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Treatment of acute unilateral lung disease with differential lung intubation followed by asynchronous independent lung ventilation

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Introduction

For management of patients with unilateral lung disease independent lung ventilation is becoming popular since it can overcome most of the problems associated with conventional ventilation such as regional alveolar overdistention, increased ventilation-perfusion mismatching, venous admixture, and barotrauma [1–3]. Furthermore, applying selective positive end-expiratory pressure (PEEP) to the injured lung, one can selectively improve its volume at end expiration (i.e., its functional residual capacity) and to decrease the overall shunt fraction [4].

We had successfully applied asynchronous independent lung ventilation (AILV) in one patient with acute unilateral lung disease, in whom conventional ventilatory treatment methods failed to improve his clinical condition.

Case report

A 76-year-old man involved in a road accident was brought to the emergency room of the Hillel Yaffe Medical Center. On admission, he was disoriented, agitated and pale, tachypneic (30 breaths·min^{-1}), tachycardic (125 beats·min^{-1}) and with arterial blood pressure of 150/110 mmHg. The physical examination revealed decreased air entry over the left hemithorax and muffled heart sounds.

Endotracheal intubation was immediately performed, and a chest drain was inserted in the left hemithorax, anterior to the midclavicular line [5], releasing a large amount of air and a small quantity of blood. Two 16-gauge peripheral catheters were placed in the arm veins, and fluid replacement was promptly initiated.

The laboratory data were as follows: Hemoglobin 12 g%, Ht 38%, WBC 9000/mm^2. Glucose 170 mg%, BUN 10 mg%, Na^+ 140 mEq·l^{-1}, K^+ 6.2 mEq·l^{-1}. The arterial blood gases were: pH 7.15; $Paco_2$ 20 mmHg; Pao_2 60 mmHg (FIo_2 0.6); Bicarbonate 14 mEq·l^{-1}.

The diagnostic peritoneal lavage was positive, and a splenectomy was performed. During surgery, the estimated blood loss was about 2500 ml, and fluid replacement consisted of 4000 ml of Hartman's solution and 1500 ml of whole blood. The measured urinary output was 100 ml. At the end of surgery, the patient was transferred to the postanesthetic care unit, and connected to an MA 1 ventilator (Puritan Bennet, CA, USA) provided with an IMV low-resistance continuous-flow external device (IMV Assembly, Intersurgical, Middlesex, UK). He was hemodynamically stable, with the following laboratory results: Hb 8.7 g%, Ht 27%, Na^+ 133 mEl·l^{-1}, K^+ 5.2 mEq·l^{-1}, Ca^{++} 8/9 mg%, BUN 7 mg%, Creatinine 1 mg%, pH 7.30, $Paco_2$ 30 mmHg, Pao_2 150 mmHg (FIo_2 0.4) and HCO_3^- 18 mEq·l^{-1}. The chest roentgenogram showed a chest drain in place and multiple fractures of ribs over the left hemithorax.

An epidural catheter was inserted at the T5/T6 level and plain 0.25% bupivacaine was continuously infused at a rate of 8 ml/h for postoperative analgesia [6]. Artificial ventilation was maintained for the next 4 days with a gradual reduction of the IMV frequency, and with achievement of spontaneous breathing and maintenance of good exchange parameters [oxygen saturation

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(Sa_{O_2}) ≥ 90% at FI_{O_2} of 0.21 with spontaneous breathing on 5 cmH_2O constant positive airway pressure (CPAP)] [7], was extubated and the chest drain was removed.

On the following day, he was transferred to the surgical ward, presenting acceptable blood gas results (pH 7.37, Pa_{CO_2} 37 mmHg, Pa_{O_2} 75 mmHg at room air, HCO_3^- 21 $mEq \cdot l^{-1}$) and good clinical parameters (quiet respiration with a rate of 22 $breaths \cdot min^{-1}$).

Seventy-two h later, he suddenly became agitated, extremely tachypneic (48 $breaths \cdot min^{-1}$), with cyanosis and profuse sweating. Orotracheal intubation was immediately performed, and the patient was admitted to the respiratory care unit.

Controlled mechanical ventilation was started using the Erika Ventilator (Engstrom Medical, Bromma, Sweden), with a FI_{O_2} of 1. The registered compliance was then 10 $ml \cdot cmH_2O^{-1}$. On physical examination, no air entry was detected over the left hemithorax and the chest radiogram revealed massive collapse of the left lung (Fig. 1).

Under sedation with midazolam and muscular relaxation with atracurium, a 7-mm flexible fiberoptic bronchoscope was introduced through an adequate swivel connector (Portex, Kent, UK) adapted to the orotracheal tube to release the bronchial obstruction. However, a marked decrease in the pulse oxygen saturation from 96% to 80%, despite administation of 100% oxygen, and frequent multifocal premature beats, led to interruption of the procedure. A new chest roentgenogram showed no improvement (Fig. 2).

The orotracheal tube was then changed by a left model double-lumen tube, inner diameter 5.4 mm each

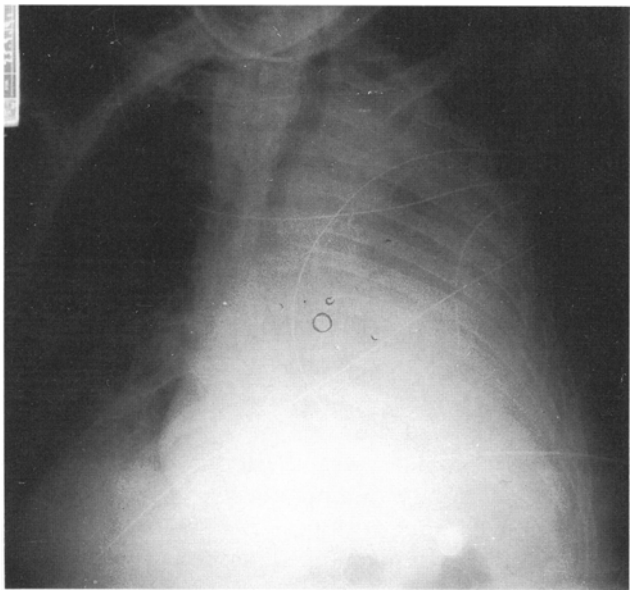

Fig. 1. Chest radiogram showing massive collapse of the left lung

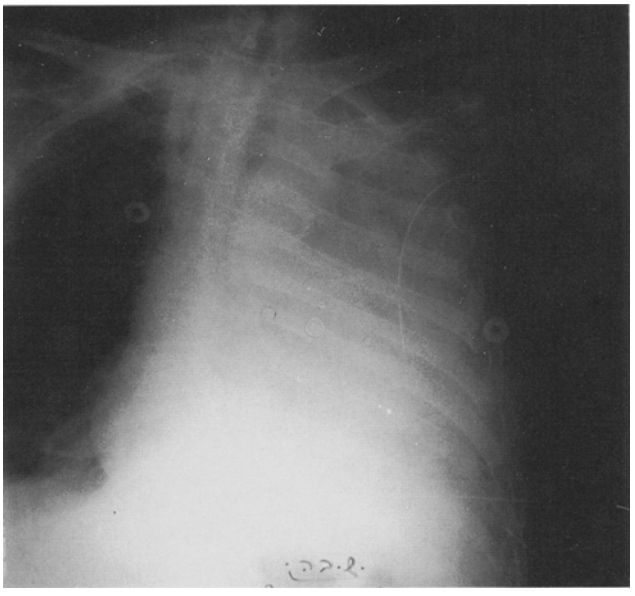

Fig. 2. No improvement after fiberoptic bronchoscopy

lumen, (Broncho-Cath, Mallinkrodt, Northampton, UK), and following assurance of adequate positioning of the tube, as proposed by Benumof and Alfery [8], the right lung was ventilated with 500 ml tidal volume and a frequency of 14 $breaths \cdot min^{-1}$ with 5 cmH_2O positive end expiratory pressure. Through the left lumen, aggressive tracheobronchial toilette was instituted, with repeated infusion of 10 ml of normal saline, selective suction and manual inflation of the left lung. During the procedure, the pulse oxymetry was 95%, despite an FI_{O_2} of 0.5.

As soon as some air entry was detected over the left hemithorax, the left lung was connected to a second ventilator and ventilated with similar parameters to the contralateral lung, but with a positive end-expiratory pressure of 10 cmH_2O.

The pulse oxygen saturation increased to 98% and the FI_{O_2} was then decreased to 0.4. The arterial blood gas results were as follow: pH 7.39; Pa_{CO_2} 36 mmHg; Pa_{O_2} 160 mmHg; bicarbonate 20 $mEq \cdot l^{-1}$.

Twenty-four h later, the patient's condition allowed us to change the double-lumen tube to a regular one, and after a few hours, when breathing spontaneously through 5 cmH_2O at a FI_{O_2} of 0.21 and a pulse oxygen saturation of 96%, extubation was performed. The chest roentgenogram showed complete resolution of the atelectasis (Fig. 3).

Discussion

Mechanical ventilation with or without positive end-expiratory pressure is widely used in the treatment of

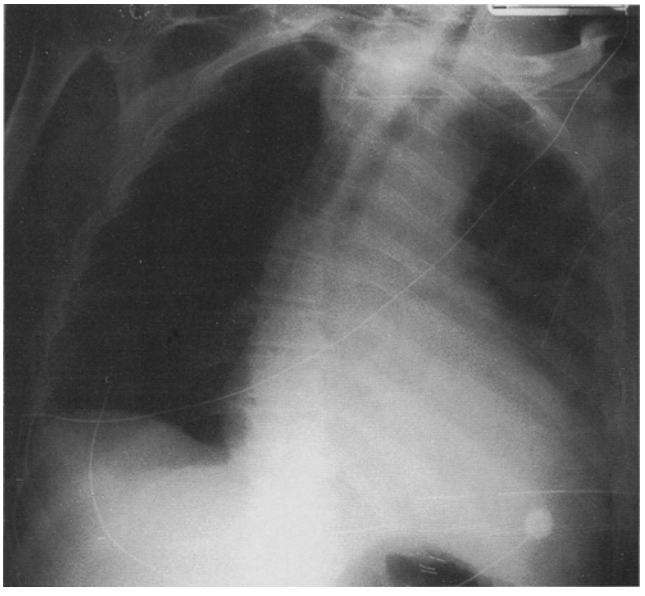

Fig. 3. Resolution of the atelectasis after Asynchronous Independent Lung Ventilation (AILV)

ventilatory failure. However, in the presence of unilateral lung disease with different lung compliances, the delivered tidal volume may diverge toward the more compliant lung, with consequent overdistention which increases the vascular resistance and shifts the blood flow to the stiffer lung [3]. Since the healthier lung is usually more compliant and the more affected lung is less so, the perfusion is forced away from the healthier to the sicker parenchyma. This may increase the ventilation-perfusion mismatching, leading to a decrease in arterial blood oxygenation [9].

Lung collapse following chest trauma is a frequent complication, even without evidence of bronchial occlusion [10]; this is related to the decreased alveolar tension as a consequence of decreased amounts of surfactant substance [11]. In addition, pain limits the patient's ability to cough effectively, which may lead to accumulation of tracheobronchial secretions, inducing bronchial plugging and atelectasis [12].

Conventional treatment of lung atelectasis involves adequate pain relief, antibiotics, physiotherapy and postural drainage [13].

Some authors have recommended the use of fiberoptic bronchoscopy for patients with massive symptomatic lung collapse, who lack central air bronchograms and fail to respond or tolerate 48 h of adequate respiratory physiotherapy [14]. However, fiberoptic bronchoscopy may induce airway trauma, cardiac arrhythmias, and may even worsen the arterial oxygenation [13].

In fact, we noted in our patient a profound hemoglobin desaturation, despite the administration of 100% oxygen, associated with multifocal premature ventricular contraction during fiberoptic bronchoscopy, which led to interruption of the procedure.

Independent lung intubation allowed selective bronchial lavage and suctioning releasing the obstructed bronchus, while oxygenation was preserved during the procedure. Moreover, further asynchronous independent lung ventilation maintained good aeration of the affected lung with good oxygenation, despite the decrease in the delivered oxygen concentration.

In conclusion, differential lung intubation followed by asynchronous independent lung ventilation represents a real improvement in the methodology of ventilatory therapy, and may be useful in dangerous hypoxic situations caused by unilateral lung disease when an adequate level of oxygenation is not achieved by conventional modes of ventilation.

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