postulated⁶ may conceivably be involved in withdrawal responses.

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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.

When the patient was anaesthetized by us for repair of the other undescended testis, pentothal induction was followed by use of pancuronium for intubation. We found no anatomical problems with the airway and our intubation was not difficult. Nitrous oxide, oxygen and fentanyl were used for maintenance for the one hour and forty-five minute procedure. The rectal temperature remained 98.6° F throughout the procedure and the postoperative period. The neuromuscular blockade was reversed using atropine and neostigmine.

We report these two children and three anaesthetic experiences since we believe that as experience is gained and our understanding increases with anaesthetizing children with the Prader-Willi syndrome: (1) Although Palmer and Atlee² suggest that problems may occur with the use of non-depolarizing relaxants, a narcotic-relaxant technique can be utilized safely; (2) despite the presence of hypotonia, neuromuscular blockade with good reversal is possible; (3) if the intraoperative temperature acutely rises, steps must be taken to avert a malignant hyperthermic reaction; and (4) thought should be given as to whether muscle biopsies should be done to help define those patients at risk for malignant hyperthermia.

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