Use. The technique has been in use now for four years, and no serious complication has arisen. No difficulties have presented themselves in routine use after the initial realization that the airway should not be passed immediately after induction in a manner analogous to that employed with an endotracheal tube after a short-acting muscle relaxant.

Passed by the nasal route, the airway seems no more traumatic than a similar endotracheal tube; the holes do not affect the profile of the tube enough to damage the mucosa. Secretions collecting in the pharynx do not block the holes significantly, and there has been no trouble with gastric inflation, despite the tripping of the criopharyngeal sphincter. With cricoid pressure and gentle inflation, IPPV is possible for short periods, although prolonged use is not recommended.

Two specific problems have arisen, and should be borne in mind. First, on one occasion, indirect intubation of the larynx was effected, unintentionally; this possibility can be reduced by pulling the larynx forward during passage of the airway. Second, once an airway insecurely fixed to its connection worked loose, and was found when retrieved to be working its way down the esophagus under the influence of active peristalsis.

REFERENCE


Hemodynamic Interaction between Pancuronium and Morphine

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The following is a report of hypertension and tachycardia which occurred in a patient given pancuronium bromide immediately after induction of anesthesia with morphine.

REPORT OF A CASE

A 54-year-old Caucasian woman weighing 73 kg was admitted to the Hospital of the Medical College of Pennsylvania with symptoms of increasing angina pectoris. She underwent coronary angiography and was subsequently scheduled for aortocoronary saphenous-vein bypass graft. Propranolol had been discontinued one week prior to operation.

Preoperative medication consisted of morphine, 10 mg, and scopolamine, 0.4 mg, intramuscularly, one hour before the induction of anesthesia. Upon her arrival in the operating room, a catheter was inserted into the left radial artery and electrocardiographic leads were placed on the patient’s limbs and chest. A large-bore intravenous catheter was inserted, and the patient was rapidly hydrated with 500 ml of Ringer’s lactate solution. Heart rate, ECG, and blood pressure were recorded continuously on a Hewlett Packard 7786-A monitoring system. Initially, blood pressure was 180/90 torr, and heart rate was 78 beats/min.

Morphine, 1 mg/kg, was slowly infused while the patient was ventilated with 50 per cent nitrous oxide in oxygen. By the conclusion of the morphine induction, blood pressure had decreased to 120/60 torr, and heart rate had decreased to 54 beats/min. Pancuronium, 0.15 mg/kg, was then given to facilitate endotracheal intubation. With the onset of clinically apparent skeletal muscle relaxation, and prior to endotracheal intubation, blood pressure increased markedly to 200/110 torr, and heart rate increased to 96 beats/min. The larynx was exposed and sprayed with local anesthetic, and the trachea was easily intubated. Because the tachycardia and hypertension persisted for several minutes,
halothane, 1 per cent, was added to the inspired gases. Both blood pressure and heart rate promptly decreased and stabilized at values of 140/80 torr and 60 beats/min, respectively. The remainder of the anesthetic course was uneventful.

**DISCUSSION**

Since pancuronium can be administered without the risk of a precipitous decrease in blood pressure, it would appear to be the muscle relaxant of choice in anesthesia for cardiac surgery. The drug is unique in that it has no tendency to produce hypotension regardless of the primary anesthetic agent.

Kelman and Kennedy investigated the cardiovascular effects of pancuronium in mechanically ventilated patients anesthetized with nitrous oxide and phenoperidine. Premedication consisted of papaveretum and scopolamine. Pancuronium, 0.07 mg/kg, caused a marked increase in heart rate accompanied by lesser increases in cardiac output and mean arterial pressure. Calculated total peripheral resistance was unchanged, suggesting little or no ganglion-blocking activity in man. Similar results were obtained in premedicated patients during light halothane anesthesia by Stoelting. Coleman et al. used slightly larger doses of pancuronium, 0.1 mg/kg, in unpremedicated patients, and observed significant increases in heart rate, cardiac output, and blood pressure. These changes did not occur when pancuronium was preceded by intravenous administration of atropine, suggesting that the cardiovascular effects of pancuronium are largely the result of its vagolytic activity. In experimental animals and isolated guinea-pig atria, pancuronium selectively blocks cardiac muscarinic receptors. It not only suppresses the inhibitory effects of vagal stimulation, but also antagonizes the negative inotropic and chronotropic actions of parasympathomimetic compounds without appreciably modifying their peripheral vascular effects.

Stoelting and Gibbs determined the cardiovascular responses to morphine, 1 mg/kg, and morphine-nitrous oxide in patients with various types of operable heart disease. In the group with coronary-artery disease, the only significant changes caused by morphine were decreases in mean arterial pressure and heart rate. The subsequent addition of nitrous oxide produced a decrease in cardiac index, an increase in systemic vascular resistance, and further reductions in mean arterial pressure and heart rate. The simultaneous decreases of heart rate and blood pressure during infusion of morphine and after the addition of nitrous oxide suggest vagal stimulation by both of these agents. Bradycardia of central origin was one of the first physiologic effects of morphine to be discovered.

The increased vagal tone induced by morphine-nitrous oxide markedly accentuated the vagolytic property of pancuronium to
produce tachycardia and hypertension. A primary aim in the anesthetic management of patients with coronary-artery disease is to minimize myocardial oxygen consumption. Since there is a close relationship between the oxygen requirement of the heart and the product of mean systolic blood pressure and heart rate,10 the combination of hypertension and tachycardia should be avoided.11 It would thus appear that large doses of pancuronium following large doses of morphine or morphine-nitrous oxide should be used with extreme caution in patients with coronary-artery disease.

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The Cardiovascular Effects of Carbon Dioxide in Man Awake and during Diethyl Ether Anesthesia

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Carbon dioxide and diethyl ether both stimulate the cardiovascular system of man by increasing sympathetic activity.1,2 The effects of these gases on the cardiovascular system have previously been examined at two end-tidal ether concentrations and normocapnia.2 In this paper, we describe the cardiovascular responses of human volunteers to several inspired concentrations of carbon dioxide during anesthesia with 3 and 4.5 per cent ether.

METHODS

We studied eight healthy, unpremedicated male volunteers. The experimental design,