postulated⁶ may conceivably be involved in withdrawal responses.

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REFERENCES

- 1 Manson HJ, Dyke G, Melling J, Gough M. The effect of naloxone and morphine on convulsions in mice following withdrawal from nitrous oxide. *Can Anaesth Soc J* 1983; 30: 28–31.
- 2 Gillman MA. Nitrous oxide at analgesic concentrations – An opiate agonist: Further evidence. *Anesth Analg* 1982; 61: 394–5.
- 3 Ahmed MS, Byrne WL. Opiate receptor binding studies: Influence of a reversible sulfhydryl agent. In: Way EL, ed. *Endogenous and exogenous opiate agonists and antagonists*. New York: Pergamon. 1980: 51–4.
- 4 Daras C, Cantrill RC, Gillman MA. (³H) – Naloxone Displacement: Evidence for nitrous oxide as opioid receptor agonist. *Europ J Pharmacol* (in press).
- 5 Way WL, Hosobuchi Y, Eger EL, Johnson BH. Anesthesia does not increase cerebrospinal fluid opiate peptides in Humans. *Anesth Analg* 1982; 61: 223.
- 6 Gillman MA, Kimmel I, Lichtigfeld FJ. The dual-system hypothesis of pain perception. *Neurological Res* 1981; 3: 317–27.
- 7 Gillman MA, Lichtigfeld FJ. The effect of nitrous oxide and naloxone on orgasm in human females – a preliminary report. *J Sex Research* (in press).
- 8 Eisenberg RM. Further studies on the acute dependence produced by morphine in opiate naive rats. *Life Sciences* 1982; 31: 1531–40.

REPLY

The explanation put forward by Drs Gillman and Lichtigfeld for the effects we observed seems a reasonable alternative to our tentative hypothesis; there remains a need for further experimental work before the underlying mechanisms can be clearly delineated.

The whole area of interaction between anaesthetic agents and endogenous opiate systems is of considerable importance, both clinically and from a basic pharmacological point of view.

Demonstration of the mode of interaction between an inert gas and a control system within the central nervous system would be of great importance to our understanding of anaesthetic mechanisms. Perhaps more direct biochemical analysis will, in time, shed light on the problem.

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Anaesthetic considerations in the Prader-Willi syndrome

To the Editor:

We read with interest the article by Yamashita *et al.*¹ concerning the anaesthetic management of children with the Prader-Willi syndrome.

We would like to add our experience with three anaesthetics involving two children with the Prader-Willi syndrome.

The first was a six-year-old male child who underwent an uneventful nitrous oxide-oxygen-halothane anaesthetic, following an oral intubation using intravenous succinylcholine, for repair of bilateral undescended testes.

The second child was a three-year-old male scheduled for bilateral orchiopexy. Following an inhalation induction with nitrous oxide-oxygen-halothane, he was reportedly a difficult intubation, with the use of intravenous succinylcholine. He was then maintained on nitrous oxide-oxygen-halothane and curare for relaxation. His initial rectal temperature was 98.6° F but increased to 101.8° F after 45 minutes. An arterial blood gas revealed respiratory acidosis. The patient was actively cooled and the case was immediately terminated. No dantrolene sodium was given. The neuromuscular blockade was reversed using atropine and neostigmine, without difficulty. The postoperative temperature remained below 100° F. The CPK rose to 240 (normal 8–132) eight hours following anaesthesia.