unless the external cardiac massage produced a higher pressure in the chest than 40 cm of water, at which point the safety valve would open and the ventilator would automatically recycle. After resuscitation of the patient, it may be necessary to move him from the position at the time of arrest to the Intensive Care Unit. To do this, a small oxygen cylinder can be connected to the respirator. The cylinder is placed alongside the patient on his stretcher (fig. 2). The patient can then be transferred to the Intensive Care Unit with assured adequate ventilation. Should the patient recover sufficiently to be able to breathe spontaneously, and adequately, adjustments can then be made to the respirator to convert it to a McGill-type system for spontaneous respiration. The patient will be breathing 100 per cent oxygen.

The ventilator has been used on 24 cardiac arrests in the past four months, and functioned perfectly in all but two. The first of these two was a patient in a status asthmaticus, whose hypoxia precipitated the cardiac arrest, and it was found necessary to increase the power of the respirator by manually squeezing the reservoir bag and holding down the safety valve (pressures of 60–70 cm of water were necessary to ventilate the patient). The second patient developed a left pneumothorax following the intracardiac injection of epinephrine, and, in this case, the safety valve consistently opened during inspiration, warning us that the patient was not being adequately ventilated and that, in fact, the compliance had certainly dropped. The appropriate treatment was then instituted.

**CONCLUSION**

A simple, inexpensive, light, portable, efficient ventilation system is described. It is suitable for treatment of cardiac or respiratory arrests; it is efficient and satisfactory in operation in these emergency situations until such time as the patient can be connected to a more conventional ventilation system.

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**REFERENCES**


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**Inhibition of Hiccup by Pulmonary Inflation**

ANIS BARAKA, M.B., B.CH.

Hering and Breuer have shown that maintained distention of the lungs of anesthetized animals inhibits respiration; the effect is a reflex mediated by afferent vagal fibers. In recent years, similar mechanisms have been demonstrated in anesthetized man by Guz et al., who showed that deep inflation of the lungs produces respiratory inhibition which can be abolished by bilateral vagal block.

Hiccup is an abnormal respiratory pattern which might be subject to the same reflex control as normal respiration. The purpose of the present investigation is to observe in anesthetized man the effect of pulmonary inflation on hiccup developing during surgery.
Fig. 1. Polygraph recording of airway pressure and air flow in a patient developing hiccup during gastrectomy. Hiccup resulted in airway pressure peaks as low as −10 cm H₂O. Manual compression of the bag to maintain the airway pressure at about 20 cm H₂O failed to stop hiccup. When the pressure was increased up to 30–40 cm H₂O, hiccup immediately stopped.

METHOD

The subjects of the study were ten adults who developed hiccups during upper abdominal surgery, viz., cholecystectomy and gastrectomy. All patients were free of cardiopulmonary disease; they ranged in age from 30 to 54 years.

Premedication consisted of pethidine, 100 mg, and atropine, 0.6 mg, injected intramuscularly an hour before operation. Sleep was induced with thiopentone, 250–350 mg, followed by suxamethonium, 100 mg. All tracheas were intubated with a cuffed tracheal tube. Anesthesia was then maintained by a mixture of nitrous oxide (3 l/min) and oxygen (1 l/min), supplemented with tubocurarine, 20–40 mg. Respiration was manually controlled throughout the procedure, using a Boyle III circuit.

The airway pressure was measured by a Statham strain gauge. This was connected to the side arm of a T-piece incorporated between the tracheal tube connection and the anesthesia circuit. Respired air passed through a heated Fleisch pneumotachograph connected to a Statham differential strain gauge. Both airway pressure and air flow were recorded on a Grass Polygraph.

When hiccup was observed, the airway pressure was increased for 10–20 seconds by sustained manual compression of the reservoir bag while adjusting the outlet valve. The threshold airway pressure which stopped hiccup with its associated air flow was observed.

RESULTS

In all patients, hiccup developed during visceral manipulations. The jerky diaphragmatic contraction resulted in negative airway pressure peaks as low as −10 cm H₂O. Interruption of surgical manipulation did not stop the hiccups. In all patients, however, hiccup stopped immediately when the airway pressure was increased for 10–20 seconds by manual compression of the reservoir bag. The threshold pressure required to stop hiccup ranged from 30 to 40 cm H₂O. Figure 1 shows the response observed in one of the patients.

COMMENT

Hiccups are frequently observed during upper abdominal surgery in patients anesthetized with nitrous oxide, oxygen and tubocurarine. The afferent limb of the reflex is the vagus nerve, phrenic nerve, and the sympathetic chain from the sixth to the twelfth thoracic
segments; the efferent limb is the phrenic nerve. Once hiccup is triggered it often persists even after surgical manipulations have ceased. The sequence of events suggests a self-perpetuating cycle.

Sustained pulmonary hyperinflation stopped hiccup in all patients. The immediate cessation of hiccup following inflation suggests a reflex action. It is probable that the Hering-Breuer inflation reflex can inhibit not only the normal respiratory pattern, but also the abnormal pattern of hiccup.

The problem with this and comparable studies of the treatment of hiccup, however, is that hiccup often stops spontaneously. Methylphenidate has just been shown to be no more effective than saline. There will always be some doubt that hiccup has stopped as a result of the specific treatment rather than spontaneously.

An End-expiratory Sampler for Use during Positive-pressure Ventilation in Man

EUGENE R. LUCIER, M.D., AND LEROY D. VANDAM, M.D.

Several methods for collection of end-expiratory gases have been devised for use in subjects breathing spontaneously. Others applicable to positive-pressure ventilation involve the use of pressure-activated switches and pumps. The simple apparatus described below for collection of end-expired gases during controlled ventilation uses the positive pressure applied to the airway as an energy source. The sample (5–10 ml/breath) is collected during inspiration from the expiratory side of a nonrebreathing valve connected to the subject's airway. A small balloon contains the sample temporarily until it is removed for analysis. The performance of the sampler was evaluated by comparison of end-expiratory carbon dioxide tension with the carbon dioxide tension in arterial blood drawn simultaneously.

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REFERENCES


MATERIALS FOR APPARATUS

2 20-ml Multi-fit syringes 1 elastic band
1 20-ml toy balloon 3 lengths of intravenous tubing
1 50-ml plastic bottle 1 small baseboard
1 two-hole rubber stopper 1 one-way valve from sphygmomanometer bulb

The assembled apparatus is shown in figure 1. Pressure applied to the subject’s airway is transmitted to the first syringe, forcing the plunger out. The plunger of the second syringe, coupled to that of the first, is withdrawn, pulling air out of the plastic bottle and drawing a sample of gas from the expiratory line into the reservoir balloon. Thus, the sample is collected throughout inspiration. When inspiration ceases, the elastic band empties the syringes—one to the subject’s inspiratory line, the other to the atmosphere through the one-way pop-off valve inserted in the negative pressure line. The pop-off was inserted to