger in malignant hyperpyrexia (MH) (Ellis, Clarke and Appleyard, 1974), implying that the use of greater concentrations of nitrous oxide might lead to MH episodes in susceptible patients. If true, this might explain episodes of MH that occurred in a patient who was anaesthetized with barbiturates, opiates and nitrous oxide (Fitzgibbons, 1981).

We examined the triggering action of nitrous oxide in a cylindrical hyperbaric chamber (90 cm long and 44 cm diameter) in six Poland China swine 20–40 kg in weight. Three pigs were susceptible to MH (halothane challenge), two were not (halothane–suxamethonium challenge) and one was "in-between": Pa_{CO_2} increased to 8.7 kPa and BE decreased to −12 after halothane and suxamethonium; however these changes occurred slowly and then gradually reverted to normal without therapy (Gronert, 1979). MAC for nitrous oxide in swine is likely to be between 133 and 213 kPa (Quasha, Eger and Tinker, 1980).

The pigs received atropine 0.4 mg i.v. and were anaesthetized with thiopentone 15–20 mg kg^{-1}; the trachea was intubated and the animals breathed 70% nitrous oxide in oxygen while an arterial cannular was inserted. The pig and the transducer were put into the chamber and depth of anaesthesia was monitored visually by the pattern and frequency of spontaneous ventilation. Ventilation became slower and more regular as anaesthesia deepened; eventually ventilation became uneven and jerky, but Pa_{CO_2} remained less than 7.6 kPa even to the point of apnoea. Oesophageal temperature was monitored by thermistor. Arterial pressure, heart rate, and blood-gas tensions were monitored via the arterial catheter; samples were withdrawn via a stopcock in the wall of the chamber. A 12-litre flow of nitrous oxide and oxygen was sufficient to prevent carbon dioxide accumulation within the chamber, and gas flows were adjusted to maintain chamber Po_2 (Beckman paramagnetic oxygen analyser) at 27–73 kPa with arterial Po_2 ranging from 11.2 to 44 kPa.

All pigs responded similarly to hyperbaric nitrous oxide. Arterial pressure and heart rate were stable, and temperature decreased slowly. Exposure times are shown in table I.

TABLE I. *Exposure time (min) to hyperbaric nitrous oxide*

	Chamber pressure (kPa)			
	147–159	160–172	173–187	Total
MH pigs (3)	5	15	75	95
	10	70	—	80
	62	—	—	62
Normal pigs (2)	13	45	15	73
	8	47	—	55
In-between pig (1)	23	63	—	86

No episodes of MH occurred, as shown by normal values for Pa_{CO_2}, base excess, muscle tone, and temperature. Two MH pigs and one normal pig became apnoeic in the chamber, at respectively P_{N_2O} 193, 165, and 90 kPa (the last episode occurred during decompression). Apnoea was treated by decompression within 3–4 min and immediate hyperventilation after the pig was removed from the chamber. One of the apnoeic pigs (90 kPa) died, with signs of decompression sickness, "the bends."

We presume that these exposures were of sufficient length and depth to exclude anaesthetic concentrations of nitrous oxide as a trigger in porcine MH.

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REFERENCES

Ellis, F. R., Clarke, I. M. C., and Appleyard, T. N. (1974). Malignant hyperpyrexia induced by nitrous oxide and treated with dexamethasone. *Br. Med. J.*, **4**, 270.
Fitzgibbons, D. C. (1981). Malignant hyperthermia following preoperative oral administration of dantrolene. *Anesthesiology*, **54**, 73.
Gronert, G. A. (1979). Muscle contractures and adenosine triphosphate depletion in porcine malignant hyperthermia. *Anesth. Analg. (Cleve.)*, **58**, 367.
Quasha, A. L., Eger, E. I. II, and Tinker, J. H. (1980). Determination and applications of MAC. *Anesthesiology*, **53**, 315.