6. One 2½ inch flat flashlight with batteries
7. One small roll of 1 inch adhesive tape
8. One ball-point pen.

In figure 3 the endotracheal tubes and their method of carriage are shown. They are assembled (aluminum stilets in place) in the inverted lid of an old bread box and encircled at each outer third by wide rubber bands. The tubes are placed according to size, in chronological order, from the top to the bottom in the lid, and are easily removed and replaced when the occasion demands.

The “Carry All” when fully loaded, as shown in figure 1, weighs only 10½ pounds. It is light, compact and portable. It may quickly and easily be transported to the x-Ray or cystoscopy departments, on the stretcher with a patient going back to his room, to the wards, or from operating room to operating room. The equipment present in the box permits the anesthesiologist to prepare for and carry out anesthesia for almost any type of surgical procedure, major or minor, pediatric or adult.

In addition to using the apparatus routinely for our daily surgery schedule, it has proved its worth in such emergencies as the resuscitation of the newborn (endotracheal ventilation), treatment of convulsion (adult and pediatric) and cardiac arrest. With this box, the anesthesiologist need not carry odds and ends in and about anesthesia machines or in pockets. All that is needed is a mobile anesthesia machine, the “Carry All” and the patient.

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PROLONGED APNEA FOLLOWING CARDIAC EXPLORATION

The potential hazards of positive pressure respiration are well known (1-3). In an extensive review of the subject, however, Watrous (4) concluded that manually assisted and controlled respiration during anesthesia facilitated the maintenance of a nearly normal physiologic state. It has been demonstrated that although positive pressure is of benefit in the treatment of emphysematous patients (5), it can also reduce venous inflow and cardiac output, particularly in those whose circulation is already impaired (1). Maloney (6) among others, recently has revived interest in the importance of a negative pressure phase in mechanical respirators.

The following is a case report of a patient who presented both these conditions to a severe degree, namely, chronic obstructive emphysema and rheumatic heart disease.

A 45 year old white man was scheduled for a mitral commissurotomy. The preoperative diagnosis was rheumatic heart disease, grade 3, with mitral stenosis and auricular fibrillation and chronic obstructive emphysema. Essentially, the preoper-
tive treatment had consisted of the continuation of administration of digitalis, diuretics, and penicillin inhalations. Quinidine had been given at one time, then abandoned. The blood pressure was 100 mm. systolic and 80 mm. diastolic; the pulse was 82, and the hemoglobin was 18 Gm. per 100 cc. The drugs given preoperatively were morphine, 10 mg., and atropine, 0.16 mg.

Anesthesia was induced with 200 mg. of pentothal® and cyclopropane and maintained with endotracheal ether. After the patient was placed in the right lateral position fluids were given by infusion. Mean arterial pressures were determined according to the method of Alman (7). This needle later became displaced. Respirations were manually controlled since apnea had ensued after placing the patient in position.

The anesthetic course during the procedure was fairly satisfactory except for a slight to moderate hypotension; the systolic pressure by palpation remained about 75 to 80 mm. of mercury and mean arterial pressures from 60 to 70 mm. of mercury. At one time during the procedure, neosynephrine®, 0.5 mg., was given intravenously and resulted in a slight rise of blood pressure, then prompt return to its previous level. The auricular fibrillation with a slow ventricular rate was recorded on a direct writing electrocardiogram.

The operative procedure consisted only of cardiac exploration. The left auricular appendage was calcified and it was decided not to proceed with the commissurotomy. An additional finding was a markedly voluminous lung with scattered emphysematous blebs. The chest wall was closed and the patient placed in the supine position. Estimated blood loss was 500 cc. A small amount of mucus was obtained by suction through the endotracheal tube. There were no spontaneous respiratory movements.

The significant changes during the ensuing four hours may be seen on the chart. Marked circulatory deterioration resulted when the carbon dioxide absorber was turned off. Tachycardia occurred promptly with the administration of atropine, 0.2 mg. and there was another episode in which blood pressure or pulse could not be obtained. The patient’s condition improved when a second blood transfusion was given, and neosynephrine 5 mg., and cedilanid®, 0.4 mg., were given intravenously, and improvement was sustained when the patient was placed in the respirator. Within fifteen minutes the blood pressure was 110 mm. systolic and 80 mm. diastolic and spontaneous, shallow respirations had started. Clinically, the patient appeared to be better oxygenated and reacted readily to painful stimuli. After forty-five minutes he was taken out of the respirator. The subsequent course was fairly satisfactory, but repeated aspirations were necessary for troublesome accumulations of mucus. He was discharged from the hospital two weeks after the procedure, apparently in as good condition as at the time of admission. He was re-admitted several weeks later and died eventually in cardiac failure.

**DISCUSSION**

Although carbon dioxide studies were not done, the probable mechanism initially of apnea was one employed frequently, namely, overdistention and overventilation (8). This was markedly facilitated by the central respiratory depression resulting from hypotension during the procedure. Possible contributory causes were the anesthetic drugs and high oxygen concentrations (9). Since apnea did not start until after intubation and positioning, the effect of intubation probably was merely contributory and not a major factor. Decreasing oxygen concentrations by employing nitrous oxide as a diluent in as little as approximately 20 per cent concentrations resulted in the appearance of cyanosis. Higher concentrations of nitrous oxide did not result in any attempts at spontaneous respiration, ostensibly through anoxic receptors. Furthermore, spontaneous respirations quickly followed when the patient was placed in the respirator, high concentrations of oxygen still being administered.

Manual inspiratory pressures used averaged 8 mm. of mercury except for full lung expansion at closure when pressure of 15 mm. of mercury was used. Pentothal was employed during induction to aid in avoiding excitement resulting from poor alveolar diffusion. With this exception the
anesthetic management paralleled closely that we have employed for patients undergoing mitral commissurotomies.

Near the end of the procedure when inspiratory pressure of less than 8 mm. of mercury was used, the blood pressure rose, but slight cyanosis developed. Occasional, very shallow respiratory efforts appeared for a short while. Spontaneous breathing was not present after the patient was placed in the supine position and a suction catheter was inserted. For the next hour circulation was relatively stable. When attempts were made to allow the patient to initiate his own respirations, cyanosis appeared. All anesthetic agents had been discontinued thirty minutes before closure.

The circulatory deterioration that followed shutting off the carbon dioxide absorber could well have been related to myocardial depression (10). On the assumption that vagal reflexes were contributing to the apnea, atropine was administered and the marked tachycardia resulted. We had had this experience previously, as have others (11). Apparently this led to more circulatory depression. Administration of blood with neosynephrine and ecdlialid caused moderate improvement. The former was added to decrease the tachycardia in addition to its pressor effect. Recovery followed when the patient was placed in the respirator. Its use was delayed because of transportation difficulties.

Although the organic diseases in this patient differed from those in the cases reported by Maloney (8), the cause of prompt improvement is believed to be the same. Clinically it seems, by the conventional methods, we were unable to oxygenate this patient properly without causing some circulatory embarrassment. A positive and negative phase in the tank respirator simulates a “suck and blow” type (2). This mechanism, by its salutary effect on the circulation, apparently caused the prompt recovery.

CONCLUSION

A case is presented of apnea of four hours' duration following cardiac exploration in a patient with rheumatic heart disease and pulmonary emphysema. The possible causes and management are discussed. It is believed the most important factor in recovery was the circulatory improvement following the use of the tank respirator.

REFERENCES


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