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Pulmonary Barotrauma Resulting from a Faulty Hope® II Resuscitation Bag

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Hand resuscitators often are needed during transfer of patients requiring controlled ventilation. We describe a patient in whom a faulty resuscitator valve caused high airway pressures and resulted in pulmonary barotrauma and subcutaneous emphysema.

REPORT OF A CASE

A 61-year-old woman was rushed to the operating room for emergency control of an upper gastrointestinal hemorrhage. Other medical problems included kyphoscoliosis, restrictive lung disease, and an unresectable pelvic tumor. During transit to the operating room she suffered a cardiac arrest and cardiopulmonary resuscitation was instituted promptly. A tracheal tube was inserted to facilitate ventilation of the lungs and to protect against aspiration of secretions and blood.

Anesthesia was induced with 100 mg ketamine and 15 mg metocurine, iv, and maintained with 50% nitrous oxide. Prior to skin incision, arterial blood pressure was 110/70 mmHg and heart rate 104/min. Ventilation was controlled with a tidal volume of 600 ml and a peak inspiratory pressure of 35 cmH_2O. Laparotomy revealed erosive gastritis and a subtotal gastrectomy was performed. One liter fresh frozen plasma and 2.3 l lactated Ringer's solution were given iv. Arterial blood pressure remained at 80 mmHg (systolic) despite iv fluid replacement and maintenance of central venous pressure at 15 mmHg. Dopamine and later epinephrine infusions were used to maintain a systolic blood pressure between 80 and 100 mmHg. Nearing the end of surgery, with a FI_{O_2} of 0.5 Pa_{O_2} was 90 mmHg, Pa_{CO_2} 42 mmHg, and pH_a 7.38. A transport stretcher equipped with a Hope® II resuscitator was prepared to transfer the patient to the recovery room. The resuscitator was checked for flow of oxygen and the ability to deliver positive pressure and to refill spontaneously. Once the patient was placed on the stretcher, controlled ventilation was attempted. After two to three breaths, ventilation became extremely difficult, and the patient appeared cyanotic. A diagnosis of tension pneumothorax was considered and the patient was moved back to the operating table and ventilated via an anesthesia machine. Ventilation then became easy and breath sounds were heard over both lung fields. The patient was transferred back to the stretcher and again difficulties ensued with manual ventilation. The patient was ventilated using the anesthesia machine until another Hope® II resuscitator was obtained. Using the second resuscitator, the patient was transferred to the recovery room uneventfully. Within 5–10 minutes, subcutaneous emphysema was noted over the neck and upper chest. Breath sounds were equal over both lung fields, the trachea had not deviated from the midline, and a chest roentgenogram showed no pneumothorax. Peak inspiratory pressures (which were 35–40 cmH_2O intraoperatively), however, had now increased to 50 cmH_2O. The subcutaneous emphysema got progressively worse over the next 30 minutes during which time cardiovascular instability increased; the patient expired 30 minutes later. Postmortem examination was not performed.

When the Hope® II resuscitator was inspected, there were no obvious abnormalities. Specifically, the unit was assembled properly. The bag appeared to fill and empty appropriately. With the patient connection occluded manually, the bag could not be compressed. After compressing the bag with the patient connection open, the bag refilled promptly when the patient connection was either open or closed. During these maneuvers, the ball-valve moved normally. However, when the valve cap was removed and the ball removed from the ball cage, a fracture line traversing the ball across its entirety was obviously present.§

When the involved Hope® II resuscitator was later connected to an anesthesia bag simulating a patient, the malfunction produced by the cracked ball was demonstrated easily. Figures 1 and 2 demonstrate what was observed with the fractured ball. Specifically, for the first few breaths there was no impediment to inspiration when the bag was compressed. When the bag compression was relieved, however, the two portions of the fractured ball separated, and gas could not be released from the "patient" portion of the circuit. Several valves from other Hope® II resuscitator units then were examined, and in one instance, a crack involving only part of the circumference of the ball-valve was discovered.

DISCUSSION

Pulmonary barotrauma results from unusually high pressure differences between anatomic compartments in the chest. Precipitating factors include mechanical ventilation, non-compliant lungs, presence of emphysematous bullae, violent coughing, and inspiration or expiration against a closed glottis. However, the injury does not always involve the surface of the lung and the pleura. Therefore, patients with barotrauma need not have concurrent pneumothorax. When the injury occurs in the peri-bronchial area, gases can dissect prox-

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§ Hope® II resuscitators in our institution are cleaned and sterilized in accordance (Ohio Medical Products, Madison, Wisconsin) with the manufacturer's recommendations. They are first washed with Super Edisonite non-filming detergent and dried at 130° F. They are then reassembled and sterilized with ethylene oxide at 130° F and aerated prior to subsequent patient use. Contact with alcohols and alcohol-based substances is avoided to prevent softening of the plastic.

imally towards larger airways and can then communicate with the mediastinum, the peritoneum, and subcutaneous tissues of the neck, face, and chest. If there is concurrent vascular injury, gas embolism can occur with disastrous consequences. Although some of the anatomic and pathophysiologic mechanisms appear subtle, the clinical course of events usually occurs over a short period of time. Appropriate therapeutic measures may be lifesaving. The mechanical problem encountered by us was communicated to the manufacturer (Ohio Medical Products) as well as to the Food and Drug Administration (FDA). Seven other instances of ball-valve separation had been reported previously from other institutions; however, all had been discovered before any complications occurred. Within three weeks of our report, the manufacturer issued an urgent Medical Device Alert advising users to carefully inspect ball-valves during reassembly after cleaning. Within three months of our report, newly designed ball-valves were in production. The ball is now made of clear plastic and has an increased surface area of contact at the solvent weld site. In our institution, all ball-valves have been replaced and are now subjected to mechanical stress under direct observation during reassembly.

In this patient, the inability of the lungs to deflate during positive pressure ventilation resulted in very high airway pressures. The trauma must have occurred instantaneously since ventilation became progressively difficult with the next few breaths. The pleurae were not involved, as judged by clinical and roentgenographic examination, otherwise the placement of a chest-tube would have been necessary. If injury occurs at the bronchial level, gases may dissect within the lung tissue and through peribronchial areas to subcutaneous tissues. In this patient, there was subcutaneous emphysema but no pneumothorax. The lungs became less compliant and made cardiorespiratory support difficult. Pulmonary barotrauma that does not result in pneumothorax, but manifests itself as subcutaneous or mediastinal emphysema and dissection of gases within the lung parenchyma is extremely difficult to treat. Chest tube placement could have been performed to treat pneumothorax in the event that it should occur later. Large bore needles or small skin incisions have been used to allow exit of subcutaneous gases. In most instances, unfortunately, no specific treatment is available. Resuscitator malfunction could have been diagnosed earlier by detecting the increased resistance to inspiration and noting that humidified gases did not exit through the tracheal tube when manual pressure on the resuscitator was released at the end of inspiration.

Manual resuscitators that are currently available have one drawback. It is inconvenient to check that normal

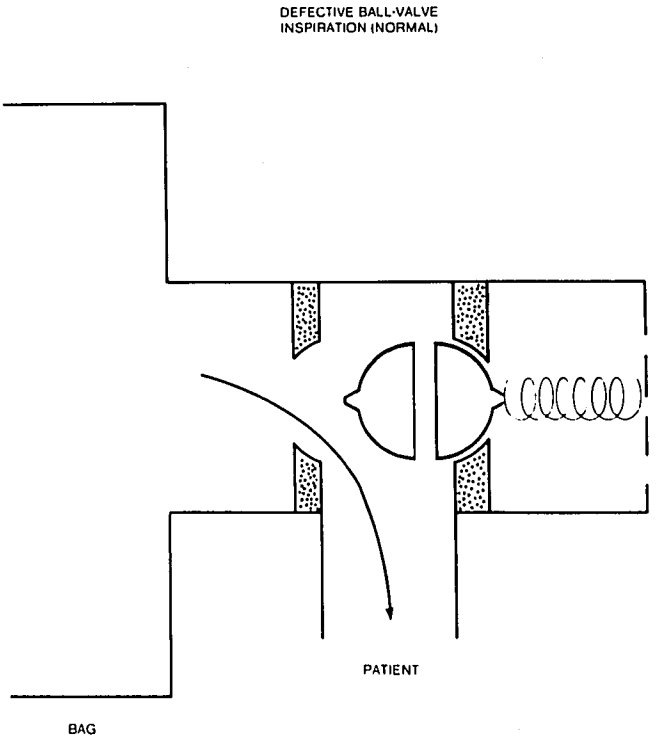

FIG. 1. Inspiration is possible in spite of a defective fractured ball-valve.

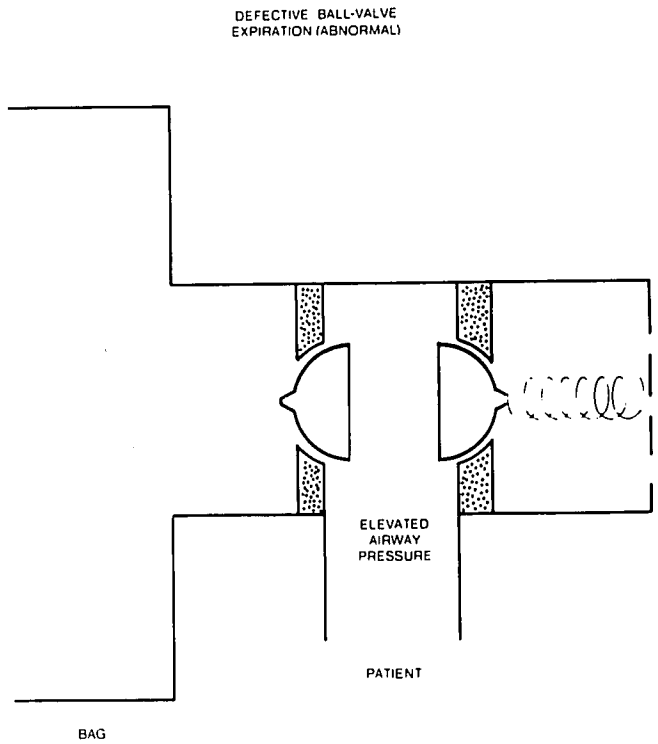

FIG. 2. Expiration is not possible when part of the fractured ball-valve occludes the expiratory port.

expiration can occur. One means of doing this is to attach an anesthesia bag to the patient port and inflate the bag until it has positive pressure at the end of inspiration. This patient illustrates that faulty manual respirators may appear normal and can result in complications. To the anesthesiologist it stresses the importance of carefully checking equipment before and during its use. Pulmonary barotrauma should be high on the list of differential diagnosis in cardiorespiratory emergencies.

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Appraisal of Epinephrine Administration to Patients under Halothane Anesthesia for Closure of Cleft Palate

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To achieve optimal local hemostasis in patients undergoing general anesthesia, epinephrine frequently is injected. During the closure of a cleft palate in pediatric patients, the injection of epinephrine is important in this regard. However, when halothane is concomitantly used, cardiac arrhythmias may occur.

Katz *et al.*[1] suggested that 1 μg/kg epinephrine is a safe dose for adults under halothane anesthesia. Johnstone *et al.*[2] demonstrated that 2.1 μg/kg was the dose producing premature ventricular contractions (PVCs) in 50% of patients (ED50) when epinephrine in saline was injected for hemostasis during halothane anesthesia. When 0.5% lidocaine was added to the injection solution, the ED50 increased to 3.7 μg/kg. Melgrave[3] and Wallbank[4] found that children could tolerate greater amounts of epinephrine on a body-weight basis than adults. They used from 2.5 to 5.5 μg/kg of epinephrine together with local anesthetics for pediatric patients under halothane anesthesia and found no arrhythmias.

The frequency of cardiac arrhythmias when epinephrine is used during halothane anesthesia in pediatric patients has not been determined. We, therefore, determined the incidence of cardiac arrhythmias when epinephrine was given together with halothane anesthesia for closure of cleft palate.

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Key words: Anesthetics, volatile: halothane. Anesthetics, local: lidocaine. Anesthesia: pediatric. Sympathetic nervous system: catecholamine epinephrine. Complications: arrhythmia.

MATERIALS AND METHODS

The subjects studied were 50 patients (27 boys and 23 girls) who underwent closure of a cleft palate. All the patients were considered to range in risk from ASA I to II. The age range was from 13 to 20 (15.9 ± 1.5 SD) months, and the body weight range was 8.4 to 14.5 (10.4 ± 1.1 SD) kg. Parental consent was obtained for each child. The study design was approved by a human experimentation committee.

The patients were premedicated with im injections of atropine, 0.25 mg, and hydroxyzine, 12.5 mg. Halothane was inhaled in 100% oxygen via a non-rebreathing system. The patients breathed spontaneously at all times. Halothane 4% from a factory-calibrated vaporizer was given for 10 min and orotracheal intubation was performed without use of a muscle relaxant. Anesthesia was maintained with 2.5% halothane. One per cent lidocaine with 1:100,000 epinephrine (LID-E) was injected by the surgeon. A dental block syringe with a 27-gauge needle was used for the LID-E injection. One milliliter of LID-E was administered every 30 s, and the volume of LID-E for closure of cleft palate was limited to 8 ml. The ECG was monitored continuously with an oscilloscope. If PVCs occurred during the injection, the injection was halted until the arrhythmia disappeared. After completion of the LID-E infiltration, we waited 5 min before starting surgery so that the effect of the epinephrine and lidocaine could be observed. The concentration of the inhaled halothane was gradually decreased to 0.7% during surgery, and the halothane discontinued 5 min before the end of surgery. The trachea was extubated when the patient regained consciousness.

RESULTS

The amount of LID-E used in this study varied from 6 to 11 (8.04 ± 1.06 SD) ml. The limit of 8 ml of LID-