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Methods and devices

Tracheal and alveolar gas composition during low-frequency positive pressure ventilation with extracorporeal CO_2-removal (LFPPV-ECCO$_2$R)

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Abstract. Tracheal and alveolar gas composition was studied by mass spectrometry in a patient with severe ARDS treated by low frequency positive pressure ventilation/extracorporeal CO_2-removal (LFPPV-ECCO$_2$R). Measured alveolar gas concentrations were compared with values derived from standard respiratory equations. As a result we found that during LFPPV-ECCO$_2$R with a constant endotracheal O_2-flow, alveolar gas composition cannot be predicted reliably from standard equations. The reasons for this finding are discussed. We conclude that monitoring of alveolar gas composition by mass spectrometry is of great value during LFPPV-ECCO$_2$R if P_AO_2, $P_{(A-a)}O_2$ and $\dot{Q}_{va}/\dot{Q}_t$ are to be determined correctly.

Key words: ARDS – Alveolar gas composition – Low frequency positive pressure ventilation with extracorporeal CO_2-removal – Mass spectrometry

Low frequency positive pressure ventilation combined with extracorporeal CO_2-removal (LFPPV-ECCO$_2$R) is a new type of treatment for severe adult respiratory distress syndrome (ARDS) not responding to conventional treatment. Its aims and techniques have been previously defined both experimentally and clinically by Kolobow et al. and Gattinoni et al. [5 – 11].

By removing part of the body's CO_2-production with partial veno-venous prepulmonary bypass through a membrane lung (ML), the necessity for alveolar ventilation is reduced, enabling the use of mechanical ventilation with respiratory rates as low as 0.66 min^{-1} [5]. Oxygenation is also effected by these mechanical lung inflations as well as by a continuous flow of pure oxygen which is given directly into the trachea to avoid alveolar hypoxia during apnea. Thus, O_2-uptake and CO_2-removal are dissociated during LFPPV-ECCO$_2$R. As the reduced lung ventilation during LFPPV-ECCO$_2$R is accompanied by lower peak and mean airway pressures in comparison with conventional mechanical ventilation, it is speculated that LFPPV-ECCO$_2$R not only lessens the risk of barotrauma but also improves the patient's hemodynamic state [6]. Finally LFPPV-ECCO$_2$R may prove superior in terms of allowing better healing of the diseased lungs [8]. While this concept seems sound we found it difficult to predict reliably alveolar PO_2 (P_AO_2) during LFPPV-ECCO$_2$R with a supplemental O_2-flow on the basis of the conventional alveolar gas equation [13] which requires a steady-state gas exchange as a precondition for application. P_AO_2 however must be known to calculate the alveolar-arterial partial pressure difference for oxygen $P_{(A-a)}O_2$ and venous admixture ($\dot{Q}_{va}/\dot{Q}_t$) both of which serve as important parameters of lung function during treatment of ARDS.

In a patient who was treated with LFPPV-ECCO$_2$R because of severe ARDS we studied tracheal and end-tidal gas composition by mass spectrometry in order to determine the influence of low frequency mechanical ventilation and supplemental endotracheal O_2-flow.

Patient, material and methods

The case

A 15-year-old boy was transferred to our hospital 4 days after two episodes of aspiration following surgery for a peritonsillar abscess. During this time fulminant respiratory failure had developed requiring CMV with PEEP up to 25 cmH_2O and a F_IO_2 of 0.8 to 1.0 to maintain a P_aO_2 above 60 mmHg.

On admission his venous admixture was calculated to be 45 percent according to the Berggren formula [1]. In this situation LFPPV-ECCO$_2$R (respiratory rate: 3–5 min^{-1}) was instituted using two 4.5 m^2 spiral core membrane lungs (Sci-Med, Minneapolis, Minnesota) arranged in series and ventilated with warm humidified room air at subatmospheric pressure. Venous drainage and return (blood flow: 0.8–1.2 l · min^{-1}) was carried out with a double lumen cannula previously described by Pesenti [12]. A supplemental flow of pure humidified oxygen was directly fed into the trachea by means of a small tube advanced from a side port of the y-piece. Pressure limited CMV (Servo 900 C, Siemens-Elema) was used for LFPPV with a PEEP of 17 to 25 cmH$_2$O being produced by an underwater seal.

During the first days lung function seemed to improve as judged by a diminished $P_{(A-a)}O_2$ and the F_IO_2 set and measured at the ventilator could be reduced to 0.3.

Sampling tracheal and end-tidal gas with a mass spectrometer however revealed that P_AO_2 was much higher than expected on the basis of the F_IO_2 set at the ventilator. This was most probably due to a relatively high supplemental O_2-flow. At this point analysis of tracheal gases by mass spectrometry was introduced as a monitoring technique.

LFPPV-ECCO$_2$R had to be stopped after 10 days of bypass due to infection and bleeding problems at the cannulation site without any improvement in lung function. Seven days after termination of bypass the patient died due to hypoxemia and respiratory acidosis.

Measurements

Fractional dry gas concentrations were measured with a mass spectrometer (Perkin-Elmer MGA 1100 A, Pomona, California) and recorded (HP-7758 A). Mass spectrometer and recorder were calibrated frequently during the course of the measurements using test gases (8% CO_2 in oxygen, 4% CO_2 in oxygen, 100% oxygen, 100% nitrogen, room air). Argon concentrations could not be measured with this type of mass spectrometer thus accounting for a maximum error of 1% in the measurement of summed gases. Gas was sampled (flow: 60 ml · min^{-1}) via a small soft catheter advanced into the tracheobronchial tree under sterile conditions through a gas tight side port of the y-piece. The position of the sampling catheter and the catheter used for O_2-insufflation was checked for on the routine chest X-rays. Gas was sampled for short time intervals only in order to prevent obstruction of the sampling catheter by tracheal secretions and to avoid changes in the gas composition by the sample flow itself.

Measurements were taken during successive days with changing either the F_IO_2 at the ventilator or the magnitude of the supplemental O_2-flow. Moreover a set of measurements was obtained at various points of the tracheobronchial tree by slowly advancing the sampling catheter down to the right lower lobe.

Blood gases and pH were measured with electrodes (AVL-gas check) at 37 °C, whereas Hb, O_2Hb, CoHb and MetHb were measured simultaneously with a Co-Oximeter (Mo. 282, Instrumentation Lab., Missouri).

Calculations

Dry gas concentrations (F_x) as measured with the mass spectrometer were converted to partial pressures according to:

$$P_x = F_x (P_B - 47).$$

For comparison with the measured end-tidal PO_2 a theoretical P_AO_2 was calculated from the simplified alveolar gas equation [13] according to:

$$P_AO_2 = F_IO_{2,\,\text{ventilator}} (P_B - 47) - \frac{P_aCO_2}{R}$$

arbitrarily assuming R = 0.8.

$\dot{Q}_{va}/\dot{Q}_t$ was calculated according to the Berggren formula corrected for measured CoHb and MetHb [1].

Results

A typical recording with gas sampling at the carina and a tidal volume of 400 ml is shown in Figure 1. Measured F_IO_2 in the trachea with the beginning of the inflation cycle was 0.45. However already during the inflation hold tracheal F_IO_2 is increased by the supplemental O_2-flow. Similarly expired gases were diluted by the O_2-flow. Immediately before the next inflation cycle tracheal O_2-concentration had risen to 0.9. Likewise real end-tidal F_ECO_2 was obscured by the supplemental flow. Thus, it was impossible to measure true inspired and expired gas concentrations during LFPPV-ECCO$_2$R due to the dilution of these gases by the endotracheal O_2-flow. Only if this O_2-flow was stopped at end-inspiration expired fractional gas composition and therefore alveolar gas composition could be measured (Figure 1).

It is apparent from Figure 1 however that measurement of alveolar gas concentrations was reliable for only one breath. As can be expected F_AO_2 decreases with each successive breath without the supplemental O_2-flow. Thus it was impossible to measure

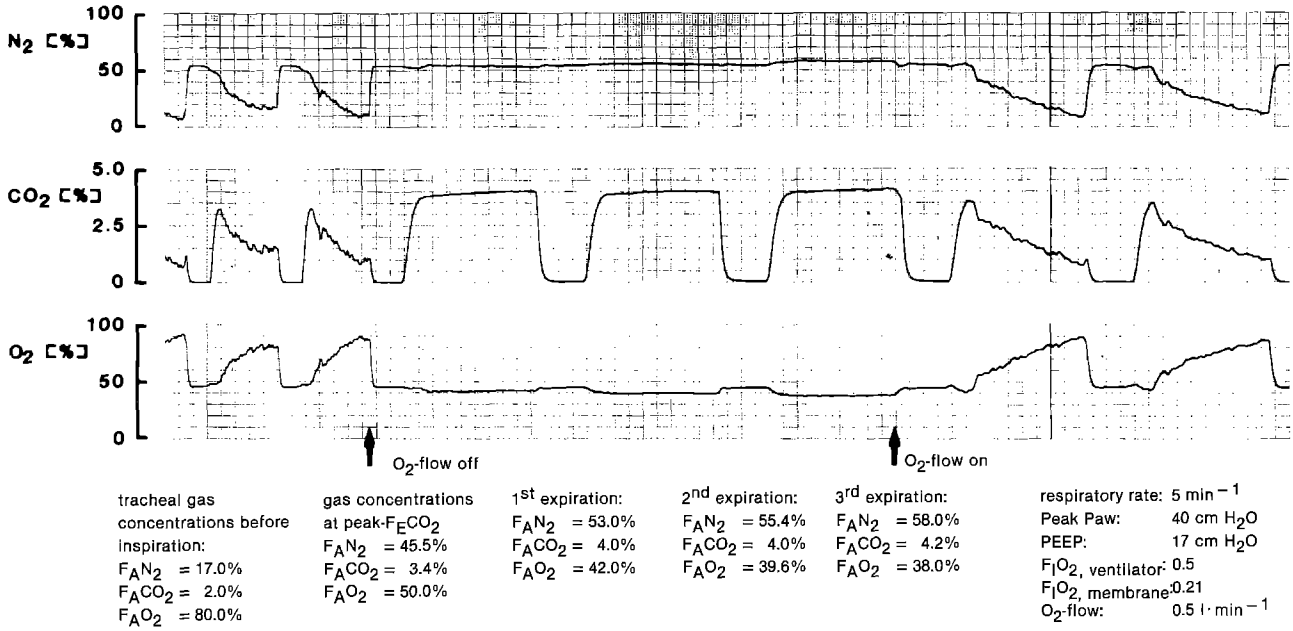

Fig. 1. Gas sampling from trachea at the carina. Note the progressive increase in tracheal oxygen concentration during apnea when endotracheal O_2-flow is on. When the supplement O_2-flow is stopped a plateau of alveolar gas concentrations becomes visible. With each successive breath end-tidal FO_2 drops until endotracheal O_2-insufflation is resumed

F_AO_2 and F_ACO_2 continuously without disturbing the steady-state. Gas concentrations measured under conditions with a supplemental O_2-flow on were not the same throughout the tracheobronchial tree and at the y-piece. Generally O_2-concentrations were higher when measured at the level of the carina than deeper in the bronchial tree. This certainly reflects the proximity of the gas sampling catheter to the orifice of the catheter used for O_2-insufflation but regional differences in lung gas exchange and establishment of tracheoalveolar gas concentration gradients during apnea periods may also have played a role. Repeatedly a $P_{(a-A)}CO_2$ of 19 to 26 mmHg was calculated indicating a large dead space ventilation. Figure 2 compares the "P_AO_2" calculated from the F_IO_2 set at the ventilator according to the alveolar gas equation (as defined in the methods section), the "P_AO_2" calculated from the expired FO_2 measured at peak F_ECO_2 with the O_2-flow on and the P_AO_2 measured at the alveolar plateau with the O_2-flow off.

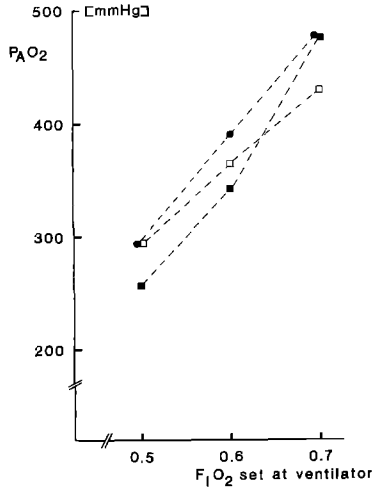

Fig. 2. Measured and calculated alveolar pO_2 during ECCO$_2$R-LFPPV. P_AO_2 is overestimated at low F_IO_2 and underestimated at higher F_IO_2 by calculation from the alveolar gas equation. □ calculated from F_IO_2 set a ventilator; ● measured under tracheal O_2-insufflation at peak F_ECO_2; ■ measured with tracheal O_2-insufflation stopped at end-inspiration (alveolar plateau). ECCO$_2$R-LFPPV (5 min^{-1}); O_2-insufflation: 0.5 l · min^{-1}; F_IO_2, membrane: 0.21

Under the conditions studied true alveolar P_AO_2 is overestimated at lower F_IO_2 and underestimated at higher F_IO_2 in comparison with values calculated from the alveolar gas equation as defined. This difference will markedly increase for any F_IO_2 when the magnitude of the supplemental O_2-flow increases (Figure 3).

Discussion

During controlled mechanical ventilation using respiratory rates of 8 to 16 min^{-1} and tidal volumes in the range of 12 – 15 ml · kg^{-1} convective gas transport into the lungs is sufficiently high to minimize great fluctuations in P_AO_2. Likewise during spontaneous breathing fluctuations of only 4 mmHg have been reported in the P_AO_2 of healthy adults [2].

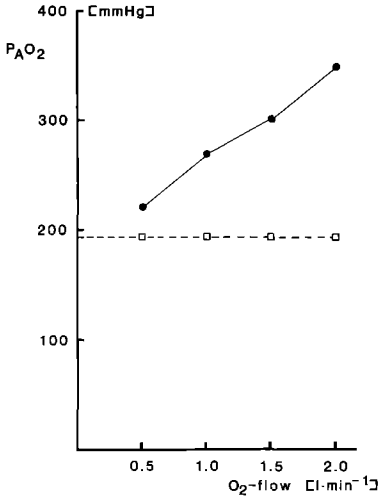

Fig. 3. "P_AO_2" calculated and measured with variable magnitudes of supplemental O_2-flow during ECCO$_2$R-LFPPV (see text). □ calculated from F_IO_2 set at ventilator (0.35); ● measured under tracheal O_2-insufflation at peak F_ECO_2

In contrast a supplemental O_2-flow is clearly required during LFPPV-ECCO$_2$R to prevent alveolar hypoxia especially if very low respiratory rates and oxygen concentrations set at the respirator are used. The need for a supplemental endotracheal O_2-flow however presents serious problems when P_AO_2, $P_{(A-a)}O_2$ or $\dot{Q}_{va}/\dot{Q}_t$ are to be calculated during LFPPV-ECCO$_2$R. Calculations based on the concept of the ideal alveolar gas equation using the F_IO_2 set at the ventilator and arbitrarily assuming a RQ of 0.8 are not correct as shown by the data presented. Calculation of P_AO_2 from the alveolar gas equation requires a steady respiratory state as a precondition. This however is not guaranteed during LFPPV-ECCO$_2$R using respiratory rates as low as 1.5 min^{-1} employed clinically. Theoretically P_AO_2 during LFPPV-ECCO$_2$R (without endotracheal O_2-flow) can be calculated according to the mass balance equation assuming constant $\dot{V}O_2$ and alveolar volume:

$$P_AO_{2,\,final} = P_AO_{2,\,initial} - \frac{\dot{V}O_2 \cdot (P_B - 47) \cdot \text{apnea time}}{\text{alveolar volume}} \quad (2)$$

where $P_AO_{2,\,final}$ and $P_AO_{2,\,initial}$ denote P_AO_2 after one breath and immediately before the next breath.

Accordingly, assuming an initial P_AO_2 of e.g. 200 mmHg, a $\dot{V}O_2$ of 400 ml · min^{-1}, P_B of 760 mmHg and an alveolar volume of 2000 ml, the P_AO_2 drops to 128.7 mmHg after 30 s of apnea. If alveolar volume is decreased to 1000 ml the other parameters being unchanged, the final P_AO_2 would drop to 57.4 mmHg.

Without a supplemental O_2-flow therefore the P_AO_2 during LFPPV-ECCO$_2$R would show a large amplitude and be very sensitive to changes in lung volume. Obviously P_AO_2 cannot be calculated on the basis of gas concentrations set at the ventilator. Moreover it has recently been demonstrated in healthy volunteers that the lung does not behave as a simple one-compartment model during breath-holding at low lung volumes [4]. Instead measured arterial oxygen saturation during a 30-s breath holding maneuver at low lung volumes decreases more than expected by calculations based on the mass balance equation given above. This is probably explained by closure of dependent lung units at low lung volumes [4]. A similar situation probably arises with the markedly reduced functional residual capacity in ARDS. Theoretically the drop in P_AO_2 during LFPPV can be prevented if the magnitude of the supplement O_2-flow equals $\dot{V}O_2$ across the lungs, provided complete gas mixing between alveolar gas and supplement O_2-flow occurs. It is doubtful however if this is possible even if one takes into account cardiogenic gas mixing [3], which may have an important role during LFPPV-ECCO$_2$R. Another important parameter that hampers calculation of P_AO_2 during LFPPV-ECCO$_2$R using standard equations is that the RQ across the patient's lungs during extracorporeal CO_2-elimination markedly deviates from normal RQ and depends on the magnitude of extracorporeal blood flow as well as on metabolic CO_2-production. Only for comparison with measured values we arbitrarily have chosen a RQ of 0.8 for calculation, a value commonly assumed when applying the simplified alveolar gas equation. We realise however that the RQ across the patient's lungs may be in the range of 0.3 during ECCO$_2$R-LFPPV. Moreover part of the patient's oxygen consumption is effected across the membrane lung and nitrogen fluxes between membrane lung and the patient's lungs must be taken into account when considering alveolar gas composition.

Finally, clinically used gas flowmeters rarely have a high accuracy in the range from 200 to 500 ml · min^{-1} and frequently there are small leaks in the patient-ventilator circuit which are not easily prevented.

All of these factors make it impossible in our opinion to reliably predict and calculate P_AO_2 during LFPPV-ECCO$_2$R. Most paramagnetic or polarographic oxygen analysers used in the clinical setting are not sufficiently fast to measure alveolar oxygen concentrations during LFPPV-ECCO$_2$R. Thus mass spectrometry seems the only method available to measure P_AO_2 during LFPPV-ECCO$_2$R. Even then however, P_AO_2 in perfused alveoli may be overestimated due to the large dead space in severe ARDS.

In conclusion P_AO_2, $P_{(A-a)}O_2$ and $\dot{Q}_{va}/\dot{Q}_t$ cannot be calculated during LFPPV-ECCO$_2$R with sufficient accuracy without the aid of a mass spectrometer. Calculations based on the F_IO_2 set at the ventilator can be grossly misleading and may cause errors in evaluation of patients during LFPPV-ECCO$_2$R. Therefore monitoring of alveolar gas composition during LFPPV-ECCO$_2$R by mass spectrometry is recommended.

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