CORRESPONDENCE

Anesthesiology
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The Precipitation of Rocuronium in a Needleless Intravenous Injection Adaptor

To the Editor:—The mixture of sodium thiopental and rocuronium bromide (Zemuron) results in the immediate formation of a white precipitate. This can be avoided during anesthetic induction by adequately flushing the intravenous line after the injection of sodium thiopental, before the injection of rocuronium bromide. This conventional wisdom holds true for standard needle intravenous infusion systems but is confounded by newer adaptations that render the infusion system needleless.

I have observed on several occasions the formation of such a precipitate after sequential intravenous injection of sodium thiopental/rocuronium bromide through a needleless Y-injection site adaptor (Access pin with Safesite Valve by B. Braun Medical Inc.). It is apparent that a small aliquot of the initial injected drug, sodium thiopental, remains trapped in the void space of the needleless system adaptor. This small volume is not affected by subsequent flushing of the intravenous line, because the infusate never reaches the void space. The injection of rocuronium bromide with its associated turbulence leads to the mixing of the two drugs within the needleless adaptor and causes the formation of the precipitate.

This reaction has resulted in the complete occlusion of two intravenous lines during anesthetic induction. Obviously, this is an inopportune time to be pressed to start a new intravenous infusion. With regard to the new needleless systems—let the buyer beware.

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Thoracic Sympathetic Blockade Does Not Imply Vagal Dominance

To the Editor:—We would like to congratulate Kamibayashi *et al.* on their interesting paper.[1] However, we wish to offer an alternative view to one of their conclusions. The authors stated, "Sympathetic activity to the heart in epidurally anesthetized animals was significantly attenuated, while parasympathetic activity was not affected. Therefore, the activity in the parasympathetic nerve may be relatively dominant to sympathetic tone after epidural treatment, and this situation is similar to vagal nerve stimulation." Although it is intuitive to suggest that, when the sympathetic efferent nerves are blocked, a relative vagal dominance would exist, clinically this is not the case. Rather, in patients with a cardiac sympathectomy after high spinal block (T4–C7),[2,3] spectral analysis of heart rate variability results in a loss of both sympathetic and vagal components. This indicates a state of reduced sympathetic and vagal outflow[4] and probably results from sympathetic afferent blockade to central neural centers.[5] Therefore, the vagal outflow, which was not directly blocked by the anesthetic, was not sufficient to maintain normal heart rate variability.[6] In their dog model of thoracic sympathectomy,[1] the baroreceptor pressor response after epinephrine probably created a vagal dominant state, but we submit that this state would not exist in the absence of systemic hypertension. Therefore, we believe that sympathectomy of the heart alone does not necessarily result in a state of vagal dominance but that vagal dominance exists only after sympathetic blockade in the presence of vagal stimulation.

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In Reply:—We appreciate the useful suggestion to our study expressed by Introna *et al.* Based on their study using spectral analysis of heart rate variability, which showed that both sympathetic and parasympathetic activity reduced after cardiac sympathectomy with spinal block,[1] we agree that vagal dominance after epidural anesthesia might not exist, although we did not evaluate sympathovagal balance after thoracic epidural blockade.[2] In our dysrhythmogenic experiments,[2] bilateral vagotomy did not affect the dysrhythmogenic threshold significantly in dogs without thoracic epidural blockade, suggesting that vagal stimulation alone induced by baroreceptor pressor response after epinephrine infusion was not enough to affect halothane-epinephrine dysrhythmias. However, vagal stimulation played a significant role in preventing the dysrhythmias in dogs with thoracic sympathetic blockade, although vagal outflow may have been reduced through the central nervous system.[3] These observations suggested that sympathetic activity as well as sympathovagal balance might be important in the myocardial sensitization by halothane.

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Parkinsonian Signs May Be Related to Bupivacaine Excess

To the Editor:—Muravchick and Smith described Parkinsonian signs in a patient after general anesthesia.[1] Of interest, bupivacaine was used, both for intercostal blockade as well as for wound infiltration in a total dose of 225 mg (45 ml of 0.5%). This is the maximum dose that can be used.[2] Indeed, Wood[3] considers 2 mg/kg the highest safe limit, which, in the reported case (80 kg), would have been 160 mg bupivacaine. The rate of injection and rapidity with which blood concentrations of bupivacaine are achieved can alter its toxicity signs. The use of epinephrine could have delayed the absorption of bupivacaine so that a toxic concentration would have been reached