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Diltiazem and vecuronium: neuromuscular and cardiovascular effects

It is well known that calcium channel blockers augment the action of neuromuscular relaxants.[1-3] Additionally, calcium channel blockers attenuate increases in blood pressure (BP) and heart rate (HR) at the tracheal intubation.[4,5] However, no previous studies investigated both the calcium channel blocker-induced effect on the time to the onset of neuromuscular blockade and its attenuating effect on undesirable increases of BP and HR at the tracheal intubation. Forty-five adult patients were studied. Diltiazem 0.1 mg·kg^{-1}, 0.2 mg·kg^{-1}, and normal saline were administered in group 1 (n=15), group 2 (n=15), and control group (n=15), and thereafter, 5 mg·kg^{-1} thiopentone and 0.2 mg·kg^{-1} vecuronium were administered to facilitate tracheal intubation. Anaesthesia was maintained with nitrous oxide 66 %, oxygen 33 %, and sevoflurane 2.0 %.

Times to onset of neuromuscular blockade in groups 1 2, and control were 86.4 ± 20.2, 84.7 ± 25.9, and 115.7 ± 24.4 sec, respectively (mean ± SD, $P < 0.05$ for group 1 and group 2 *vs* control group). During induction of anaesthesia, changes in SBP and DBP in group 2 were less than those in groups 1 and control. However, changes in the HR did not differ (Figure).

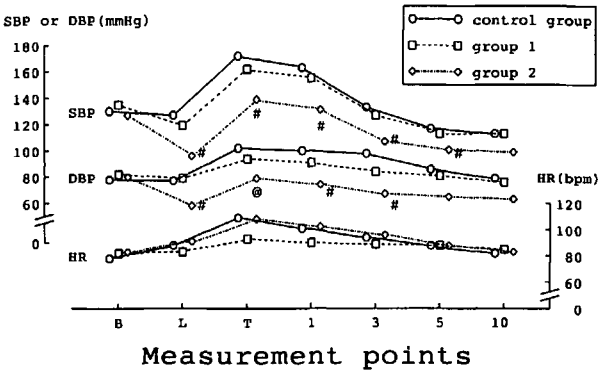

FIGURE Changes in systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) in the group 1 (□), group 2 (◇), and control group (○). Measurement points: B, baseline value; L, just before the start of laryngoscopy; T, tracheal intubation; 1, 1 min after tracheal intubation; 3, 3 min after tracheal intubation; 5, 5 min after tracheal intubation; 10, 10 min after tracheal intubation. Values are mean. SD bars are omitted for clarity. # $P < 0.05$ as compared to group 1 and control group; @ $P < 0.05$ as compared to control group.

Administration of diltiazem prior to tracheal intubation reduces time to onset of vecuronium-induced neuromuscular blockade, and attenuates changes in blood pressure caused by tracheal intubation.

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Using continuous pressure monitoring to aid central vein cannulation

Morbidity associated with central vein cannulation includes accidental arterial puncture which can result in fatality.[1] If the artery is punctured, then the pulsatile flow of blood, its colour and blood gas analysis will usually provide adequate evidence of misplacement of the introducer needle. If, however, the patient is severely shocked, haemodiluted and well oxygenated and a blood gas analyser is not readily available then the diagnosis of arterial puncture may be difficult to make. Measurement of the blood pressure and its

waveform in the punctured vessel should determine whether it is arterial or venous. The attachment of a pressure transducing line to the introducer needle will allow the pressure of the vessel to be measured before proceeding with dilatation.

This can be achieved by attaching a three-way tap between the syringe and introducer needle with the pressure transducing line on the side arm (Figure). Once the vessel is cannulated the pressure can be compared with that measured through the arterial cannula. If the pressure is venous then the guide wire can be passed through the tap after removal of the syringe or the tap can be removed altogether at this point.

The advantage of transducing the introducer needle at the time of insertion in a hypotensive patient is that, if an artery is accidentally punctured, it may be detected immediately before the puncture site is dilated with potentially fatal consequences.

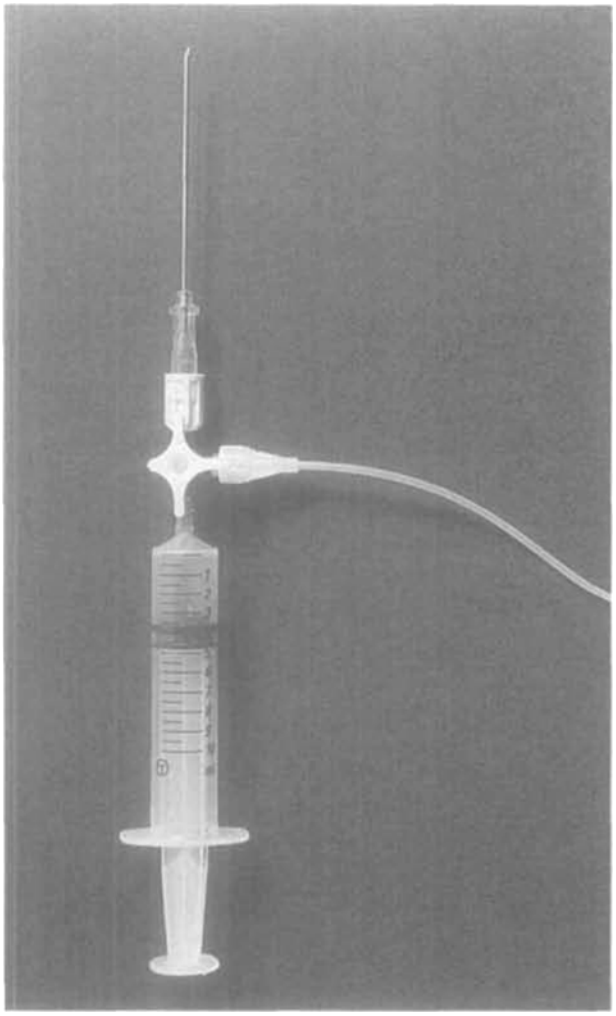

FIGURE Pressure moniter line attached to guide needle with a three-way tap for central vein cannulation.

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Intravenous anaesthesia and leukocytes

We would like to comment on Dr. Krumholz's paper "The influence of intravenous anaesthetics on polymorphonuclear leukocyte function" (Can J Anaesth 40: 770–4, 1993).[1]

We examined the effects of intravenous sedatives such as diazepam and flunitrazepam and published the report in the *British Journal of Anaesthesia* in 1991,[2] which was cited in the review by Salo[3] in 1992. As demonstrated in our paper, flunitrazepam powder is not water soluble and the depressive effects of the commercially available flunitrazepam injection on neutrophil function including superoxide production are mainly due to additives such as propylene glycol, ethanol or benzyl alcohol. In our study, we further investigated the effects of these additives on the O_2 producing system in neutrophil. We recommend that any solution for intravenous injection should be examined with caution on the effects of additives.

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