Anticholinesterase Drugs and the Transplanted Heart

To the Editor—I perused with interest the report by Dr. Sawasdiwipachai et al.1 detailing bradycardia followed by cardiac arrest in an infant cardiac transplant recipient after intravenous administration of neostigmine–glycopyrrolate. The authors speculate that rejection of the conducting system may have contributed to this response that is considered to be unusual because of the low likelihood of parasympathetic reinnervation. Asystole preceded by bradycardia after neostigmine–glycopyrrolate administration has been described in three adult transplant patients.2,3 and neostigmine has been shown to produce an atropine-sensitive dose-dependent bradycardia in recent (<6 months) and remote (>6 months) adult cardiac transplants.4 Contrary to the authors’ assertion, rejection in these patients was neither confirmed nor refuted. The observation that remote cardiac transplants may demonstrate greater bradycardic responses to neostigmine compared with recent transplants may be explained by weak, variable parasympathetic reinnervation and/or by a denervation supersensitivity to the cholinergic agonist effect of neostigmine.5 The influence of rejection on these responses is an intriguing confounding variable that remains to be determined. Regardless of the underlying mechanisms mediating the bradycardia in cardiac transplant patients after anticholinesterase administration, it is clear that caution should be exercised when reversing neuromuscular block even when a muscarinic antagonist is coadministered with the anticholinesterase. To avoid a potentially catastrophic response to neostigmine, the authors suggest avoidance of neuromuscular block if possible, or use of short-acting drugs if paralysis is required. They speculate that this problem may be circumvented by the use of new reversal agents such as sugammadex. Another possibility not considered is reversal of neuromuscular block with edrophonium (and of course a muscarinic antagonist!). While edrophonium, too, produces bradycardia in cardiac transplant recipients, the decrease in heart rate is smaller in magnitude and much more consistent compared with neostigmine.6

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References


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