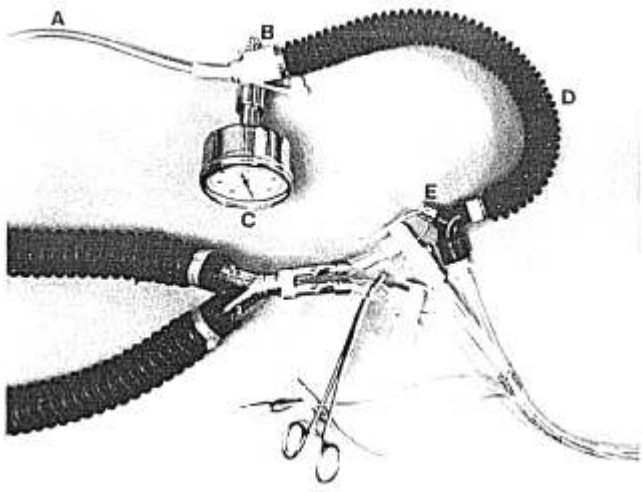

FIG. 1. The endobronchial CPAP device consisting of (A) oxygen connecting tube (#H8294-003119, Bard-Parker, Rutherford, New Jersey); (B) swivel adapter (#625109, Portex, Inc., Wilmington, Massachusetts); (C) inspiratory force meter (#60-60) with connector (#00-275-1, Clarence A. Smith, Inc., Arlington, Massachusetts); (D) Flexible corrugated adapter (#1100554, North American Drager Co.); and (E) elbow pop-off valve (Clarence A. Smith, Inc., Arlington, Massachusetts).

but could be modified easily for use with other endobronchial tubes.

It is pictured in figure 1 and consists of a length of oxygen tubing (A) from a flowmeter fitted to the rubber septum of a Portex swivel adapter (B). The 15-mm O.D. sidearm of (B) holds an aneroid manometer (C) measuring airway pressure. The 15-mm I.D. end of (B) is attached to a rubber flexible adapter tube (D). The distal (patient) end of this tubing is fitted with an elbow connector with integral pop-off valve (E). This fits on the standard endotracheal tube connector of either Bronchocath lumen when the corresponding right-angle connector with suction port is clamped and disconnected during one-lung ventilation. In use, the oxygen flow rate and/or pop-off valve is adjusted to provide the desired amount of CPAP as measured on the manometer. We also have used the valve assembly and mask adapter for the Magill breathing system in place of the elbow connector (E) with success.

We have found that the ready availability of this device and its ease of operation simplify the management of hypoxemia during one-lung anesthesia.

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Propranolol prior to ECT Associated with Asystole

To the Editor:—A recent Clinical Report by Hood and Mecca[1] recommends the use of intravenous propranolol to attenuate the hypertensive response to ECT. We would like to report a case illustrating the occurrence of asystole associated with the use of iv propranolol in combination with ECT.

REPORT OF A CASE

A 68-year-old woman with her sixth episode of severe depression was scheduled for ECT. Electroconvulsive therapy was recommended because the current episode did not improve with an outpatient trial of pharmacotherapy. The patient's medical history included a myocardial infarction 5 yrs prior to admission and insulin-dependent diabetes mellitus. Admission laboratory tests were all within normal limits except for a fasting blood sugar of 337 mg/dl and the ECG, which showed an old anteroseptal myocardial infarction. Physical examination was unremarkable. The morning of the first scheduled treatment, the patient exhibited anxiety prior to treatment, with a pulse rate of 100/min and blood pressure of 180/120 mmHg. Before induction of anesthesia, propranolol, 1 mg iv, was administered, and the standard administration of atropine 0.4 mg iv was omitted. Thiopental 1.9 mg/kg, and succinylcholine, 0.5 mg/kg, were used to induce general anesthesia and muscle relaxation. Oxygen (100%) was administered via mask by positive pressure from the onset of induction. The treatment was monitored continuously by single-channel ECG and EEG. A bidirectional brief pulse electrical stimulus was delivered bilaterally without eliciting a seizure. The stimulation, however, was followed by progressive slowing of sinus rhythm for a period of 5 s, ultimately resulting in asystole. Cardiopulmonary resucitation was instituted, following which regular sinus rhythm resumed after a total of 15 s of asystole. The patient recovered uneventfully from the anesthesia. Cardiologic evaluation at follow-up reported no sequelae from the event. ECT treatment was interrupted in favor of further pharmacotherapy and psychotherapy. After 4 months of treatment without

any symptomatic improvement, the patient was referred back to ECT. She was premedicated with atropine, 0.5 mg im and 0.4 mg iv, and then was given 0.9 mg/kg methohexital and 0.5 mg/kg succinylcholine. Electric stimulation of sufficient intensity to elicit a generalized seizure was used during the treatment. The patient tolerated a course of 13 treatments without cardiac complications and with good remission of symptoms.

Cardiac arrest is a well-documented, although rare, complication of ECT. In the present case, the tendency to bradycardia and asystole was probably heightened by the use of beta blockade. The subconvulsive electrical stimulation also could have contributed. Experimental data support the notion that an adrenergic mechanism is involved in the phenomenon of vagal escape[2] and that, in the presence of sympathetic blockers, a shock-induced activation of the autonomic nervous system can lead to a parasympathetic mediated cardiac arrest. This does not normally occur with ECT because the seizure elicits a marked peripheral sympathetic response that results in a rise in heart rate. With a subconvulsive shock, the central parasympathetic mechanism was unopposed and resulted in a slowing of the heart rate; a phenomenon exacerbated by beta blockade. It seems probable that propranolol was involved, at least in part, in the pathogenesis of the asystole. Therefore, we feel that one should exercise caution when using the combination of propranolol and ECT.

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Respiratory Depression Following Only 0.4 mg of Intrathecal Morphine

To the Editor:—Intrathecal and epidural opiates have been used successfully to produce postoperative analgesia. However, respiratory depression is a problem with this technique with doses of 1 mg or more. However, we observed a case of respiratory depression with a much smaller dose, 0.4 mg, of intrathecal morphine. A 74-year-old patient scheduled for a peripheral orthopedic procedure was premedicated with 5 mg of diazepam orally, 2 h preoperatively. Analgesia was obtained using 0.8 ml heavy lidocaine (*i.e.*, 40 mg lidocaine) inserted via a spinal needle at L_{2-3} with the patient in the left lateral position and maintained 15 degrees head up. This was followed by 0.4 mg of preservative-free morphine in 5% dextrose water. Flunitrazepam, a total of 1 mg, was given iv intermittently during the procedure.

Postoperatively, inspired oxygen was increased with the help of a 28% ventimask. It was instructed that she be kept slightly head up. Accidently the patient was put head down for half an hour. Two-and-a-half hours later, the patient was cyanotic, with a respiratory rate of 6 breaths · min^{-1}, an arterial blood pressure of 70/50 mmHg, and a heart rate of 46 beats/min. Naloxone 0.4 mg was given iv and 0.4 mg im. This resulted in immediate improvement in respiratory rate to 10 breaths · min^{-1}, arterial blood pressure to 100/50 mmHg, heart rate to 50 beats/min, and her color returned to normal. No further naloxone was required.

The intrathecal morphine was the only narcotic given during the perioperative period. Pain relief lasted for 36 h.

The cause of the respiratory depression is most likely due to rostral spread of the morphine following the normal flow of cerebrospinal fluid from the lumbar region to the basal cisterns and which may then enter the ventricular system.[1]

The large difference in incidence of respiratory depression following epidural as compared with intrathecal morphine is probably due to the fact that morphine does not as readily enter the cerebrospinal fluid when

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