CORRESPONDENCE 687

# Amiodarone and anaesthesia: concurrent therapy with ACE inhibitors – an additional cause for concern?

### To the Editor:

We were interested to read the article by Chassard et al.<sup>1</sup> and the accompanying editorial on "Amiodarone and Anaesthesia" in the journal.<sup>2</sup>

We wish to report two further cases of serious hypotension requiring massive doses of alpha-agonists which occurred in two consecutive patients receiving amiodarone undergoing cardiopulmonary bypass (CPB). These patients had taken oral amiodarone 200 mg daily for more than six months. Their serum amiodarone concentrations were in the therapeutic range and are, in that respect, typical of previous reported cases.<sup>3</sup>

Patient JF (51 yr) underwent combined heart and kidney transplantation receiving an anaesthetic based on midazolam, trichloroethylene and papavertum. Progress was uneventful until institution of CPB when mean arterial pressure (MAP) decreased from 60 to 25 mmHg. This hypotension was resistant to treatment with metaraminol (total 6 mg) and phenylephrine (total 10 mg). Weaning from CPB was only achieved after starting a noradrenaline infusion. The cardiac output was measured at 8 L·min<sup>-1</sup> and systemic vascular resistance at 270 dyne·sec·cm<sup>-5</sup>; noradrenaline infusion was increased to a maximum of 0.5 μg·kg<sup>-1</sup>·min<sup>-1</sup> to maintain adequate pressures coming off bypass.

Patient SP (44 yr) underwent coronary artery bypass grafting with a midazolam, fentanyl and propofol technique. His early operative progress was very similar to JF. He maintained a MAP of 60 with dopamine 4  $\mu g \cdot kg^{-1} \cdot min^{-1}$ , but this decreased to 25 mmHg on institution of CPB. Metaraminol 2 mg was ineffective and a total of 12 mg was complicated by dysrhythmias and hypotension. This subject was also found to have a low systemic vascular resistance but cardiac output was also low and contributed to the hypotension. He was eventually weaned from CPB with an intra-aortic balloon pump, adrenaline 0.05  $\mu g \cdot kg^{-1} \cdot min^{-1}$  and noradrenaline at 0.015  $\mu g \cdot kg^{-1} \cdot min^{-1}$ 

We would like to bring to attention a further possible interaction. Both these patients had been taking an angiotensin converting enzyme inhibitor, enalapril 5 mg daily. We also noted that the second of Liberman's two patients was taking captopril in addition to amiodarone.<sup>3</sup> It is known that patients taking amiodarone have a state of non-competitive alpha and beta blockade. They, there-

fore, have difficulty compensating for vasodilatation.<sup>2</sup> We suspect that ACE inhibitors further compound this problem and plan to study this further.

It is difficult to justify the preoperative withdrawal of amiodarone; due to its long half life, this would have to be done so far in advance of surgery that the overall risk to the patient would be greatly increased. This argument may not apply to ACE inhibitors and perhaps we should consider preoperative withdrawal of these drugs in patients also taking amiodarone given their possible contributory effect to hypotension during cardiac surgery.

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- 2 Teasdale S, Downar E. Amiodarone and anaesthesia (Editorial). Can J Anaesth 1990; 37: 151-5.
- 3 Liberman BA, Teasdale SJ. Anaesthesia and amiodarone. Can Anaesth Soc J 1985; 32: 629-38.
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# M.O.U.T.H.S.

# To the Editor:

Despite the frequency of airway examination in anaesthetic practice, anaesthetists lack a coherent pattern of description of the airway. From examination of residents in both departmental and Royal College examinations and from review of anaesthetic records, we think a simple system of organization of information may be useful. Such a system is offered by the mnemonic, "MOUTHS". This acronym represents the components shown in the Table.

Previous authors have focussed on specific components of airway evaluation. "MOUTHS" includes all of the factors necessary for this task. We would suggest that the schematic diagram of the teeth and a copy of the Figure showing pharyngeal structures be incorporated into the

## TABLE

	Components	Descriptors	Assessment activities
M	Mandible	Length and subluxation	Measure mental-hyoid distance and anterior displacement of mandible
0	Opening	Ease, symmetry and range	Assess and measure mouth opening in cm
U	Uvula	Visibility	Assess pharyngeal structures and classify
T	Teeth	Dentition (Figure)	Assess for presence of loose teeth and dental appliances
Н	Head	Flexion, extension, rotation of head and cervical spine	Assess all ranges of movement
S	Silhouette	Upper body abnormalities, both anterior and posterior	Identify potential impact on control of airway <sup>2</sup> of large breasts, buffalo hump, kyphosis, etc.

	(Right)	(Left)	
			(Maxilla)
7,6,5,4	1,3,2,1	1,2,3,4,5,6,7	
7,6,5,4	1,3,2,1	1,2,3,4,5,6,7	
			(Mandible)

FIGURE Schematic representation of anterior view of dentition, with teeth numbered sequentially from midline. Indicate abnormalities as: A = Absent; B = Bridge; C = Caps; L = Loose.

anaesthetic record. These would act as an "aidememoire," help to ensure more complete documentation, and then be available for review of charts fro audit. We would welcome comments.

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- Samsoon GLT, Young JRB. Difficult tracheal intubation: a retrospective study. Anaesthesia 1987; 42: 487-90
- 2 Davies JM, Weeks S, Crone LA, Pavlin E. Difficult intubation in the parturient. Can J Anaesth 1989; 36: 668-74.

# Erratum

Re "Calcium-channel blockers and anaesthesia" in the January issue of Canadian Journal of Anaesthesia.

In Table I, the specificity for slow calcium channels of Group B is "++" (and not +++)

Durand P-G, Lehot J-J, Foëx P. Calcium-channel blockers and anaesthesia. Can J Anaesth 1991; 38: 75-89.