et al.—will have been evacuated from within the stomach in a couple of hours. The remainder should cause no harm, even if general anaesthesia is urgently required, provided that the correct procedure of induction is followed. I have never fully understood the current obsession with the comparison or relatively small volumes of gastric residue. I will accept that the presence of an inordinate volume (say half a litre or more) could pose the threat of passive regurgitation, or even of vomiting, before the application of cricoid pressure, or even that a small quotient of that quantity would seep past the site of oesophageal obstruction. Surely, though, the hazard posed by the presence of 100 ml of gastric contents is in practice no greater than that posed by 15 ml, provided that the acidity of the material is outside the critical range.

Finally, may I remind your readers—although this might appear to contradict some of my own tenets of prophylaxis—that it is only since we began to starve our labouring patients, and those coming to elective surgery, that we have experienced the epidemic of acid-aspiration syndrome.

J. S. CRAWFORD

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REFERENCES


ANTAGONISM OF NEUROMUSCULAR BLOCK

Sir,—In the discussion between Foldes (1981) and Payne and Hughes (1981) regarding the way atropine and neostigmine should be administered to reverse neuromuscular block, Clarke and Mirakhur (1982) concluded that both drugs are better administered together unless there is pre-existing bradycardia of sufficient severity to warrant prior administration of the anticholinergic drug.

It will be noted that the only consideration is the heart rate, both in this correspondence as well as in the majority of, if not all, research and review articles. However, this is not the only reason why anticholinergics are given. A major reason is the control of secretions. This effect is better observed if the anticholinergic is given before neostigmine. Atropine also has bronchodilator effects and, in fact, was first used for this reason in 1836. Anticholinergics are also preferred to be used by some before neostigmine in bowel surgery (Mirakhur et al., 1978).

As regards the effects on the heart rate, how does one explain the discrepancy between minimal effects as demonstrated by Payne and Hughes (mean heart rate 56.8 beat min⁻¹ ± 4.9 (SEM) increasing to 78.1 beat min⁻¹ ± 4.7 after atropine) and gross tachycardia described by others. The answer must lie in the type of anaesthetic administered and consideration of the mode of action of anticholinergic drugs.

Taking atropine into consideration, it does not cause an increase in heart rate directly. It blocks the parasympathetic nerves (vagus) to the heart and releases the sympathetic drive. The heart then responds to this drive: the greater the sympathetic stimulation at that moment, the greater the heart rate. In fact the parasympathetic can be considered as the governor and the sympathetics the motor of the heart. The route and dose of atropine, in fact, play a lesser role. The major factor is the amount of sympathetic stimulation (nervous and humoral) or stress that is present. This in turn is dependent on the type of anaesthetic and premedication the patient has received.

I believe that adequate premedication and anaesthesia will result in minimal stress and sympathetic stimulation. Hence, if atropine is used before neostigmine in such a patient, there will be minimal change in heart rate as demonstrated by Hughes and Payne.

In fact, the heart rate after atropine provides a simple and direct model for assessing stress in a patient. This whole field merits further study and research, including the problem of respiratory secretions and postoperative chest complications.

I therefore feel that the conclusions of Clarke and Mirakhur (1982), and Rosner, Kepes and Foldes (1971) only relate to a part of the problem. The effects on the patient as a whole must be considered. Also, one should not seek umbrage in the fact that the majority is right. History has proved this wrong many a time.

F. S. REDDY

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REFERENCES


Sir,—Dr Reddy rightly points out that the purpose of administering an anticholinergic drug with neostigmine is not only the consideration of effects on heart rate, but also on secretions and bowel activity. However, these need to be considered in their proper perspective and importance.

The main reason why consideration of cardiovascular effects has assumed such importance during antagonism of neuromuscular block is that these effects can have immediate and disastrous results, as was demonstrated by the early case reports of Macintosh (1949), Clutton-Brock (1949) and Pooler (1957).

Dr Reddy implies that adequate premedication and anaesthesia attenuate the effects of atropine, but does not specify any particular agents or techniques. Atropine administration is associated not only with greater increases in heart rate in anaesthetized subjects (Jones, Deutsch and Turndorf, 1961; Eger, 1962) but also with a higher incidence of arrhythmias in those anaesthetized with halothane (Farman, 1967; El-kard and Andersen, 1977; Mirakhur and Jones, 1982). The increase in heart rate of 37.5% in the study of Payne and Hughes (1981) would be considered significant, particularly in those with cardiovascular disease. It would have been reduced by simultaneous administration of atropine and neostigmine (Ovassopian, 1969; Rosner, Kepes and Foldes, 1971; Mirakhur et al., 1981), or better still, by...
glycopyrrolate and neostigmine (Mirakhur and Dundee, 1983). Many well controlled studies have now established that neostigmine and the anticholinergic give greater cardiovascular stability when administered together.

Regarding the use of atropine for assessment of stress in anaesthetized patients, it would appear to be more relevant to measure this in terms of hormonal changes rather than the response of the heart rate to atropine.

Administration of an atropine-neostigmine mixture does not produce detectable bronchospasm in healthy patients (Hammond, Wright and Sale, 1983) although asthmatics may be at some risk. There is also only anecdotal evidence to show that the control of secretions is better when atropine is given before neostigmine, unless it is done perhaps 15–20 min before. It is unlikely that atropine administered 3–5 min before neostigmine makes any difference in secretions. Mirakhur, Jones and Dundee (1981) were unable to detect any difference in the incidence of unacceptable secretions between patients given atropine or glycopyrrolate before or with neostigmine.

The effects of atropine and neostigmine on bowel activity are perhaps more complex and equivocal. While Bell and Lewis (1968), in a retrospective study, showed that barium enema demonstrated a 36% incidence of anastomotic leak following neostigmine in comparison with only 4% in those not given neostigmine, Wilkins and his colleagues (1970) showed an increase in bowel activity in only 20% of patients irrespective of whether atropine was given before or mixed with neostigmine, and they showed that this increase in bowel activity could be prevented by halothane anaesthesia. There are few reported cases of leakage from intestinal anastomosis in recent years, either because it is not as easy to demonstrate as heart rate effects, or because it is not a real problem. In high risk patients such as those having surgery for diverticular disease, it may be better to avoid neostigmine altogether or else to administer it under halothane anaesthesia.

In conclusion, the effects on heart rate of neostigmine and atropine are important and are minimized by administering the two drugs together. Heart rates are even more stable when neostigmine is administered mixed with glycopyrrolate (Mirakhur and Dundee, 1983). Dr Reddy should now perhaps follow the majority in this issue.

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REFERENCES

CLOSED CIRCUIT ANAESTHESIA
Sir,—It was with pleasure and surprise that we read the paper by Beatty and colleagues (1982). Pleasure, to read a paper devoted to closed circuit anaesthesia, but surprise to discover the nature of the contents. In the introduction to their paper, Beatty and colleagues referred to an earlier publication by ourselves (Bushman et al., 1977) on closed circuit anaesthesia. They state as one premise for their current work the fact that one of the breathing systems described by us had an unexpectedly high resistance to breathing. It is only necessary to read through this same paper to find that no such problem was ever experienced or described.

Beatty and colleagues then proceeded to describe a demand valve with a bellows using an internal spring to overcome this high inspiratory resistance. While in no way questioning the validity of the results of that paper, we feel that the motivation for the work was based on false premise. Although the laboratory assessment of their system is well presented, their work is very largely a repeat of our earlier work on an identical topic.

In conclusion, although it is indeed encouraging to find an increasing interest in closed circuit anaesthesia, it is somewhat disappointing that little or no scientific progress appears to have been made in the intervening five years.

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REFERENCES