considerable pain. This subsequently requires management possibly resulting in greater overall opiate dose. Similarly, opiates play a useful role in maintaining haemodynamic stability during induction of anaesthesia.^{3,4} Under these circumstances we consider it more appropriate to use opiates to reduce the high incidence of postoperative pain or haemodynamic lability and then take all available measures to help reduce the thirty per cent possibility of vomiting.

M.G.A. Palazzo MB CHB MRCP FFARCS
L. Strunin MD FFARCS FRCP(C)
Department of Anaesthesia,
Foothills Hospital at the University of Calgary,
Calgary, Alberta, T2N 2T9

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Nitrous oxide and its opioid emetic properties

To the Editor:

In their interesting review on emesis and anaesthesia, Palazzo and Strunin, state that nitrous oxide (N₂O) is well known as an emetic agent.¹ Although these authors mention that N₂O causes vomiting by both peripheral and central effects, their discussion of peripheral effects is quite detailed, whilst that of the central effects of N₂O is limited. In this respect, they only cite one author who claims that N₂O does not stimulate the vomiting centre, but give no further details.

Nitrous oxide has been shown in many studies both at the receptor level² and in vivo both in animals and man³ (for further references see ref. 3),

to interact with the endogenous opioid system and it is thus not surprising that it shares with other opiate and opioid substances substantial emetic properties. It is therefore possible to suggest that whilst the peripheral effects of N₂O may be of importance in producing emesis that its central effects are no less so.

Dr. M.A. Gillman
South African Brain Research Institute
Johannesburg 2000, South Africa

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Muscarinic cardiac effects of pyridostigmine

To the Editor:

I should like to thank Drs. Donati, Bevan and Bevan for their interesting review of neuromuscular blocking drugs.¹ I was, however, disappointed to read that the slower action of pyridostigmine makes the drug unsuitable for antagonism of neuromuscular blockade, since it happens to be my favourite drug for this purpose.

Much has been made of Fogdall and Miller's report on pyridostigmine,² in which its muscarinic cardiac effects were stated to be no different from those of neostigmine. However, I submit that their data support an alternative conclusion. Although the degree of bradycardia induced by the two drugs appeared similar, in the presence of pancuronium or d-tubocurarine blockade, its onset was much more rapid with neostigmine. This difference was most striking in the subjects receiving the smallest dose of atropine, 0.3 mg, in whom maximum mean

depression of heart rate occurred within 4 minutes after neostigmine 2.5 mg but took 8 minutes with pyridostigmine 14.5 mg. In addition, many today would consider 10 to 12 mg of pyridostigmine equipotent with neostigmine 2.5 mg.

If, as suggested by Donati *et al.*, it is desirable to add as little anticholinergic drug as possible when administering an anticholinesterase, then in my experience pyridostigmine wins hands down over neostigmine and edrophonium. I have found it quite satisfactory to give the drug alone at an appropriate time (usually when fascial closure is nearly complete), closely observing the heart rate over the ensuing 10 minutes. In the event that the rate threatens to drop to an unacceptable nadir, atropine can be given. Most often, bradycardia is mild and no atropine is needed.

The relatively prolonged onset time of pyridostigmine (often some 10–15 minutes) poses no practical problem, since early experience quickly taught me to give the drug at a suitably advanced time in anticipation of final closure.

Christopher P. Bates MD FRCP(C)
Department of Anaesthesia
UBC Health Sciences Centre Hospital
The University of British Columbia
Vancouver, British Columbia, V6T 2B5

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