Intensive Care Med (1984) 10:169–177

Intensive Care Medicine
© Springer-Verlag 1984

Review article

Variations of regional lung function in acute respiratory failure and during anaesthesia

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Accepted: 25 October 1983

Abstract. Acute respiratory failure and anaesthesia impede ventilation of dependent lung units and perfusion of non-dependent ones, creating considerable ventilation-perfusion ($\dot{V}/\dot{Q}$) mismatch. General PEEP can improve $\dot{V}/\dot{Q}$ but it cannot restore it to normal. To improve matching, ventilation must be distributed in proportion to regional blood flow. This can be accomplished by (1) placing the subject in the lateral position, (2) ventilating each lung in proportion to its blood flow (differential ventilation), and (3) applying PEEP solely to the dependent lung to ensure even distribution of inspired gas within that lung (selective PEEP). Differential ventilation with *equal* distribution of the tidal volume between the lungs and a selective PEEP of 10 cm H_2O to the dependent lung resulted in *equal* distribution of perfusion between the lungs in anaesthetized healthy subjects, suggesting "optimum" $\dot{V}/\dot{Q}$ matching. Using this ventilator setting as a rule of thumb in patients with acute, severe, *bilateral* lung disease, arterial oxygen tension was improved by an average of 45% compared with that during general PEEP, with no reduction in cardiac output. It is concluded that differential ventilation with selective PEEP can offer considerable improvement in gas exchange in acute, bilateral lung disease. However, long-term studies are required before a final evaluation can be made.

Key words: Acute respiratory failure – Anaesthesia – Differential ventilation – Perfusion – Airway closure – Selective PEEP

In adults anaesthesia is regularly accompanied by hypoxaemia unless supplementary oxygen is added to the inspired gas [32]. The hypoxaemia follows from impaired matching between ventilation and perfusion. Ventilation is preferentially distributed towards non-dependent lung units while perfusion is forced towards dependent ones. This "physiological" inequality of ventilation and perfusion may also occur in patients with acute, severe pulmonary insufficiency and add to that caused by their pulmonary disease, leading to severe hypoxaemia. In this paper, the mechanisms of "physiological" mismatching are analysed and possible measures to counteract them discussed.

Ventilation distribution

In the awake, healthy subject, ventilation increases down the lung from top to bottom. This is a consequence of the sigmoid shape of the pressure-volume curve of the lung and the vertical pleural pressure gradient [31] (Fig. 1). Dependent lung units are located on the lower, steeper part of the pressure-volume curve, and non-dependent units on the upper, flatter part. For a given increase in transpulmonary pressure, the lower lung regions expand more than the upper ones. However, in older subjects ventilation may be reduced in dependent lung regions, this being attributed to airway closure [31]. Closure of the airway may occur when the peribronchial pressure exceeds that in the airway. Since the intrathoracic, and thus peribronchial, pressure is higher in dependent than in non-dependent regions, airway closure begins at the bottom of the lung. Intrathoracic pressure increases during expiration, and this causes airway closure to spread up the lung during a sustained exhalation. In young subjects, airways close only during a deep expiration, below the functional residual capacity (FRC). Under such circumstances the ventilatory distribution will remain unaffected during normal

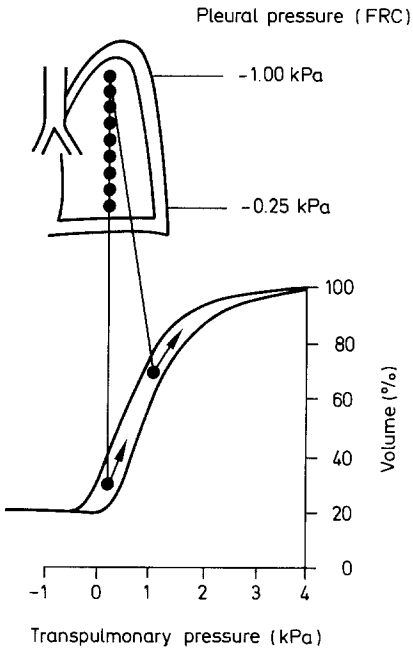

Fig. 1. A transpulmonary pressure-lung volume curve [after West JB (1970) Ventilation/blood flow and gas exchange. Blackwell, Oxford, p 31]. Note the sigmoid shape of the curve, causing a successively smaller volume increase when the pressure rises. The vertical pleural pressure gradient results in different locations of non-dependent and dependent lung regions on the pressure-volume curve. For a given increase in pressure, the volume increases more in dependent than non-dependent regions

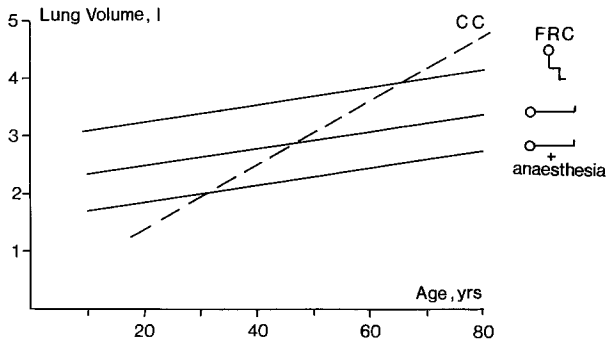

Fig. 2. Schematic drawing of the age dependence of resting lung volume (FRC) and closing capacity (CC), and of the influence of body posture and general anaesthesia on FRC

breathing. In older subjects, airway closure occurs at a higher lung volume, within or even above the tidal volume. The distribution of ventilation to dependent regions will thereby be affected, i.e. either reduced or abolished.

A change from the upright to the supine position reduces the resting lung volume, i.e. FRC, by 0.5 – 1 l [13], while the lung volume at which airways begin to close, the closing capacity, remains essentially unaltered [11, 28]. Airway closure within or above the tidal volume may thus occur more easily in the supine than in the upright position. Taking into consideration both age and body position, it can be predicted that in the upright position airways will close above FRC in subjects aged 65 years or older, whereas in the supine position such closure will occur above FRC in 45-year-old subjects [11, 28] (Fig. 2).

FRC is further reduced during general anaesthesia, by as much as 0.5 – 0.7 l. This has been demonstrated both in gas dilution tests [12, 27] and by body plethysmography [21, 44]. According to most reports, during general anaesthesia FRC is reduced, while the closing capacity remains unaltered [16, 17, 18] (different findings, of a decrease in closing capacity in parallel with the decrease in FRC, have been presented by Juno et al. [23]). Airways may thus close above FRC more easily during general anaesthesia than in the awake state. Assuming that the fall in FRC is similar at all ages, it may be anticipated that airway closure will occur above FRC in subjects as young as 30 years, and with an increasing distribution with increasing age (see Fig. 2).

The distribution of airway closure, as assessed by separate measurements in each lung, has been studied both in awake and in anaesthetized subjects [15, 20]. Neither FRC nor the closing capacity differed between the lungs in the supine subject, indicating an even distribution of these volumes in the horizontal plane. With the subject in the lateral posture, airway closure occurred earlier in the dependent than in the non-dependent lung (asynchronous onset of airway closure). In the anaesthetized subject in the lateral position it could also be shown that closing capacity did not differ from that in the supine position. However, FRC of the dependent lung was decreased and virtually coincided with the residual volume of this lung, while FRC of the non-dependent lung was increased. This resulted in airway closure well above FRC of the dependent lung but far below that of the non-dependent lung (Fig. 3) [20]. This must have resulted in a considerable hindrance to ventilation of the dependent lung. Similar vertical differences in ventilation occur when the subject is supine, although here they may be slightly less marked, as less pressure is exerted on dependent lung regions by mediastinal organs [34].

As well as being affected by the shape of the pressure-volume curve of the lung and the airway closure phenomenon, the intrapulmonary gas distribution will also depend upon the regional lung resistance. This is a function of regional volume and increases with decreasing lung volume [4, 31]. With the subject in the lateral position, the small volume of the dependent lung will thus increase the airway resistance,

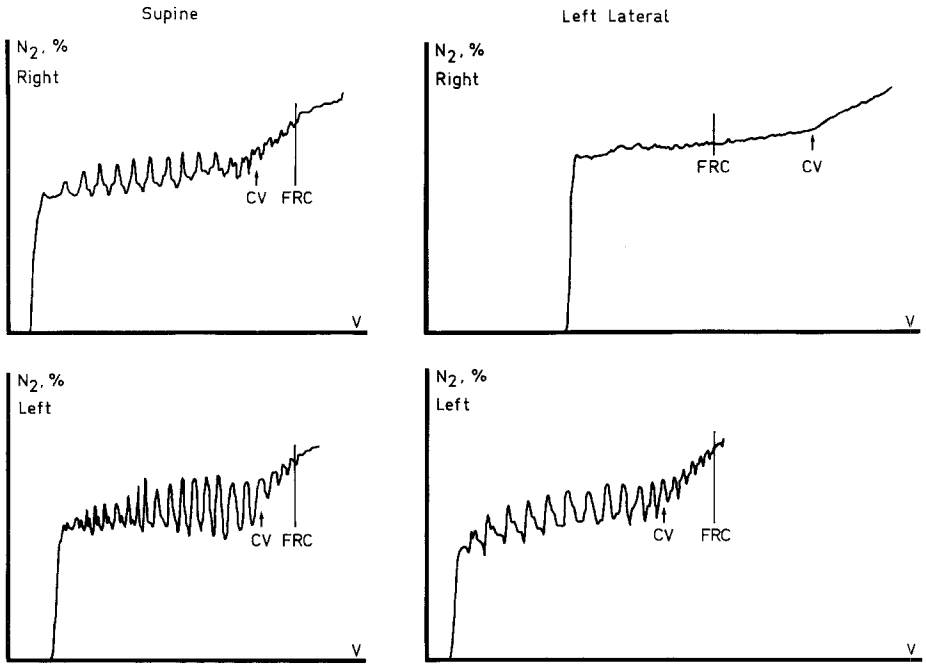

Fig. 3. Recording of airway closure by the single breath nitrogen washout test. Left panels show measurements with the subject supine, during halothane anaesthesia. Note that the closing capacity (CC) exceeds FRC in both lungs. The right panels show results in the same subject in the left lateral position. Airway closure occurs far below FRC in the non-dependent (right) lung but well above FRC of the dependent (left) lung. (From Bindslev et al. [7]. By courtesy of the editor of Acta Anaesth Scand)

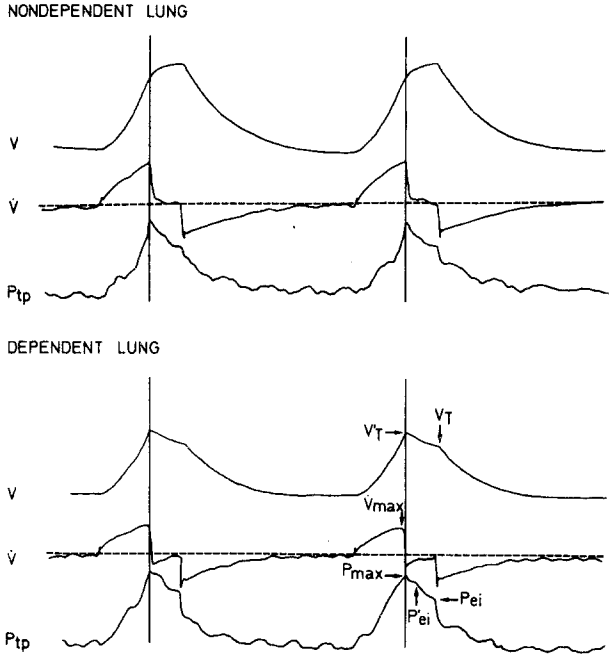

Fig. 4. Simultaneous recordings of inspired volume, gas flow and transpulmonary pressure in the left lateral position. (Upper panel shows recordings from the non-dependent and lower panel from the dependent lung.) During the end-inspiratory pause (EIP), the volume of the non-dependent lung is increased due to redistribution of gas from the dependent lung. (V: volume; $\dot{V}$: gas flow; P_{tP}: transpulmonary pressure; V_T': tidal volume before EIP; V_T: tidal volume after EIP; $\dot{V}_{max}$: peak gas flow; P_{max}: maximal inflation pressure (peak pressure); P'_{ei}: transpulmonary pressure at no flow before EIP; P_{ei}: transpulmonary pressure at no flow after EIP). (From Bindslev et al. [8]. By courtesy of the editor of Acta Anaesth Scand)

which will further impede the ventilation of this lung. However, in the anaesthetized healthy subjects the contribution of this factor to the ventilatory distribution appears to be small [8]. Finally, during the end-expiratory pause, if it occurs, redistribution of inspired gas from the more poorly ventilated dependent to the better ventilated non-dependent lung may take place (Fig. 4). This is because the time constant is shorter in the dependent lung [8]. In view of the higher resistance of this lung this may not be appreciated immediately. However, the difference in compliance between the lungs is even greater, with the lower compliance in the dependent lung. Since the time constant can be considered to equal compliance times resistance, it follows that it can be shorter in dependent lung units.

Regional perfusion

In a healthy subject, perfusion increases from apex to base in the upright posture and from ventral to dorsal regions in the supine posture, this being due to the increasing hydrostatic pressure down the lung [42]. A small reduction in regional perfusion may be seen in the lowermost region of the lung [39]. This minor reduction in dependent-lung perfusion may be due to compression of extra-alveolar vessels by interstitial oedema and, in case of airway closure, to hypoxic vasoconstriction – diverting the bloodflow to better ventilated regions [5, 43]. The uppermost region may be poorly or not at all perfused if the pulmonary arterial pressure is low.

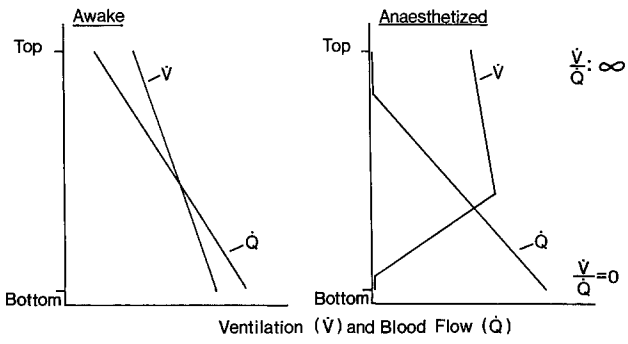

Fig. 5. Schematic drawing of the vertical distribution of ventilation and perfusion of the lung in the awake (left panel), and the anaesthetized (right panel) subject. For further explanation, see text

During general anaesthesia, the pulmonary arterial and systemic pressures may be reduced. The decrease in the former will impede perfusion of the uppermost, non-dependent lung regions. Institution of mechanical ventilation (IPPV) will increase alveolar pressure, which will further interfere with the perfusion of non-dependent regions [42]. The anaesthetic agent may reduce or abolish the hypoxic vasoconstrictor response which favours perfusion of poorly ventilated, mainly dependent lung regions [5, 38]. Thus, during anaesthesia these factors act in common to force perfusion down the lung, creating an increased preference for dependent perfusion, in opposition to the preference for non-dependent ventilation. The increased intrathoracic pressure may also impede venous return [41], and the anaesthetic may have a cardio-depressant action [30], both of which may reduce the cardiac output.

$\dot{V}/\dot{Q}$ matching

To create optimum gas exchange, there must be ideal "matching" between ventilation and perfusion. In the awake, upright subject, both ventilation and perfusion increase down the lung, the ventilation-perfusion ratio ($\dot{V}/\dot{Q}$) being not far from 1 at all vertical levels of the lung (Fig. 5). However, in the supine, anaesthetized subject there is no longer good matching between ventilation and perfusion. The impaired ventilation and, in percent of total perfusion, augmented blood flow in the dependent lung cause low $\dot{V}/\dot{Q}$ ratios and even true shunt. Upper lung units may be poorly or not at all perfused but still ventilated, creating a high $\dot{V}/\dot{Q}$ ratio and dead space. Thus, $\dot{V}/\dot{Q}$ may vary from zero to infinity (see Fig. 5). Moreover, using a multiple inert-gas elimination technique [40], an increased dispersion of $\dot{V}/\dot{Q}$ ratios has been demonstrated during anaesthesia and mechanical ventilation with the development of, or increase in true shunt ($\dot{V}/\dot{Q} = 0$) and regions with low $\dot{V}/\dot{Q}$ ($\dot{V}/\dot{Q} < 0.1$) as well as of regions with higher $\dot{V}/\dot{Q}$ ($V/Q > 10$). The impairment in $\dot{V}/\dot{Q}$ matching was more obvious in middle-aged and elderly patients [9, 14] than in subjects below 30 years of age [36]. This age dependence would fit with the idea that airway closure has a major impact on gas exchange, young subjects suffering no or little airway closure during anaesthesia, in contrast to those above 30 years of age (cf Fig. 2).

$\dot{V}/\dot{Q}$ in acute respiratory failure

So far the discussion has dealt with healthy subjects during anaesthesia. A fall in FRC, even more pronounced, is also seen in patients with acute respiratory failure. Katz et al. [24] reported a fall in FRC to 55% of the predicted value in the supine position, corresponding to a 1.3 l reduction, in 21 patients suffering from acute respiratory failure after severe trauma, major surgery, or metabolic or infectious disease. Pontoppidan and co-workers [33] proposed that such a fall in FRC promotes airway closure; to date, no studies on this subject have been reported. It should be noted that various conditions that reduce FRC may also reduce the closing capacity, e.g. chest strapping [37]. However, the latter reduction is less than that in FRC, and the promotion of airway closure within or even above the tidal breath is therefore most likely. This means that in acute respiratory failure the vertical distribution of ventilation may be similar to that seen in anaesthetized subjects, and possibly the decrease in ventilation of the dependent lung is even more marked. High airway and alveolar pressures are often required for adequate ventilation, which will force perfusion down the lung. $\dot{V}/\dot{Q}$ inequality similar to, or more advanced than that in the anaesthetized subject may thus ensue. This mismatching, which is "physiological" in the sense that it mimicks that seen in healthy, elderly, anaesthetized subjects, adds to the $\dot{V}/\dot{Q}$ inequality caused by the disease through other mechanisms, creating the well-known severe and sometimes life-threatening hypoxaemia.

To make the description more complete, it should be added that in general patients suffering from chronic obstructive lung disease present with an increased FRC. To what extent they have ventilatory impairment predominantly of dependent lung regions during anaesthesia and in intensive care remains to be demonstrated.

The prone position and general PEEP

How can the disturbances in $\dot{V}/\dot{Q}$ be restored to normal? Since the fall in FRC seems to be the crucial issue, measures to increase FRC might be beneficial. Rehder et al. [36] have tested the prone position with no support of the abdomen. By this means the diaphragm will not be forced into the thoracic cavity by the abdominal contents, and FRC should thus remain at a higher level than in the supine position. However, this position has limited use.

The reduction in FRC can also be counteracted by the application of positive end-expiratory pressure (PEEP), which may thus improve oxygenation of the blood [1, 25]. However valuable, PEEP has not become an indispensable method in intensive care and has shown no clearcut advantage in routine anaesthesia [22, 29]. There may be two explanations for the lack of benefit. Firstly, the increased intrathoracic pressure reduces cardiac output by impeding venous return [10], and a concomitant increase in alveolar pressure forces lung blood flow to dependent regions [42]. Secondly, the effect of PEEP on the regional lung volume is unfavourably distributed, dependent lung tissue being less expanded than non-dependent tissue. This has been demonstrated by constructing pressure-volume curves for each lung separately with the human subject in the lateral position [7]. Certain assumptions are necessary here, such as a linear pleural pressure gradient, but the value assigned to this will not interfere with the analysis [19]. The crucial reason for the unfavourable effect of PEEP is the sigmoid shape of the pressure-volume curve of the lung, in conjuction with the different locations of each lung on these curves – a consequence of the pleural pressure gradient (Fig. 6). The dependent lung is positioned on the lower, flatter part of its pressure-volume curve, while the non-dependent lung lies higher up on the steep portion of its pressure-volume curve. During inspiration or mechanical inflation, the airway and alveolar pressures increase equally in the two lungs. Application of a certain PEEP will then increase the dependent less than the non-dependent lung volume. Application of a PEEP of 10 – 12 cm H_2O should suffice to raise the dependent lung volume above its closing capacity in most subjects with normal lungs [7]. This amount of PEEP raises the dependent lung volume by the 0.2 – 0.3 l necessary to counteract airway closure. Simultaneously, however, the non-dependent lung volume increases by 0.8 – 1.0 l and approaches the maximum expansion. Not only does this entail unnecessary impedance to cardiac output, but it also increases the danger of barotrauma [26]. It may thus be concluded that PEEP does not provide the physiological basis for an optimum match between ventilation and perfusion, and that it may cause lung damage.

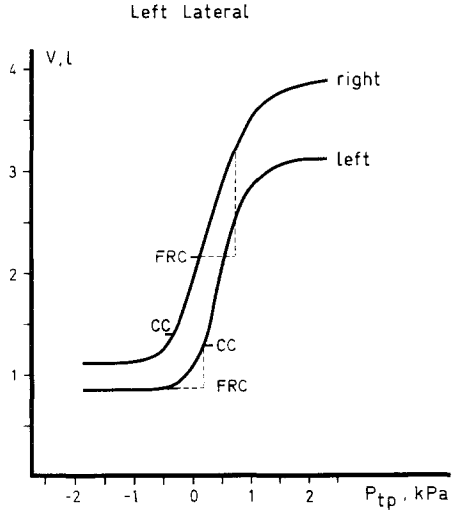

Fig. 6. Separate pressure-volume curves of the non-dependent (right) and dependent lung (left) in the lateral posture during anaesthesia. The resting lung level (FRC) and closing capacity (CC) of each lung are indicated, and also the effect of applying a positive end-expiratory pressure of approximately 1.3 kPa (13 cmH 0) (assuming equal compliances of the thoracic wall and the lungs and, thus, a transpulmonary pressure change (P_{tp}) of 0.65 kPa). Note the considerably larger increase in the non-dependent than in the dependent lung volume (From Bindslev et al. [7]. By courtesy of the editor of Acta Anaesth Scand)

Differential ventilation with selective PEEP

To create optimum "matching" between ventilation and perfusion, the ventilator should distribute the inspired gas in proportion to the regional perfusion, with the least possible interference with the total lung blood flow. This can be achieved, within certain limits, by ventilating anatomical lung units separately, delivering less gas to non-dependent and more to dependent units than would be the case if the tidal volume were distributed freely. The configuration of the human bronchial tree does not permit a perfusion-matched distribution of ventilation in the *supine* subject. However, this can be accomplished by (1) placing him in the *lateral* position, (2) ventilating each lung separately in proportion to its perfusion (differential ventilation), and (3) applying PEEP solely to the dependent lung to ensure the distribution of gas to lower regions within that lung (selective PEEP). By these means an improved ventilation-perfusion ratio within separately ventilated units can be achieved. Moreover, lower overall intrathoracic pressure and less lung expansion may be expected than with general PEEP. Interference with the total lung blood flow and the danger of barotrauma should thus be diminished.

Differential ventilation and selective PEEP are already being used during thoracic surgery in the treatment of unilateral lung disease. However, the reason for using them has not been the same as suggested here. During thoracic surgery, "one lung ventilation" facilitates intervention in the non-ventilated lung. In unilateral lung disease a stiff but well perfused lung may become unventilated unless differential ventilation is instituted. The point in the present analysis of lung function is that in the healthy lung, as well as in diffuse bilateral lung disease (which by far outnumbers unilateral lung disease), ventilation and perfusion are poorly matched by normal physiological mechanisms. This matching can be considerably improved by differential ventilation and selective PEEP, improving arterial oxygenation.

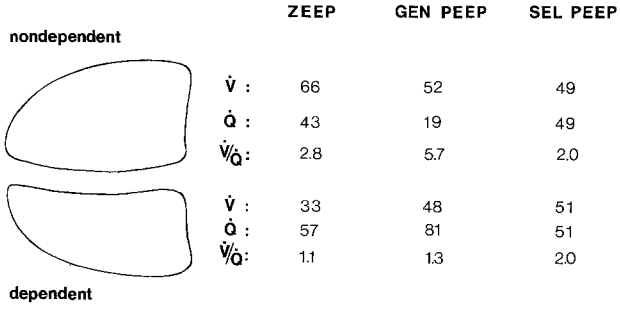

Fig. 7. A schematic drawing of the average distributions of ventilation ($\dot{V}$) and perfusion ($\dot{Q}$) between the lungs (in per cent of total ventilation and blood flow). The $\dot{V}/\dot{Q}$ ratios have been calculated from the absolute values of $\dot{V}$ and $\dot{Q}$. Note the predominance of dependent lung perfusion during zero end-expiratory pressure (ZEEP) and free distribution of ventilation (left column). With a general PEEP of 10 cmH_2O and free distribution of ventilation, perfusion was further squeezed down the lungs (middle column). When a selective PEEP of 10 cmH_2O was applied to the dependent lung only, and ventilation was distributed evenly between the lungs (differential ventilation), the distribution of perfusion also became even (right column)

Effects of differential ventilation on perfusion

In order to achieve optimum "matching" between ventilation and perfusion, the perfusion distribution between the lungs has to be known. Present knowledge on regional lung blood flow is based on measurements made without the use of PEEP or during general PEEP. Since differential ventilation and selective PEEP will affect the intrathoracic pressure and regional lung inflation, this ventilation concept will also have an impact on the distribution of perfusion. To investigate this question a study was made in subjects with healthy lungs during enflurane anaesthesia prior to elective surgery. Two synchronized ventilators (Servoventilator 900B, Siemens-Elema) were used, cardiac output determined by thermodilution and the perfusion distribution between the lungs was assessed by injecting a bolus of radioactive Xenon into a central vein and measuring the activity over each lung with a gamma camera [3]. The results showed that the perfusion distribution varied from 2/3 passing through the dependent lung with no PEEP and free distribution of ventilation, to 2/3 passing through the non-dependent lung with a selective PEEP of 16 cm H_2O and an equal distribution of ventilation between the lungs. In between these settings, a selective PEEP of 8 – 10 cm H_2O and an equal distribution of ventilation between the lungs caused a similarly even distribution of perfusion between the lungs, a condition that most probably is favourable for the pulmonary gas exchange (Fig. 7). With a *general* PEEP of approximately 10 cm H_2O on the other hand, perfusion was squeezed further down the lung, so that 80% passed through the dependent lung. Such a perfusion distribution cannot result in optimum $\dot{V}/\dot{Q}$ matching. It was also noted that cardiac output fell to a smaller degree with differential ventilation and selective PEEP than when general PEEP of the same magnitude was applied. Indeed, in patients with acute, severe bilateral lung disease necessitating ventilator treatment, cardiac output remained the same with selective PEEP as with no PEEP [2]. The maintenance or smaller reduction of the lung blood flow with selective as compared with general PEEP could be attributed to smaller increases in both intrathoracic pressure and pulmonary vascular resistance. It was thus concluded that differential ventilation with an *equal* distribution of ventilation between the lungs, and a selective PEEP of approximately 10 cm H_2O applied solely to the dependent lung, results in an *equal* distribution of perfusion between the lungs. This $\dot{V}/\dot{Q}$ matching most probably improves gas exchange.

Differential ventilation and selective PEEP in bilateral lung disease

Having established the ventilator setting creating "optimum matching" between ventilation and perfusion in lung-healthy subjects (equal distribution of ventilation between the lungs and selective PEEP of 10 cm H_2O), this setting was used as a "rule of thumb" in a pilot study on patients with severe acute respiratory failure due to diffuse and more or less uniform, bilateral lung disease, necessitating ventilator treatment. The material comprised five patients (1 woman, 4 men) with a mean age of 43 years (range 29 – 67). The diagnoses were bilateral bronchopneu-

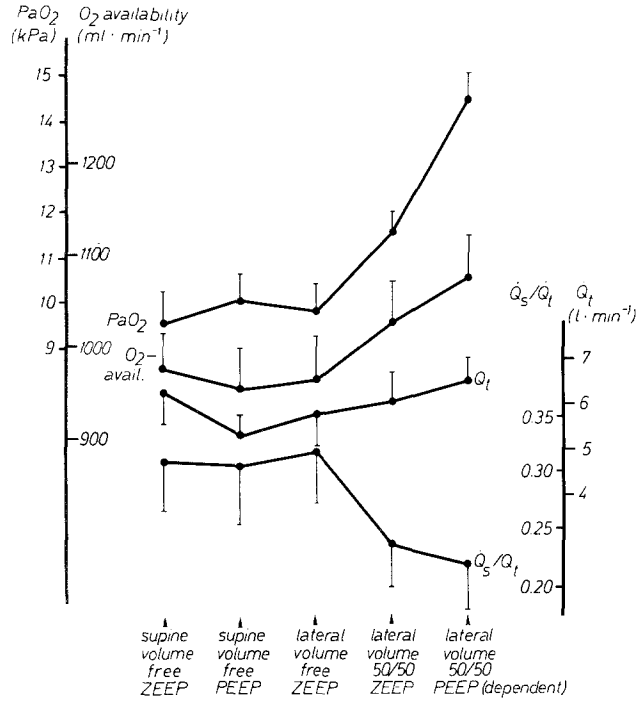

Fig. 8. Cardiac output ($\dot{Q}_T$), arterial oxygen tension (PaO_2), venous admixture ($\dot{Q}_S/\dot{Q}_T$) and oxygen availability (O_2 avail) in five patients with acute, severe, bilateral respiratory failure and the effect of differential ventilation with selective PEEP. ZEEP: zero end-expiratory pressure; PEEP: positive end-expiratory pressure; PEEP (dependent): PEEP to the dependent lung only; free: free distribution of the tidal volume between the lungs, assumed from other studies to be 2/3 to non-dependent and 1/3 to dependent lung; 50/50: 50% of tidal volume to each lung; Note the considerable increase in PaO_2 with "50:50" distribution (differential ventilation), and the further increase with the addition of selective PEEP to the dependent lung

monia, aspiration pneumonia and near-drowning. The inspiratory oxygen concentration was, on an average, 55%, the respiratory frequency 20 breaths/min, and the minute ventilation was adjusted so as to result in an arterial carbon dioxide tension of 4–5 kPa (30–37.5 mmHg). The order of the ventilator settings and body positions to be described was randomized. In the supine position with no end-expiratory pressure, the average PaO_2 was 9.5 kPa (71 mmHg) and cardiac output, as measured by thermodilution, was 6.1 l/min (Fig. 8). Venous admixture, calculated according to the standard shunt equation [6] was as high as 32%. The oxygen availability, i.e. the product of cardiac output and arterial oxygen content, was 970 ml/min (Fig. 8). The application of a general PEEP of 10.0 cm H_2O caused a moderate increase in PaO_2 to 10 kPa (75 mmHg) and a fall in cardiac output to 5.1 l/min. This caused a decrease in oxygen availability, despite a slight reduction in venous admixture to 30% of cardiac output. A change in body position to left lateral and discontinuation of PEEP restored cardiac output, venous admixture and arterial oxygenation to the same levels as in the supine position without PEEP. After exchanging the conventional single-lumen tracheal tube for a double-lumen orotracheal tube (left main bronchus intubation), the tidal volume was deliberately distributed so that 50% went to each lung (differential ventilation). With no PEEP, a claear increase in PaO_2 to 11.4 kPa (86 mmHg) occurred. Cardiac output was much the same as when the tidal volume had dispersed freely without the use of PEEP. Venous admixture was reduced to 24%. The oxygen availability was increased. With an even distribution of the tidal volume between the lungs and the application of a PEEP of 10 cm H_2O solely to the dependent lung (differential ventilation with selective PEEP), arterial oxygenation was further improved, with a PaO_2 averaging 14.5 kPa (109 mmHg). This meant an average increase of 45% compared with general PEEP, supine. Cardiac output did not decrease on application of selective PEEP, in fact a small mean increase was noted. Venous admixture was further reduced to 22% and the oxygen availability was increased to 1080 ml/min (see Fig. 8). All patients improved most with differential ventilation and selective PEEP, but there was some variation in the degree of response. Although the material was small, the impression was obtained that those who had the lowest PaO_2 during conventional ventilation with a single lumen tube improved most with differential ventilation and selective PEEP.

These findings were made during rather short studies in a limited number of patients, but they suggest that a considerable improvement in gas exchange can be achieved by differential ventilation with selective PEEP in patients with acute, severe bilateral respiratory failure, in comparison with conventional general PEEP. However, it is obvious that the ventilation technique also has some drawbacks. More equipment (two ventilators or a special "flow-dividing" unit) and greater supervision are required. The proper positioning of the double-lumen tube must be checked intermittently. Clearing of the airways from secretions may be more difficult, since thinner catheters must be used. On the other hand, access to both main bronchi is granted.

Differential ventilation and selective PEEP during general anaesthesia

In the operating theatre the lateral position has limited use. However, it must be kept in mind that this position may cause airway closure to be predominant in the dependent lung and it is even possible that

the entire lung may be closed off if the abdominal pressure is raised and/or if the diaphragm is pushed cranially during the operation. Differential ventilation and selective PEEP may thus also constitute a valuable tool during certain surgical procedures. In current studies on healthy subjects during inhalation anaesthesia prior to scheduled elective surgery, the effect on gas exchange has varied from minor improvement in younger subjects in whom airway closure has been assumed to be of a small extent, to considerable improvement in middle-aged subjects. It is conceivable that the benefits are greater during the surgical procedure if this causes the diaphragm to be pushed cranially, reducing FRC. Thus, differential ventilation with selective PEEP may be beneficial to the gas exchange in patients in whom the movement of the diaphragm is severely interfered with, by the surgery itself or because of their body constitution (e.g. obesity). However, further studies are required before any conclusions can be made.

The mismatching of ventilation and perfusion observed in acute respiratory failure and during anaesthesia impedes gas exchange and may lead to severe hypoxemia. General PEEP can improve matching of ventilation and perfusion but cannot restore it to normal. This can, however, be achieved by placing the subject in the lateral position, ventilating each lung in proportion to its blood flow (differential ventilation) and applying PEEP solely to the dependent lung to ensure an even distribution of inspired gas within that lung (selective PEEP). This technique offers to improve gas exchange in acute bilateral lung disease. Long-term studies are required before a definitive conclusion can be made.

Acknowlegdements. This study was supported by grants from the Swedish Medical Research Council, (No. 4X-5315), the Laerdal Foundation and the Karolinska Institute.

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