Open lung in ARDS

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Every year, millions of patients worldwide receive ventilator support during surgery. Mechanical ventilation has become an important therapy in the treatment of patients with impaired pulmonary function and particularly in patients suffering from adult respiratory distress syndrome (ARDS). ARDS is caused by multiple factors and is characterized by respiratory dysfunction including hypoxemia and decreased lung compliance. It is known that the decrease in lung distensibility is due to a disturbed surfactant system with an elevated surface tension. This increase in surface tension leads to an increase in forces acting at the air-liquid interface, resulting finally in end-expiratory collapse, atelectasis, an increase in right-to-left shunt and a decrease in p_{aO_2}.

VENTILATOR ASSOCIATED LUNG INJURY (VALI)

It has become clear, however, that mechanical ventilation can attenuate lung damage and may even be the primary factor in lung injury. The recent recognition that alveolar overdistension rather than high proximal airway pressures is the primary determinant for lung injury (ie, volutrauma instead of barotrauma) combined with shear stress evoked by repeated alveolar collaps and re-opening due to low end-expiratory volumes [1], has led to renewed interest in lung mechanics and ventilation. When ARDS patients are ventilated in a volume-controlled mode there is a risk for overdistension in non-collapsed parts of the lungs. If one assumes that 75 % of the lung is consolidated and only 25 % is ventilated, then ventilation even with small tidal volumes, *eg*, 7 mL/kg body weight, would result in tidal volumes of 28 mL/kg in the ventilated regions, with a danger of overdistension and further lung impairment. Use of pressure-controlled time-cycled modes of mechanical ventilation, in which the alveolar pressure can never exceed the peak inspiratory pressure set on the ventilator is, therefore, preferable.

In the recent international consensus conference on ventilator-associated lung injury (VALI) in ARDS[2] the question was asked what nonpharmacological approaches are currently available for prevention of VALI?

Clinical studies showed that a reduction of tidal volume, reduction of peak airway pressures combined with an increase in positive end-espiratory pressure (PEEP) resulted in improved outcome of ARDS patients [3]. Furthermore by applying a lung protective strategy pro-inflammatory mediators both in the lung as well as those circulating can be reduced[4], and reducing the circulating levels of pro-inflammatory mediators reduces the development of multi-oragn failure, the major cause of mortality in ARDS patients[5]. This lung protective strategy was already proposed in 1992 in our editorial entitled: “Open up the lung and keep the lung open”[6].

The implied rationale, however, is a matter of debate: Why should we “open the lung”? What is an “open lung”? In addition, questions concerning the methodology were asked: How can we “open the lung” and how can we keep the lung open with the least possible side-effects?

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WHAT IS AN OPEN LUNG AND WHY SHOULD IT BE OPENED?

When a lung is "open" it is characterized by an optimal gas exchange[6] and a low rate of intrapulmonary shunting (ideally less than 10 %) corresponding with a p_{aO_2} of more than 450 mmHg on pure oxygen[7]. At the same time, airway pressures are at the minimum that ensure the required gas exchange; hemodynamic side-effects are thus minimized[6].

HOW CAN WE OPEN THE LUNG?

To recruit the collapsed alveoli to improve gas exchange a high opening pressure is needed. The rationale behind the high opening pressure to recruit the lung and the need for lower pressures to keep the alveoli open can be deduced from the pressure-volume curve of an individual alveolus (Fig 1). The behavior of an alveolus is quantal in nature; it is either open or closed[8]. A critical opening pressure has to be reached before previously collapsed alveoli can be opened. Once open, alveoli remain open until the pressure drops below a critical level and immediate collapse occurs. Re-opening again requires the high recruiting pressure. Any state between open and closed is unstable and impossible to maintain. After opening of the alveoli, they should be kept open by using a ventilator setting which will keep the pressure above the critical closing pressure of the alveolus, ie, with a sufficient high PEEP level. Because the alveoli are open during the whole ventilation period no collapse of alveoli occurs, reducing shear forces to a minimal level.

In summary there are three steps to open the lung[9].

1. A critical opening pressure must be overcome during inspiration

2. This opening pressure must be maintained for a sufficiently long period of time

3. During expiration, no critical time that would allow closure of lung units should pass, by using auto or intrinsic PEEP or applying sufficiently high PEEP levels which prevent alveolar collapse.

OPEN LUNG MANAGEMENT

"The open lung management" describes the steps and methods used to safely open the lung and how to keep it open. Fig 2 shows the predetermined sequence of therapeutic phases, each with its specific treatment objective[6,10]. As shown in Fig 2, the goal of the initial increase in inspiratory pressure is to recruit collapsed alveoli and to determine the critical opening pressure. Then, the minimum pressures that prevent the lung from collapse are determined. Finally, after an active re-opening maneuver sufficient pressure is implemented to keep the lung open.

After opening the lung and finding the lowest pressure to keep it open, the resulting pressure amplitude is minimized and at the same time pulmonary gas exchange is maximized. A reduction of the total level of support is generally possible after a successful alveolar

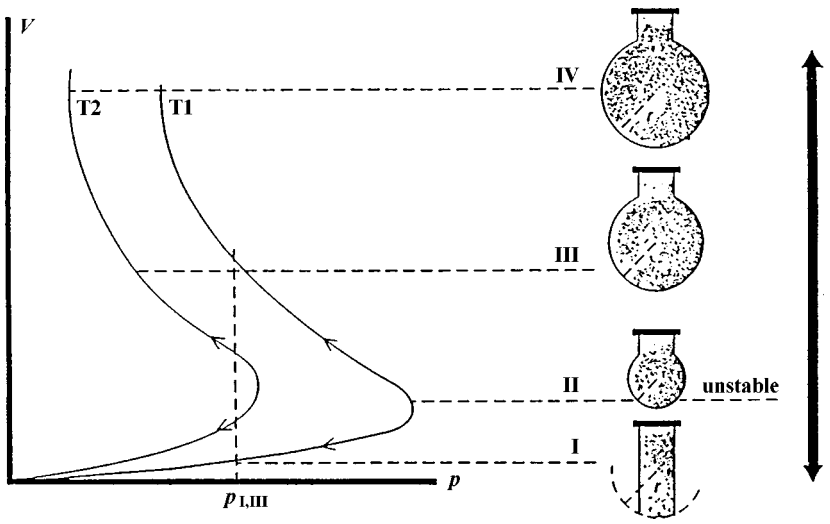

Fig 1. Physiological behaviour of the alveolus. The pressure-volume (*p*-*V*) relation is shown on the *X*-*Y* axes. The right side shows the status of the broncho-alveolar unit: its radius(r) reflects the *p*-*V* relation (I-IV). Surface tension in pathological (T1) and normal conditions (T2) is shown. The arrows indicate the direction from closed (bottom) to open (top) states and *vice versa.*

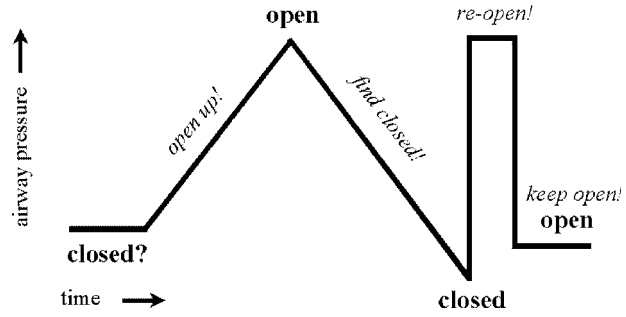

Fig 2. Schematic representation of the opening procedure for collapsed lungs. Note: the imperatives (!) mark the treatment goal of each specific intervention. The bold **words mark the achieved state of the lung. At the beginning the precise amount of collapsed lung tissue is not known.**

recruitment.

Should a renewed collapse of alveoli occur, often caused by intrapulmonary suction or disconnection, a fall in p_{aO2} indicates that a re-opening maneuver has to be performed in the same way as previously described.

FIRST RESULTS OF THE OPEN LUNG MANAGEMENT

In one of the first clinical, prospective controlled randomized studies with the "open lung approach", Amato and co-workers showed that a ventilation strategy with permissive hypercapnia resulted in a higher weaning rate from mechanical ventilator, a lower rate of barotrauma, and an improved 28-d survival in ARDS patients, compared with conventional ventilation[3].

The NIH ARDS multicenter, randomized trial compared traditional ventilation treatment, which involved an initial tidal volume of 12 mL per kilogram with ventilation with a lower tidal volume of 6 mL per kilogram of predicted body. The trial was stopped after the enrollment of 861 patients because mortality was lower in the group treated with lower tidal volumes than in the group treated with traditional tidal volumes (31.0 percent *vs* 39.8 percent), and the number of days without ventilator use during the first 28 days after randomization was greater in this group [(mean±SD), (12±11) *vs* (10±11)][11].

Data from our group suggest that early application of the open lung management in animals suffering from ARDS prevents a decrease in pulmonary compliance compared with animals ventilated in settings that do not open the lung[12]. Therefore, application of "the open lung management" should be used in each patient needing mechanical ventilation and thus minimizing VALI in patients without compromising optimal ventilation therapy.

Recently Shreiter *et al* ventilated 32 polytraumatized patients suffering from severe chest contusion according to the Open Lung Concept (OLC)[13]. Oxygenation improved significantly; p_{aO_2}/F_{iO_2} rose from 134 mmHg before start of the OLC to 522 mmHg after the recruitment procedure. For the recruitment procedure, a mean PIP of 65 mbar=cmH_2O was required, and the recruited alveoli were kept open by a total-PEEP of 22 mbar[13]. After the recruitment procedure, PIP and F_{iO_2} could be reduced, resulting in tidal volumes of 3.5 mL per kg bodyweight. Only two patients (6.25 %) died of extrapulmonary causes[13].

RECOMMENDATIONS

To minimize the effects of ventilation-induced lung injury, practical guidelines must be followed:

1 Always use a pressure-controlled ventilator setting. When ventilating in a pressure-controlled mode the risk of overdistension of healthy parts of injured lung areas (as present in inhomogenous lung injury like ARDS) is prevented.

2 Use sufficiently high levels of PEEP to prevent end-expiratory collapse and the ensuing shear forces, which will further impair lung function. Also, sufficiently high levels of PEEP can help to prevent further loss of surfactant in still healthy alveoli, halting the further spread of the disease process. Minimizing hemodynamic deterioration provides sufficient volume substitution.

3 Use as small as possible tidal volumes, again to prevent overdistension and shear forces.

SUMMARY

The basic treatment principles are:

- Open up the whole lung with the required inspiratory pressures

- Keep the lung open with PEEP levels above the closing pressures

- Maintain optimal gas exchange at the smallest possible pressure amplitudes to optimize CO_2 removal.

With the strict application of these principles, a prophylactic treatment is available, that is aimed at preventing ventilator-associated lung injury and pulmonary complication without compromising optimal ventilation.

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