tion of clinical doses of succinylcholine and the observed decreases in serum Ca^{++}.

The increase in serum K^{+} after succinylcholine is believed to be the result of leakage through traumatized skeletal muscle cell membranes. This mechanism could explain our results as well. Since very little intracellular Ca^{++} is rapidly exchangeable,[5] there is a concentration gradient to force Ca^{++} intracellularly following the disruptive depolarizations due to succinylcholine.

Another explanation for the decrease in serum Ca^{++} involves active intracellular transport during depolarization. There is considerable evidence that most excitable cells (neurons, smooth muscle cells, and myocardial cells[5–8] have a net uptake of calcium ions associated with excitation. Several papers[6–11] offer convincing data that skeletal muscle cells utilize extracellular Ca^{++} during depolarization. Calcium, concentrated in the transverse tubules, which are in free communication with the extracellular space, is transported intracellularly as depolarization proceeds past the transverse tubule. Intracellularly, it acts to initiate the events of contraction. Loss of extracellular Ca^{++} to the intracellular space may account for the decreases we observed.

The finding of significant decreases in serum Ca^{++} calcium following administration of succinylcholine may not be clinically significant. At present we know of no disease or other circumstance that would make these changes reason to modify current anesthetic techniques.

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Barotrauma, a Potential Hazard of Manual Resuscitators

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Hand resuscitators are used in intensive care units and recovery areas for resuscitation and for transport of patients who need ventilatory support. This paper reports two cases in which inadvertent modification of hand resuscitators resulted in morbidity.

Report of Two Cases

Patient 1. A 65-year-old man was admitted to the intensive care unit following elective repair of an abdominal aortic aneurysm. The patient's trachea remained intubated immediately postoperatively and he was ventilated with a constant-volume ventilator. Prior to suctioning the endotracheal tube, an attempt was made to ventilate the patient with a hand resuscitator (Hope Resuscitator, Ohio Medical Products, Madison, Wis.) connected to a source of oxygen. Following the initial manual squeeze on the resuscitator bag, the resuscitator rapidly became distended, the patient was unable to exhale, and he suddenly became agitated. Removing the resuscitator from the endotracheal tube relieved the problem, and the patient was readily ventilated with the constant-volume ventilator without incident.

Patient 2. An 81-year-old woman was admitted to the recovery room after an eight-hour operation on the biliary tract. During transport from the operating theater, the patient, whose trachea was still intubated, was ventilated with a hand resuscitator (Hope Resuscitator, Ohio Medical Products, Madison, Wis.), without supplemental oxygen and without incident. When the resuscitator was connected to a flowmeter-controlled source of oxygen in the recovery room, the resuscitator bag rapidly became tense, and the house officer was unable to ventilate the patient. The patient promptly sustained obvious subcutaneous emphysema and suf-

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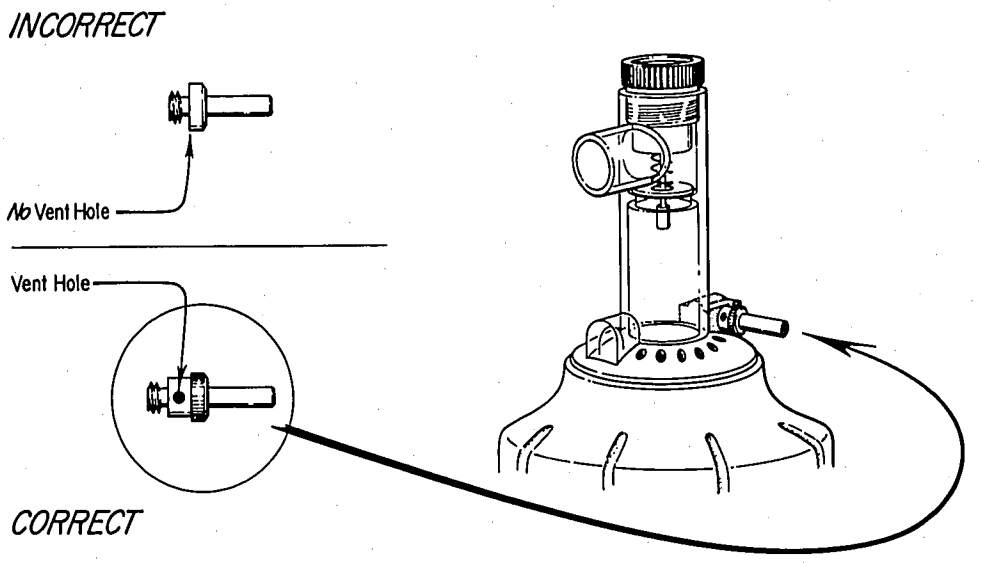

FIG. 1. The Hope resuscitator is shown with the *correct* oxygen nipple, with vent holes, as supplied by the manufacturer with the nipple in place. The *incorrect* oxygen nipple, without vent holes, but with the same thread size as supplied on the Blount Adaptor, is also shown (see text).

fered cardiac arrest. Placement of bilateral chest tubes resulted in return of hemodynamic stability, and ventilation was provided with an alternate resuscitator.

DISCUSSION

The possibility of tension pneumothorax with use of the early models of the "Hope" resuscitator was pointed out ten years ago.[1] Since then, the oxygen-inlet nipple of the resuscitator has been modified by the manufacturer to preclude such a hazard. These two case reports illustrate the danger of serious mishap when such resuscitators are, however, inappropriately modified or reassembled. Three conditions must have been present for either of the above-mentioned mishaps to have occurred: the absence of a functional preset safety release valve, a flow of gas into the O_2 nipple of the resuscitator, and the absence of side holes on the O_2 nipple to vent an excessive flow of gas safely. In each of the above cases these conditions were met. Fresh O_2 inflow exceeded 15 l/min in both cases. In the first case, the safety release valve was permanently occluded; in the second, a resuscitator without a safety release valve was employed. Finally, in each case, the O_2 inlet nipples supplied on the original equipment by the manufacturer had been replaced at the time of cleaning and reassembly by ventless nipples of the same thread size (see fig. 1). The source of such oxygen nipples, without vent holes, was found to be the Blount Percent Adaptor (Blount, Inc., Ashland, Va.), commonly used to increase the concentration of oxygen delivered by the hand resuscitator. These nipples, removed from the adaptors at the time of cleaning, resemble the original nipples in superficial appearance and thread size. They were mistakenly put on the Hope resuscitator valve bodies at the time of reassembly immediately following cleaning.

When the conditions described above are met, firm pressure on the resuscitator bag moves the valve to the inspiratory position, supplying the patient with a tidal volume. Fresh gas inflow cannot be vented because positive pressure is maintained within the bag–valve–patient system and the valve is held in the inspiratory position. Thus, with an excessively high fresh gas inflow, a rapidly increasing pressure is transmitted to the patient's pulmonary system. In the first case, early recognition of the problem with rapid disconnection of the resuscitator from the endotracheal tube prevented barotrauma. In the second case, the patient suffered multiple bilateral subpleural hematomas and pleural tears, demonstrated at autopsy.

To determine the minimum oxygen flow rate that would result in valve malfunction, a Hope Resuscitator Bag without a pressure-relief valve and incorporating a ventless O_2 nipple was used to ventilate a standard test lung (Michigan Instruments Vent Aid TTL, Collins 120L. Chain Compensated Gasometer) set for a compliance of 50 ml/cm H_2O and a resistance of 5.25 cm H_2O/l/sec. At a respiratory frequency of 12/min and an inspiratory:expiratory ratio of 2:3 with a 1-l tidal volume, the resuscitator valve was found to jam with as little as 7.0 l/min O_2 flowing into the bag. Expiratory flow rate plays an important role in determining at what O_2 flow rate the valve will stick. Sudden release of hand pressure at end inspiration reduces pressure within the bag and hastens the movement of the valve to the expiratory position. Slow release of hand pressure, however, tends to maintain positive pressure within the bag–valve–patient system during fresh gas inflow and thus maintain the valve in the inspiratory position.

Several suggestions are offered to avoid such accidents in the future. The person operating the resus-

citator must be vigilant to a sudden deterioration in compliance. When the Ohio hand resuscitator is used without the Blount Percent adaptor, the vented O_2 nipple should be permanently bonded to the resuscitator housing or have standard threads of a design to preclude its interchange with incompatible fittings. The nonvented nipple on the Blount adaptor should be permanently fixed in place to avoid any possibility of removal and interchange with the vented nipple. A pressure-limiting system should be incorporated with an audible alarm. This system should safely limit the peak pressure within the system to some arbitrary limit and warn the operator when that pressure is reached. The manufacturer should provide an indelible warning label on the resuscitator body if the resuscitator valve is likely to jam beyond a certain flow rate of O_2. As with any type of anesthetic apparatus, close inspection prior to its use is mandatory.

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Pulmonary Vascular Pressure Reading at the End of Exhalation

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Intermittent mandatory ventilation (IMV) with and without positive end-expiratory pressure (PEEP) is a commonly used ventilatory pattern in the management of patients who need mechanical ventilatory assistance.[1] Similarly, the balloon-tipped pulmonary arterial catheter is frequently used in assessing the cardiovascular status of critically ill patients.[2] Often IMV and pulmonary-artery catheters are utilized simultaneously in patient care. Presently it is recommended that pulmonary vascular pressures be obtained while the patient continues with the ventilatory pattern existing during treatment, because a different ventilatory pattern (*i.e.*, apnea and no PEEP) may cause irrelevant, misleading physiologic changes.[3] The recommended moment to read pulmonary vascular pressure during the ventilatory pattern existing during treatment is at the end of exhalation.[4]

We demonstrate and discuss a previously unreported problem in reading pulmonary arterial and pulmonary arterial wedge pressures in patients who have rapid wide swings in pleural and airway pressures. Such patients include those managed with IMV who have rapid spontaneous and mechanical ventilatory rates as well as decreased pulmonary compliance. Under these circumstances pulmonary vascular pressure patterns have wide fluctuations, and it is often difficult to determine the end of exhalation by the vascular pressure pattern alone or by trying to time the pattern with observation of the patient. We therefore simultaneously recorded airway and pulmonary vascular pressures.

Methods

Ten critically ill patients with endotracheal tubes in place, needing mechanical ventilatory assistance utilizing IMV, were studied consecutively. These patients also needed positive end-expiratory pressures (PEEP) ranging from 3 to 10 torr and $F_{I_{O_2}}$ 0.3–0.5 to maintain Pa_{O_2} 67–123 torr.

Pulmonary arterial and pulmonary arterial wedge pressures were measured with a triple-lumen balloon-tipped catheter (Edwards #93A-113-7F) introduced into the pulmonary artery by the modified Seldinger technique via the internal jugular or subclavian vein. Airway pressures were measured directly by a T-connector interposed between the endotracheal tube and the ventilator hose. Both vascular and airway transducers (Hewlett-Packard 1280C) were calibrated with standard mercury manometers and were then referenced to atmospheric pressure at the level of the left atrium. Simultaneous pulmonary vascular and airway pressure patterns were recorded by a two-channel recorder.

During each patient's clinical course many simultaneous recordings of pulmonary vascular and airway pressure patterns were made. The pulmonary vascular pressure values at the end of exhalation did not vary more than 2 torr throughout any particular recording, each recording being continuous for at least 0.5 to 2 minutes. Therefore, all exemplary pulmonary vascular pressure values given for each patient are absolute values taken from arbitrarily selected but

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