aged 68 ± 5.3 yr studied before abdominal surgery. Patients were selected who had a history of chronic stable angina pectoris grade II NYHA; sinus rhythm; electrocardiographic evidence of myocardial ischemia (no pathologic Q waves, abnormal ST segments and J point, and ischemic alterations of T-waves); and positive myocardial perfusion scintigraphy. No patients had a previous documented myocardial infarction. One-half of the patients were receiving nitrate therapy and the other half were receiving beta-blockade therapy (propranolol: 60 mg three times daily). All patients were given their last dose within 3 h of operation. Control values for heart rate (HR), systemic blood pressure (SAP), pulmonary capillary wedge pressure (PCWP), and cardiac output (CO) were obtained. Continuous recording of lead II and V_5 of the ECG was obtained during the study. Cardiovascular variables were recorded at 2, 5, and 10 min following pancuronium administration (0.08 mg/kg iv). No manipulations were undertaken while the study was performed. Statistical analysis was performed by analysis of variance. Paired *t* tests were used to assess intragroup differences. The absolute and relative increases in HR, mean SAP (SMAP), and CO in the group receiving beta-blockers and in the group not receiving beta-blockers were compared by unpaired *t* test.

As seen in table 1, significant increases in HR, SMAP, and CO were observed at 2, 5, and 10 min in patients not receiving beta-blockers but only at 2 and 5 min in patients receiving beta-blockers. Furthermore, the former group exhibited significantly greater relative and absolute increases in HR ($P < 0.001$), in SMAP ($P < 0.01$), and in CO ($P < 0.001$) than did patients receiving beta-blockers.

Our hemodynamic data indicate the possibility of either a direct or indirect beta-adrenergic effect to explain the sustained positive chronotopic action of pancuronium. Moreover, Conway *et al.*,[3] demonstrated in the isolated perfused rat heart that inhibition of neuronal uptake of noradrenaline played a role in the etiology of the chronotropic actions of pancuronium. Muscarinic receptors and adrenergic receptors produce opposite effects on the heart, but atropine may not increase heart rate in the presence of beta-adrenergic blockade, and may sensitize beta receptor to beta stimulation.[4] Finally, the relation, established by Miller *et al.*,[5] between heart rate before pancuronium and maximum increase in heart rate following pancuronium is suppressed by beta-blockers.

We conclude that beta-adrenergic receptor blockade does attenuate the cardiovascular effects of pancuronium.

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Pulmonary Artery Catheter Malfunction?

To the Editor:—In their correspondence, Bromley and Moorthy[1] reported the intraoperative failure of a pulmonary artery catheter (PAC) placed in the right external jugular vein (EJV). They concluded that variations in the EJV–subclavian vein (SCV) junction in occasional patients can lead to acute angulation and kinking of the PAC and malfunction of the catheter.

The chest x-ray presented demonstrates a straight segment of catheter entering the thorax from the right side of the neck, angulating acutely at the EJV–SCV junction and thereafter descending in a gentle curve to the right heart. Close inspection indicates the cervical catheter segment to be more radiopaque and wider than that distal to the site of kinking. The more prominent cervical segment likely represents the PAC within the sheath of the introducer–dilator assembly through which the catheter was inserted. The PAC is kinked in the SCV as it exits the sheath. While the authors suggest the acute angulation

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is anatomic in origin, the evidence they present indicates it is produced by the sheath, which is inserted to the depth of the inferior wall of the SCV, kinking the catheter between its tip and the vessel wall.

We have observed three instances of intraoperative PAC malfunction manifested by dampening of the pulmonary artery tracing and marked resistance to injection that occurred upon sternal retraction during cardiac surgery. As in the authors' case, the catheters were inserted without difficulty through the right EJV with the use of a modified Seldinger technique and flushed continuously. In both instances proper catheter function was restored by withdrawing the sheath 2–3 cm, leaving the PAC in its original position and allowing it to resume a less acute course through the EJV–SCV junction.

Pulmonary artery catheterization via the EJV is a safe and effective technique, avoiding the complications associated with internal jugular cannulation.[2-4] The risk of carotid artery puncture, cervical hematoma, phrenic nerve injury, and pneumothorax can be avoided in 75% of patients by preferential use of the EJV.[3,4] It would be unfortunate if Bromley and Moorthy's report were to discourage use of the EJV for pulmonary artery catheterization.

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Reported Nerve Conduction Velocities Clarified and Confirmed

To the Editor:—There is conflict between direct and indirect indications of conduction distance in the article by Fink and Cairns.[1] Length of nerve in compartment b of their nerve chamber is stated to be 20 mm. However, conduction distance calculated from reported latencies and conduction speeds equals about 50 mm. Inspection of the diagram of the incubation chamber rules out an additional 30-mm conduction distance in compartments a and c of the nerve chamber, assuming proportional diagraming of the chamber.

The conflict leaves open the question of whether the conduction velocities actually were half of what are stated (*i.e.*, 0.27 to 18.7 m/s, instead of 0.55 to 37.5 m/s). Regardless of which conduction velocities are correct, Fink and Cairns have confirmed elegantly the observation de Jong and I made years ago using compound action potential recordings[2]—namely, that myelinated axons having conduction velocities in the 3–15-m/s range are more sensitive to lidocaine than is a population of small unmyelinated fibers.

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