

opsy and that she would probably be ventilated postoperatively again.

General anesthesia was induced and maintained by drugs with a limited duration of action in the hope that, at the completion of surgery, neuromuscular function would return and respiratory depression due to anesthetic agents would be minimized. We felt that every opportunity to re-establish spontaneous ventilation should be taken.

We believe that early extubation after the first operation was prevented by traumatic endobronchial suction and not by the anesthetics. Subsequent events resulting in exploration of the abdomen dictated the need for prolonged mechanical ventilation.

Central venous or pulmonary artery pressures were not monitored during the first anesthetic. The patient had good myocardial function as measured by the ejection fraction and the cardiac index, and the predicted blood losses and fluid shifts were not large. The possible major complication of such invasive monitoring would include a hemopneumothorax, a serious event in patients with limited respiratory reserve. When hemodynamic instability became apparent postoperatively, a central venous pressure line was indicated, and inserted.

In summary, we have described the anesthetic management of a patient with severe pulmonary disease, due to lymphangiomyomatosis, and a coagulopathy presenting for a vaginal hysterectomy and bilateral salpingo-oophorectomy.

Anesthesiology
70:550-553, 1989

Airway Management for Unilateral Lung Lavage in Children

BURT MCKENZIE, M.D.,* ROBERT E. WOOD, PH.D., M.D.,† ANN BAILEY, M.D.‡

Bronchopulmonary lavage (BPL) has been employed since 1965¹ for treatment of alveolar filling disorders such as alveolar proteinosis or extensive bronchial obstruction such as severe asthma. Because of the lack of double-lumen

endotracheal tubes of appropriate size, it has been difficult to perform such procedures in children. We report a simple and effective technique for unilateral lung lavage in children.

CASE REPORT

A 7-yr-old, 25-kg psychotic boy was admitted for evaluation of bilateral pneumonia and respiratory distress. Flexible bronchoscopy revealed large quantities of oil in his bronchi, which upon analysis proved to be olive oil. Because of respiratory distress and hypoxemia (oxygen saturation by pulse oximetry 84% on room air), we elected to perform large-volume BPL. This child was uncooperative, and general anesthesia was considered essential for the lavage. His size precluded use of a double-lumen endotracheal tube, and an alternate plan for airway management was needed.

* Resident in Anesthesiology.

† Professor of Pediatrics; Chief, Pediatric Pulmonary Medicine.

‡ Assistant Professor of Anesthesiology and Pediatrics.

Received from the Department of Anesthesiology and Pediatrics, University of North Carolina, Chapel Hill, North Carolina. Accepted for publication November 1, 1988.

Address reprint requests to Dr. Bailey: Department of Anesthesiology, CB 7101, Burnett Womack, University of North Carolina, Chapel Hill, North Carolina 27599-7010.

Key words: Anesthesia; pediatric. Lung; bronchopulmonary lavage. Ventilation: unilateral ventilation.

REFERENCES

1. Comin BA, Lisbow A, Fredman PJ: Pulmonary lymphangiomyomatosis: A review. *Am J Pathol* 79:348-382, 1975
2. Basset F, Soler P, Marsac J, Corrin B: Pulmonary lymphangiomyomatosis. *Cancer* 38:2357-2366, 1976
3. Dishner W, Blackburn J, Levin M: Pulmonary lymphangiomyomatosis. *Chest* 85:746-749, 1984
4. Chew AT, Nouri MS: Pulmonary and retroperitoneal lymphangiomyomatosis. *NY State J Med* 2:250-252, 1979
5. Adamson D, Henricks WL, Raybin DM, Raffin TA: Successful treatment of pulmonary lymphangiomyomatosis with oophorectomy and progesterone. *Am Rev Respir Dis* 132(4):916-921, 1985
6. Banner AS, Camryton CB, Emory W: Efficacy of oophorectomy in lymphangiomyomatosis in benign metastasizing leiomyoma. *N Engl J Med* 305:204-209, 1981
7. McCarty KJ, Mossler J, McLelland R, Sicke H: Pulmonary lymphangiomyomatosis responsive to progesterone. *N Engl J Med* 303:1461-1465, 1980
8. Monteforte WJ, Kohren PW: Angiomyolipomas in a case of lymphangiomyomatosis syndrome; Relationship to tuberous sclerosis. *Cancer* 34:317-321, 1974
9. Stovin PGL, Lumm CL, Flower CDR, Dauke CS, Beely M: The lung in lymphangiomyomatosis and in tuberous sclerosis. *Thorax* 30:497-509, 1975
10. Kitzsteiner K, Mallen R: Pulmonary lymphangiomyomatosis: Treatment with castration. *Cancer* 44:2248-2249, 1980
11. Kitzsteiner K, Mallen R: Pulmonary lymphangiomyomatosis. *N Engl J Med* 304:978, 1981
12. Oxorn DC, Landringan P: Anaesthetic management for oophorectomy in pulmonary lymphangiomyomatosis. *Can J Anaesth* 34(5):512-514, 1987
13. Kaplan JA, Ed.: *Thoracic Anaesthesia*. Churchill Livingstone, 1983, pp 146-148

The patient was premedicated with ranitidine. General anesthesia was induced with ketamine and maintained with isoflurane, oxygen, and atracurium. Noninvasive monitoring included end-tidal CO_2 and pulse oximetry. A 3.5-mm flexible bronchoscope was passed through a 4.5 mm (ID) cuffed endobronchial tube 50 cm long. The bronchoscope and tube were passed through a bronchoscope adapter attached to a tight-fitting anesthesia mask. Using the bronchoscope, the endobronchial tube was guided into the right main stem bronchus with its tip just above the right middle lobe bronchus; the bronchoscope was then withdrawn. The position of the tube was confirmed by auscultation of differential breath sounds after inflation of the cuff and by visual inspection with a 2.7-mm flexible bronchoscope. The endobronchial tube was attached to a Y-tube leading to bags of warm saline. While the patient was ventilated through the mask, the right middle and lower lobes were lavaged with aliquots (250–400 ml) of saline, to a total of 8 l, using gravity to fill and drain the lung.

During the procedure, the stomach gradually became distended with gas, causing progressive difficulty with ventilation. End-tidal pCO_2 rose by 15 mmHg and oxygen saturation fell to 70%. The procedure was interrupted to pass an orogastric tube for gastric decompression; this was necessary three times during the 60-min procedure. At the conclusion of the lavage, the patient was intubated with a 6.5 mm endotracheal tube, ventilated for several hours, and then extubated. Approximately 150 ml of oil was recovered in the lavage fluid. One week later the procedure was repeated to lavage the left lung. The endobronchial catheter was positioned with its tip just above the bifurcation of the main stem bronchus. Again, gastric distention required interruption of the procedure several times.

The patient improved significantly following the procedures. However, several weeks later he again presented with respiratory distress and hypoxemia, and another series of lavages was planned. Because of the problem with gastric distention during the previous procedures, a new technique was devised for management of the airway. Interruption of the procedure for gastric decompression was not only inconvenient but also risked dislodgement of the endobronchial catheter, with potentially serious results. Therefore, after induction of general anesthesia, a 28-Fr nasal airway was inserted into the right nostril and connected to a 6-mm ETT connector. A 14-Fr oral salem sump tube was passed into the stomach and connected to suction. The mouth was then sealed with Opsite®. The endobronchial tube was passed through the left nostril as before, and positioned in the right main stem bronchus (fig. 1). The tube was secured at the nostril with tape. Ventilation was then controlled *via* the nasal airway; minimal airway resistance was detected. The right lung was lavaged with 8 l of saline. Vigorous manual percussion of the ipsilateral hemithorax, especially during the drainage phase of lavage, appeared to result in a much increased yield of oil. During the procedure the oxygen saturation remained above 90%, and end-tidal pCO_2 remained less than 50 mmHg. As before, the patient was ventilated for several hours following the lavage.

This procedure was repeated for the left lung, and the child was admitted to the inpatient psychiatric service. Two months after discharge, he again appeared with respiratory distress and hypoxemia and required bilateral lavage. A fourth set of lavages were required 2 mo later. Following each set of lavages, oxygenation returned to normal and the chest radiograph cleared completely. A total of 150–250 ml of oil was recovered from each lavage. Following intensive intervention with the family, the child has remained symptom-free for the past 6 mo.

DISCUSSION

BPL is an uncommon procedure in children, usually performed for alveolar proteinosis. Partial cardiopul-

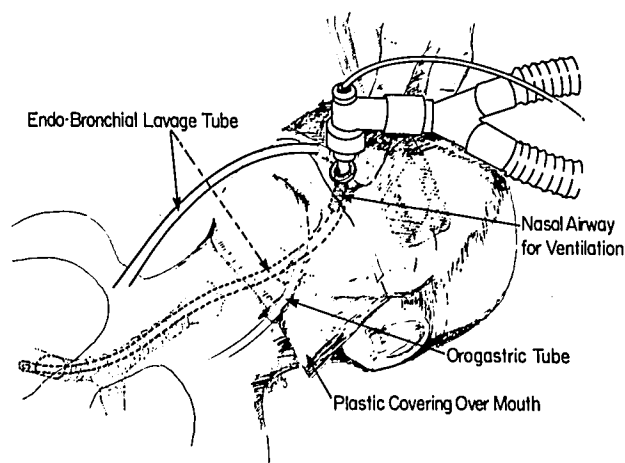


FIG. 1. Description of the airway circuit used in this case.

monary bypass has been employed, and, although allowing simultaneous bilateral lavage, has serious limitations, particularly when repeated procedures are required.^{2–4} Other techniques have been reported, usually employing small volumes of saline and isolation of the lungs by gravity. Either bronchoscopes⁵ or endotracheal tubes^{6,7} have been used for these procedures. Although such techniques may be useful in selected patients, significant morbidity (and even mortality) has resulted from failure to adequately isolate the lungs. An alternative technique is to lavage selected lung segments through a flexible bronchoscope.⁸ However, this technique is slow because the suction channel of such instruments is small (particularly the pediatric bronchoscope, with a channel only 1.2 mm in diameter), and only a single lobe or segment can be lavaged at a time. In one infant, a lung was isolated and lavaged using a Swan-Ganz catheter positioned in the main stem bronchus; ventilation was maintained through a rigid bronchoscope.⁹ Since 1975 one of the authors (R.E.W.) has performed BPL on children with alveolar proteinosis, using a bronchoscopically positioned, cuffed endobronchial tube. Typically, the procedure has been performed with the children sedated and spontaneously breathing around the endobronchial tube. This was the first patient to require a general anesthetic.

To achieve effective alveolar clearing, relatively large volumes of lavage fluid are needed. In our patient we found that volumes of at least 4–5 l were necessary; similar volumes were required in patients of similar size we have lavaged for alveolar proteinosis. Such volumes cannot be achieved without a large diameter catheter and effective isolation of the lavaged lung. The use of a long cuffed endobronchial tube has proved effective; the 4.5-mm ID tube we used allowed us to use aliquots of 250–400 ml at a rate of 1 l every 5–7 min. As has been reported with

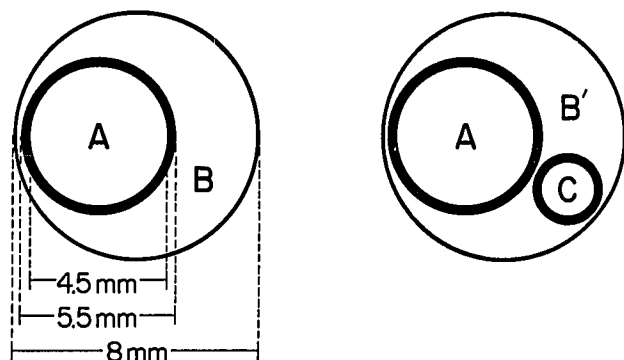


FIG. 2. Geometry of a tube within the trachea. The tracheal diameter is 8 mm, and that of the 4.5 mm ID endobronchial tube is 5.5 mm. The cross-sectional area of the endobronchial tube lumen (A) is 15.9 mm², while the cross sectional area of the trachea remaining for ventilation around the tube (B) is 26.3 mm². If the patient were to be ventilated through the 4.5 mm tube and a second tube (for lavage) were passed alongside, the largest internal cross-sectional area (C) the trachea could accommodate would be 1.76 mm², thus wasting 21.4 mm² of area (B) potentially available for ventilation.

alveolar proteinosis,¹⁰ vigorous chest percussion during the lavage greatly increases the efficiency of the lavage, even with oil (which appears in the effluent as an emulsion).

The choice of whether to lavage the right or left lung first is arbitrary and depends on which area of the lung is most involved with the pathologic process being treated. At least in theory, placement of the endobronchial catheter in the right main bronchus with its balloon cuff below the right upper lobe bronchus could allow ventilation of the right upper lobe as well as the entire left lung. Therefore, when the disease is uniformly distributed, we choose this area first.

The success of this technique depends on several factors. First, the endobronchial catheter must be positioned accurately; the flexible bronchoscope greatly facilitates this. The catheter must remain fixed in the proper position; this is facilitated by neuromuscular blockade. Vigilance in monitoring breath sounds and the position of the tube are necessary because if the tube were to slip proximally, or the balloon cuff fail, fluid could flood both lungs. Should this occur, however, the lungs could be rapidly suctioned through the endobronchial tube.

A second requirement is that the endobronchial catheter must be large enough to allow effective lavage yet small enough to allow effective ventilation around it. We used a 4.5-mm ID tube and positioned it with a 3.5-mm flexible bronchoscope. Smaller tubes could be used with smaller flexible bronchoscopes. The geometry of a cylinder within a cylinder is such that there is much more cross-sectional area available for ventilation around a sin-

gle tube than would be available through two parallel tubes (fig. 2). Thus, airway resistance is reduced and effective ventilation is maintained. Furthermore, even the use of two concentric tubes (such as a catheter passed through a rigid bronchoscope) would result in a significant reduction of the cross-sectional area available for ventilation. Although the presence of another tube would help ensure patency of the glottis, neuromuscular blockade accomplishes the same result. The adequacy of ventilation and oxygenation may be confirmed by auscultation, pulse oximetry, and capnometry.

Another requirement is that the saline must be delivered to the lung in sufficient volumes and with sufficient pressure to fill and distend the airways and alveolar spaces. It seems essential that the fluid be instilled through a catheter or tube with its tip effectively sealed around the airway (*i.e.*, with an inflatable cuff). Finally, as illustrated by the problems with the first two procedures, it is essential to provide for gastric decompression because the stomach is likely to become distended during positive pressure ventilation of the oropharynx. An orogastric tube with constant suction easily averts this problem, while sealing the mouth eliminates loss of airway pressure.

Our technique has some limitations. It is not possible to include the right upper lobe in the lavage because if the balloon cuff is placed proximal to the right upper lobe, it will slip into the trachea. An appropriately designed cuffed tube must be available.

In summary, we describe a technique for unilateral lung lavage in children under general anesthesia. Adequate lung isolation and controlled ventilation are accomplished by lavage through a bronchoscopically positioned endobronchial tube, while ventilating the contralateral lung *via* a modified nasal airway.

The authors thank Linda Williams for her assistance in preparing the manuscript.

REFERENCES

1. Ramirez RJ, Kieffer RF, Ball WC: Bronchopulmonary lavage in man. *Ann Intern Med* 63:819-828, 1965
2. Seard C, Wasserman K, Benfield JR, Cleveland RJ, Costley DO, Heimlich EM: Simultaneous bilateral lung lavage (alveolar washing) using partial cardiopulmonary bypass. *Am Rev Respir Dis* 101:877-884, 1970
3. Lippman M, Mok MS, Wasserman K: Anesthetic management for children with alveolar proteinosis using extracorporeal circulation. *Br J Anaesth* 49:173-176, 1977
4. Hiratzka LF, Swan DM, Rose EF, Ahrens RC: Bilateral simultaneous lung lavage utilizing membrane oxygenator for pulmonary alveolar proteinosis in an 8-month-old infant. *Ann Thorac Surg* 35:313-317, 1983
5. Mayer B: *Pediatric Anesthesia, A Guide to Its Administration*. Philadelphia, J. B. Lippincott, 1981, pp 158-159
6. Reas HW, Hackett PR: A technique for tracheobronchial lavage under general anesthesia. *Dis Chest* 53:605-612, 1968

7. Spock A: State of the art of lung lavage in patients with cystic fibrosis. 1000 Years of Cystic Fibrosis. Edited by Warwick WJ. University of Minnesota, Minneapolis, Minnesota, 1981, pp 113-117
8. Brach BB, Harrell JH, Moser KM: Alveolar proteinosis. Lobal lavage by fiberoptic bronchoscopic technique. *Chest* 69:224-227, 1976
9. Moazam F, Schmidt JI, Chesrown SE, Graves SA, Sauder RA, Drummond J, Heard SD, Talbert JL: Total lung lavage for pulmonary alveolar proteinosis in an infant without the use of cardiopulmonary bypass. *J Pediatr Surg* 20:398-401, 1985
10. Kao D, Wasserman K, Costley DD, Benfield JR: Advances in the treatment of pulmonary alveolar proteinosis. *Am Rev Respir Dis* 111:361-363, 1975

Anesthesiology
70:553-555, 1989

Succinylcholine-induced Hyperkalemia in a Patient with Metastatic Rhabdomyosarcoma

LAURA G. KRIKKEN-HOGENBERK, M.D.,* JAN R. DE JONG, M.D.,†
JAMES G. BOVILL, M.D., PH.D., F.F.A.R.C.S.I.‡

Severe hyperkalemia following administration of succinylcholine, resulting in cardiac arrhythmias and cardiac arrest, occurs in many conditions including burns, severe trauma, neurologic disease, and certain types of neuromuscular disorders.^{1,2} A feature common to many of these conditions is skeletal muscular involvement either due to direct trauma or muscle denervation. We report a case in which hyperkalemia and ventricular arrhythmias developed after administration of succinylcholine to a patient with extensive metastatic rhabdomyosarcoma.

REPORT OF A CASE

A 4-yr-old, 18-kg boy was admitted because of severe general malaise of 3 wk duration. On the day before admission he had developed pyrexia (38.9° C). Apart from an uneventful operation for strabismus at the age of 3 yr, there was no other relevant medical history. No details of the anesthetic given for that operation were available. Physical examination on admission showed an ill child. The only relevant finding was a temperature of 39.6° C. No focus of infection could be identified, nor was there any evidence of neurologic or muscular disorder. A radiologic skeletal survey showed widespread osteolytic areas in multiple areas. Nineteen days after admission, a tibial bone biopsy was scheduled under general anesthesia. Between admission and surgery, his temperature had remained elevated around 39° C. Prior to surgery the hemoglobin was 5.3 mmol/l (8.2 mg/dl), the hematocrit 0.27, the leukocyte count was $10.2 \times 10^9/l$ with 23% metamyelocytes. Serum electrolytes and creatinine were within normal limits. The potassium was 4.2 mmol/l and had varied between 3.4 and 4.2 mmol/l between

admission and surgery. The lactic dehydrogenase (LDH), which had been 4,110 U/l on admission, had decreased to 1,960 U/l 3 days prior to surgery, with a distribution of LDH isoenzyme fractions 1-5 of 18%, 54%, 24%, 5%, and 0%, respectively. The normal range for LDH isoenzymes fractions in our laboratory is 1, 15-26%; 2, 34-44%; 3, 23-33%; 4, 5-12%; and 5, 1-7%, respectively. The distribution pattern in our patient is not characteristic for a particular type of tissue damage.

Premedication consisted of 40 mg of oral trimeprazine. Anesthesia was uneventfully induced with halothane in O_2/N_2O . Monitoring in the induction room consisted of ECG registration and arterial blood pressure measurement by oscillometry. After induction of anaesthesia, a 20-g iv cannula was inserted, and 0.05 mg of atropine and 20 mg of succinylcholine were given iv. The trachea was then intubated and normal breath sounds heard over both lungs. During induction the ECG showed a sinus tachycardia of 160 beats/min. Just before transport to the adjacent operating room, approximately 3 min after administration of the succinylcholine, the QRS complexes were noted to have widened, and shortly thereafter the ECG showed a ventricular tachycardia with a rate of 170 beats/min (fig. 1). The systolic arterial blood pressure was 70 mmHg. Nitrous oxide and halothane (at that moment the inspired halothane concentration was 1.5%) were discontinued, and ventilation was controlled with an FI_{O_2} of 1.0. Soon after, a ventricular flutter-like pattern developed, arterial pulsations became undetectable, and external cardiac massage was started. Lidocaine (20 mg iv) was administered. The ventricular flutter changed to ventricular fibrillation. At that moment the pupils were widely dilated. After an additional 20 mg of lidocaine iv and defibrillation with an energy of 100 J, the ventricular rhythm followed with a rate of 65 beats/min and detectable peripheral arterial pulsations. Atropine (0.05 mg iv) was given, the heart rate increased, and ventricular flutter again resulted with a rate of 260 beats/min. This reverted to a sinus tachycardia of 150 beats/min after synchronized cardioversion with an energy of 100 J. The pupils narrowed and shortly thereafter the patient regained consciousness.

An arterial blood sample obtained approximately 9 min after the administration of succinylcholine showed serum potassium 7.3 mM, pH 7.35, P_{aCO_2} 4.2 kPa (31.5 mmHg), P_{aO_2} 50.5 kPa (379 mmHg), bicarbonate 20 mM, base excess -5 mmol/l, and O_2 saturation 100%. An infusion of 100 ml glucose 10% containing 2 units of insulin was started. Because the vital signs remained stable and no new arrhythmias occurred, it was decided to proceed with the biopsy. Anesthesia was recommenced with O_2/N_2O /enflurane and spontaneous respiration via a Jackson Rees circuit. In addition to a bone biopsy, a muscle biopsy

* Staff Anaesthesiologist.

† Resident in Anaesthesiology.

‡ Professor of Anaesthesiology.

Received from the Department of Anaesthesiology, University Hospital Leiden, Leiden, The Netherlands. Accepted for publication November 10, 1988.

Address reprint requests to Dr. Krikken-Hogenberk: Department of Anaesthesiology, University Hospital Leiden, P. O. Box 9600, 2300 RC Leiden, The Netherlands.

Key words: Complications: hyperkalemia; ventricular dysrhythmia. Neuromuscular relaxants: succinylcholine.