Initial concern about the impact of esmolol on succinylcholine was based upon the fact that, like succinylcholine, esmolol is metabolized by an esterase.[1] However, in contrast to rats and guinea pigs, the esterase responsible for esmolol metabolism in humans resides primarily in the cytosol of red blood cells and not in the plasma. Investigators have reported varied effects of esmolol on succinylcholine-induced block[1-3] ranging from no effect to as much as 50% prolongation. Barabas et al. identified no change in the ability to hydrolyze benzoylcholine in plasma obtained after subjects received an infusion of up to 1400 $\mu g \cdot kg^{-1}$.[1] Murthy et al. reported delay of 50% recovery of twitch height to 8.3 min in patients who received 1 $mg \cdot kg^{-1}$ of succinylcholine in the presence of 4400 $\mu g \cdot kg^{-1}$ of esmolol (vs 5.6 min in non-esmolol treated patients).[2]

It should be noted that, unless there is a marked decline in pseudocholinesterase activity, succinylcholine-induced-block typically is not prolonged in subjects with genotypically normal enzyme.[3] We believe that it is more likely that any prolongation would be a consequence of the esmolol-induced decrease of cardiac output. This delays succinylcholine redistribution, a vital feature of the rapid recovery from this relaxant.[4,5] In addition, the use of a beta antagonist may impact on catecholamine-induced changes in acetylcholine release and/or membrane reactivity.[6]

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REPLY (2)

Dr. O'Flaherty raises the possibility of an interaction between esmolol and succinylcholine leading to a prolonged duration of action when the drugs are co-administered since both drugs are metabolized by esterases. Succinylcholine is metabolized by plasma pseudocholinesterase[1] but this enzyme is devoid of activity against esmolol.[2] Esmolol is metabolized by an arylesterase found in red blood cell cytosol.[2] It is therefore unlikely that prolongation of effect for either agent could be ascribed to a competitive interaction for metabolic enzymes. Should prolongation of effect occur when these drugs are co-administered, it is likely that this is related not to a drug interaction but to a relative deficiency or abnormality of the corresponding agent-specific esterase.

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Breathlessness

To the Editor:

From antiquity, breathlessness has been considered a very serious complaint, representing a threat to life and an indication of the severity of any disorder associated with it.

In 1966, Dr. E.J. Moran Campbell and Dr. Jack B.L. Howell were able to bring together experts from many disciplines to discuss breathlessness. Their subsequently published book based upon the Manchester Breathlessness Symposium was a landmark in the evolution of our understanding of breathlessness and provided a stimulus for research in the succeeding years. The text and conference were funded by Boehringer Ingelheim of the United Kingdom. A new text is now available to assess how far this evolution has progressed.

This text entitled "Breathlessness: The Campbell Symposium" based upon a Continuing Medical Education programme held in Hamilton, Ontario, Canada from May 16 to May 19, 1991 is now available free of charge. The text is edited by Norman L. Jones, MD, FRCPC (London), FRCP(C) and Kieran J. Killian, MBBS, FRCP(I), FRCPC. Both physicians are associated with the Faculty of Health Sciences, McMaster University, Hamilton, Ontario, Canada. The text was published by Decker Periodicals Inc. of Hamilton, Ontario, Canada.

Funding for the meeting and the publication was provided by Boehringer Ingelheim (Canada) Ltd., a pharmaceutical company which is dedicated to Respiratory Therapy. The three-day conference which was organized by McMaster University, Faculty of Health Sciences, Hamilton, Ontario, Canada was held to mark the retirement of Dr. E.J. Moran Campbell and the 25th anniversary

of the Manchester Breathlessness Symposium. The text may be obtained by writing, telephone or Fax to:

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