GAS EXCHANGE AND HAEMODYNAMICS DURING THORACOTOMY

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SUMMARY
Cardiac index, systemic and pulmonary arterial pressures, carbon dioxide elimination and ventilation of each lung were studied during thoracotomy. Seventeen patients, placed in the full lateral position, were ventilated mechanically through a Carlens' tube to moderate hypocapnia. Mean cardiac index increased by 12% as the pleura was opened ($P<0.05$), with no further change during surgery on the still ventilated upper lung. Mean arterial pressure was unchanged after opening the pleura, but decreased from $114 \pm 15$ mm Hg (mean $\pm$ 1 SD) to $104 \pm 18$ mm Hg during surgery on the lung ($P<0.01$). Mean pulmonary artery pressure was unchanged. There was a significant ($P<0.01$) increase in carbon dioxide elimination from the upper lung when the pleura was opened. In addition, the ventilation of this lung increased significantly ($P<0.05$). Mean end-tidal PCO$_2$ of the lower lung increased from 4.1 to 4.2 kPa after opening the pleura, while that of the upper lung increased from 3.0 to 3.6 kPa ($P<0.01$). $V_d/V_T$ decreased from 43 to 38% as the pleura was opened ($P<0.01$). During surgical handling of the lung, marked decreases in ventilation, compliance, carbon dioxide elimination and end-tidal PCO$_2$ were observed in the upper lung. We conclude that ventilation-perfusion mismatch decreased on opening the pleura, and that neither opening the pleura nor the subsequent lung surgery (both lungs being ventilated) caused any clinically important derangements in haemodynamics or oxygenation.

Studies in man of the circulatory effects of opening the pleura have yielded conflicting results. Thus, Li, Rheinlander and Etsten (1960) found a decrease in cardiac index (with the exposed lung untouched) while Nealon and colleagues (1959) observed an increase in the majority of the patients studied.

Before thoracotomy, the patient is usually placed in the full lateral position, and ventilation is controlled. This results in the preferential distribution of inspired gas volume to the upper lung (Rehder et al., 1972), the effect being enhanced as the pleura is opened (Doerfel, 1959; Nunn, 1961). Nunn's findings, like those of Virtue and co-workers (1966), suggested that the opening of the pleura caused an increase in ventilation-perfusion mismatch additional to that created by turning the patient on his side. However, Kerr and colleagues (1973) were unable to demonstrate any significant change in the physiological deadspace to tidal volume ratio ($V_d/V_T$).

The retraction and compression of the exposed, still ventilated lung, may have more important consequences for gas exchange and circulation than thoracotomy per se. Few authors have studied this aspect systematically. The present study was designed to gather more information on the circulatory and ventilatory consequences of thoracotomy, and the surgical handling of the lung.

PATIENTS AND METHODS
Following approval by the local Human Investigations Committee, 17 patients were studied after giving informed consent. Patients were not included in the study if they had marked unilateral or bilateral impairment of lung function. A previous paper (Werner et al., 1984) has described the haemodynamics and gas exchange in these patients in the supine and lateral positions before the opening of the pleura, and detailed the patient population and the methods. Thus, only a brief account is given here.

Anaesthesia was induced with thiopentone and maintained with intermittent doses of pethidine i.v., and 50% nitrous oxide in oxygen. Pancuronium 6–8 mg was given and intermittent positive pressure ventilation commenced (Servo Ventilator 900B (Siemens–Elema, Stockholm)) via a Carlens' double-lumen tube at a respiratory rate of 10 b.p.m. The insufflation time and the post-inspiratory pause time were each 20% of the full respiratory cycle. Inspired volume was constant during the study. The tubing was arranged so that inspired gas was distributed freely between the lungs, according to the mechanical properties of each side. However, a valve system ensured that the expired gas from one of the lungs was directed back through the expirat-
ory flow meter and expiratory valve of the ventilator which performed the insufflation. The expired gas from the other lung was directed through the expiratory port of a second Servo Ventilator, which was synchronized electrically with that of the first ventilator. Each ventilator was equipped with a Siemens-Elema CO$_2$ analyzer 930 (Olsson et al., 1980). Thus, the ventilation and carbon dioxide elimination of each lung could be measured separately. Airway pressures from both lungs were measured using the standard manometers on the ventilators.

The techniques for measuring cardiac output (15 patients), blood-gas tensions, $V_D/V_T$ and systemic and pulmonary arterial pressures have been described previously. A satisfactory pulmonary arterial occlusion pressure was always obtained during insertion of the catheter, although not always in the lateral position. Ventilation, carbon dioxide elimination and airway pressures were measured simultaneously with the blood sampling.

On each side, compliance of the respiratory system ($C_n$) was calculated as: $C_n = V_T/P_{pulm}$; where $P_{pulm}$ equals pressure at the end of the end-inspiratory pause and $V_T$ is the expired tidal volume. This formula is strictly valid only if end-expiratory lung pressure and, hence, end-expiratory flow is zero. Expiration lasted 3.6 s, and before opening the pleura this was long enough for the expiratory flow to cease in most patients. However, $C_n$ was not calculated in two patients, because there was obvious end-expiratory flow from the upper lung, even at this stage. After thoracotomy an appreciable end-expiratory flow from the upper lung was present in most patients (mean value = 13% of early expiratory flow). This means that the above formula probably underestimated to some extent the true compliance following opening the pleura.

Measurements obtained on three occasions are described. Stage A: patient in the lateral position. The outer layers of the chest wall had been divided and the pleura was about to be opened. Surgery was discontinued for 1–2 min during measurements. Stage B: 5–10 min after pleural opening. A rib retractor had been inserted. Pleural adhesions, if present, had been divided so that the exposed lung could move freely. Otherwise, the surgeon avoided touching the lung, and surgery was stopped completely 1–2 min before measurements were obtained, to allow conditions to stabilize. Stage C: After 10–15 min of dissection on the exposed but still ventilated upper lung. The surgeon or an assis-
tant held parts of the lung aside with the hands, or with a retractor, to expose properly the surgical field. Major vessels or bronchi had not been divided.

Measurements during stage C were made in only 12 patients. In the remaining five the surgeon requested one lung ventilation on opening the pleura. As these five patients did not differ markedly from the others, in respect of measurements during stages A and B, values for all 17 patients (first two stages) and for the remaining 12 (stage C) are presented together in the text and illustrations.

Statistics

The (two-sided) rank sum test for paired data (Wilcoxon test) was used. $P$ values less than 0.05 were considered to indicate significance.

RESULTS

Haemodynamics

Cardiac index ($CI$) increased from $2.3 \pm 0.6$ litre min$^{-1}$ m$^{-2}$ (mean $\pm 1$ SD) before the pleura was opened to $2.6 \pm 0.8$ litre min$^{-1}$ m$^{-2}$ after opening ($P<0.05$). A decrease was observed in only two patients (by 0.4 and 0.6 litre min$^{-1}$ m$^{-2}$, respectively). There was no further significant change during retraction of the exposed lung (fig. 1).

![FIG. 1. Mean (± 1SD) arterial pressure (MAP), heart rate (HR) and cardiac index (CI). Significance of changes between successive stages: *$P<0.05$; **$P<0.01$.](https://academic.oup.com/bja/article-abstract/56/12/1343/241908)
Mean arterial pressure (MAP) was 114±14 mm Hg before the pleura was opened and there was no significant change on opening the pleura. MAP decreased to 104±18 mm Hg during surgery on the lung (P<0.01). The lowest value obtained in any patient was 80 mm Hg (fig. 1).

Heart rate increased significantly (P<0.01) as the pleura was opened with a further increase (P<0.01) during surgery on the lung (fig. 1).

Pulmonary arterial pressure. Mean pressures were 17±5, 17±6 and 18±5 mm Hg, respectively, dur-

![Graph of ventilation, carbon dioxide elimination and lung mechanics](https://academic.oup.com/bja/article-abstract/56/12/1343/241908)

**Fig. 2.** Changes in ventilation, carbon dioxide elimination and lung mechanics (mean ± 1 SD). VE = expired volume; VCO2 = expired carbon dioxide volume; Vd/VT = physiological deadspace fraction of tidal volume; compliance = compliance of respiratory system; Ppeak = peak pressure; Ppause = pressure at the end of end-inspiratory pause. *P<0.05; **P<0.01.
ing the three stages of the study (no significant change). The corresponding values for pulmonary artery occlusion pressure were 13±6, 9±6 and 11±6 mm Hg (9, 11 and 7 observations, respectively). These changes were not significant.

Central venous pressure (CVP) was 5±5 mm Hg shortly before opening the pleura. Subsequently, the mean value changed by less than 0.3 mm Hg (n.s.).

Ventilation
Total expired minute volumes (BTPS) were 8.4±1.0, 8.4±1.0 and 8.5±1.1 litre min⁻¹ during the three stages. Carbon dioxide elimination (V̇CO₂) (BTPS) was 193 ± 27 ml min⁻¹ before the pleura was opened, increased to 211 ± 23 ml min⁻¹ shortly after opening the pleura (P<0.01) and was 209 ± 30 ml min⁻¹ during surgery on the lung.

Distribution of ventilation between the lungs. Shortly before the pleura was opened, 55±5% of the expired volume came from the upper lung. This was a significant (P<0.01) increase in comparison with conditions in the supine position (not shown in the figure), when the corresponding fraction was 48±4%. There was a further increase to 57±4% after opening the pleura, but a marked decrease (P<0.01) during surgical handling of the exposed lung (fig. 2A).

Distribution of carbon dioxide elimination between the lungs. Shortly before the pleura was opened, 49±7% of total carbon dioxide elimination came from the upper lung. After opening the pleura, the fraction increased to 54±6% (P<0.01). Surgery on the lung decreased the fraction to 38±10% (P<0.01) (fig. 2A). VD/VT decreased from 43±3% to 38±4% on opening the pleura (P<0.01). There was no further significant change during surgery (fig. 2A).

Arterial P₅0₂ was 4.1±0.3 kPa before the pleura was opened and did not change subsequently (fig. 2B).

End-tidal P₅0₂. During all three stages of the study, the end-tidal P₅0₂ of the upper lung was less than that of the lower lung in each patient. The difference in end-tidal P₅0₂ between the lower and the upper lung was 1.2±0.5 kPa before opening the pleura, decreased to 0.7±0.4 kPa once the pleura had been opened (P<0.01) and then increased during surgery on the lung (1.2±0.6 kPa)—that is, the difference between the lungs increased again (P<0.01) (fig. 2B).

Airway pressure. Since both lungs were insufflated via a common inspiratory limb, peak airway pressures during insufflation were the same on both sides. The mean peak pressure was 2.5 kPa before the pleura was opened, and decreased (P<0.01) to 20 kPa following opening of the pleura. The peak pressure increased again during the surgical handling of the lung (fig. 2D).

The pressure at the end of the inspiratory pause (Pₜₚₑ) was slightly less in the upper lung than in the lower lung before thoracotomy. Pₜₚₑ decreased in both lungs once the pleura had been opened (P<0.01), with the most marked change being evident in the upper lung. There were considerable increases in Pₜₚₑ during surgical handling of the exposed lung.

Compliance of the respiratory system (C₉⁰) (fig. 2C) was greater (P<0.01) on the upper side than on the lower side before the pleura had been opened. C₉⁰ increased greatly on the upper side once the pleura had been opened. C₉⁰ of the lower side increased also (P<0.01), but the change was not as pronounced. Surgical handling of the exposed lung caused a marked decrease in compliance of the upper lung.

P₅0₂, pH and base excess. Arterial P₅0₂ (Pₐ₀₂) was 28±4 kPa before and 25±6 kPa shortly after opening the pleura (n.s.). Pₐ₀₂ decreased significantly (P<0.01) to 22±9 kPa during surgery/retraction of the upper lung. The minimum Pₐ₀₂ observed in any of the patients was 9.8 kPa. Mean mixed venous P₅0₂ obtained before opening the pleura was 4.8 kPa. The values obtained once the pleura had been opened, and during lung surgery were 5.1 and 5.0 kPa, respectively. Minimum values noted in individual patients during the three stages of the study were 4.0, 4.0 and 4.1 kPa.

Arterial pH was 7.50±0.03 before, 7.50±0.04 just after opening the pleura, and 7.49±0.05 during surgery on the lung. The corresponding figures for base excess were +1.5±1.5, +1.3±1.5 and +0.75±1.8 mmol litre⁻¹, respectively.

DISCUSSION
Previously, it was thought that thoractomy induced major changes in the circulation. Thus, Rushmer
Closely the conditions during routine procedures.

The decrease in mean arterial pressure at this point appeared to be clinically unimportant. The unchanged mixed venous PO2 testified to the adequacy of the circulation. There is a large turnover of catecholamines in the lungs (Fishman and Pietra, 1974), and we wonder whether the marked increase in heart rate during surgical handling of the lung may have been caused by catecholamine release.

The formula used for compliance \(C_L\) required that end-expiratory lung pressure was zero. This was not strictly true in all patients, since an appreciable end-expiratory flow was often present after the pleura had been opened. Consequently, the measured increase of \(C_L\) on the upper side, on opening the pleura, underestimated the true change somewhat. Ventilation on the upper side increased after opening the pleura, but not in proportion to compliance. This is explained by the fact that the distribution of inspired gas, between the lungs, was influenced not only by compliance, but also by resistance to flow in the channels of the Carlens’ tube.

The upper lung eliminated more carbon dioxide shortly after the pleura had been opened, than before. In fact, the increase in the elimination of carbon dioxide from the upper lung was proportionally greater than the increase in ventilation (fig. 2A). Furthermore, the end-tidal \(PCO_2\) of the upper lung increased more than that of the lower lung (fig. 2B). This must reflect a marked increase in blood flow through the upper lung on opening the pleura, for which there are two possible explanations. First, there was a decrease in airway pressure on opening the pleura which was particularly pronounced in the upper lung, while mean pulmonary arterial pressure was unchanged. Second, an exposed lung with normal elasticity does not fill the pleural cavity during most of the respiratory cycle. Thus, the vertical distance between the heart and the uppermost parts of the lung will be decreased and the effect of hydrostatic pressure, on blood flow through the upper lung, may be of less importance with the pleura open.

The present finding of a decrease in \(VD/VT\) on opening the pleura agrees with the finding of a closer correlation between the elimination of carbon dioxide and the ventilation of the two lungs after, than before pleural opening (fig. 2A). In contrast with our findings, Virtue and colleagues (1966) found that the fraction of the lung which was ventilated but not perfused, increased on opening the pleura, although Kerr and co-workers (1973) found \(VD/VT\) to be unchanged. Thus, it would appear that there is substantial disagreement as to what actually happens to ventilation-perfusion relations when the
pleura is opened. Moreover, comparison between the different studies is made difficult by the fact that different principles of ventilation and different endotracheal/endobronchial tubes were used. Thus, Kerr and associates (1973) used pressure-controlled ventilation, while Virtue and colleagues (1966) used a single-lumen tube with which distribution of ventilation would be different from that of a double-lumen tube. In addition, the latter workers did not state whether the upper lung had been exposed to surgery, before the measurements which were obtained.

Naturally, those measurements which were obtained during surgery on the exposed lung represented only a single sample of a rapidly varying process. Nevertheless, several changes relative to the previous stage were obvious. Thus, the compliance and ventilation of the upper lung decreased. End-tidal \( \text{PCO}_2 \) and carbon dioxide elimination of the upper lung decreased markedly, which suggests that blood flow through the lung was affected considerably by surgical manipulation. In spite of the marked changes in compliance, ventilation and carbon dioxide elimination of both lungs during the three stages of measurement, changes in arterial \( \text{PCO}_2 \) were small. The maximum variation in individual patients was less than 1 kPa. In addition, arterial oxygenation was satisfactory and the circulation was well maintained. Unlike Virtue and colleagues (1966), we found no tendency towards metabolic acidosis during thoracotomy.

The present findings support our clinical experience. Major problems in anaesthetic management during thoracotomy and surgery on the lung are most often the result of easily identifiable causes such as hypoxaemia during one lung ventilation, haemorrhage, obstruction of the airway or surgical interference with the airway. Thoracotomy \textit{per se} does not appear to cause major difficulties.

**REFERENCES**


**ECHANGE DE GAZ ET HEMODYNAMIQUE EN COURS DE THORACOTOMIE**

**RESUME**

En cours de thoracotomie, on a procédé à l'étude de l'indice cardiaque, des pressions artérielles pulmonaire et systémique, de l'élimination de l'anhydride carbonique et de la ventilation de chacun des poumons. Dix-sept patients placés en position latérale totale ont été ventilés mécaniquement au moyen d'un tube de Carlens afin de modérer l'hipocapnie. L'indice cardiaque moyen a augmenté de 12% lorsque la plevre a été ouverte (\( P < 0,05 \)), sans qu'aucun changement ultérieur ne se produise en cours d'opération dans le poumon supérieur encore sous ventilation. La pression artérielle moyenne est demeurée inchangée après l'ouverture de la plevre, mais elle s'est abaissée de 114±15mm Hg (moyenne±1 SD) à 104±18mm Hg au cours de l'opération du poumon (\( P < 0,01 \)). La pression de l'artère pulmonaire est restée inchangée. Il y a eu une augmentation significative (\( P < 0,01 \)) de l'élimination de l'anhydride carbonique du poumon supérieur une fois que la plevre était ouverte. En outre, la ventilation de ce poumon a augmenté de manière significative (\( P < 0,05 \)). L'\( \text{PCO}_2 \) courant-terminal moyen du poumon inférieur a augmenté de 4,1 à 4,2 Kpa après ouverture de la plevre, tandis que celui du poumon supérieur augmentait de 3,0 à 3,6 Kpa (\( P < 0,01 \)).
Vd/Vt est descendu de 43 à 38% lorsque la plèvre a été ouverte (P < 0,01). Au cours de la manipulation chirurgicale du poumon, des baisses marquées de la ventilation, de l’élasticité pulmonaire, de l’élimination de l’anhydride carbonique et du Pco2 courant-terminal ont été observées dans le poumon supérieur. Nous en concluons que l’incompatibilité ventilation/perfusion a diminué au moment de l’ouverture de la plèvre et que ni l’ouverture de la plèvre ni l’opération ultérieure du poumon (les deux poumons se trouvant sous ventilation) n’ont occasionné de dérangements importants dans l’hémodynamique ou l’oxygénation.

**GASAUSTAUSCH UND HÄMODYNAKIK WÄHREND THORAKOTOMIE**

Während Thorakotomie wurden Herzindex, systemischer und pulmonalarterieller Druck, CO2-Elimination und Ventilation beider Lungen untersucht. Siebzehn Patienten wurden in Seitenlagerung mechanisch mit gemäßigtter Hypokapnie über einen Carlens-Tubus beatmet. Bei Öffnung der Pleura stieg der mittlere Herzindex um 12% (P < 0,05) und änderte sich während der Operation an der weiter beatmeten oben liegenden Lunge nicht mehr. Der mittlere arterielle Druck blieb nach Pleuraoöffnung unverändert, fiel jedoch während der Operation an der Lunge von 114 ± 15 mm Hg (Mittelwert ± Standardabweichung) auf 104 ± 18 mm Hg. Der mittlere Druck in den Pulmonalarterien blieb unverändert. Nach Pleuraoöffnung kam es in der oben liegenden Lunge zu einem signifikanten (P < 0,01) Anstieg der CO2-Elimination und der Ventilation (P < 0,05). Zum selben Zeitpunkt stieg der mittlere endexspiratorische Pco2 der unteren Lunge von 4,1 auf 4,2 kPa, während er in der oberen Lunge von 3,0 auf 3,6 kPa anstieg (P < 0,01). Vd/Vt fiel nach Pleuraoöffnung von 43 auf 38% (P < 0,01). Während der Operation an der Lunge wurde in der oberen Lunge ein deutlicher Abfall von Ventilation, Compliance, CO2-Elimination und endexspiratorischem Pco2 beobachtet. Das Mißverhältnis Ventilation:Perfusion verringerte sich also bei Öffnung der Pleura, und weder Pleuraoöffnung noch die nachfolgende Lungenoperation (mit Beatmung beider Lungen) führten zu einer klinisch bedeutsamen Störung von Hämodynamik oder Oxygenierung.

**INTERCAMBIO DE GAS Y HEMODINAMICA DURANTE LA TORACOTOMIA**

**SUMARIO**

Durante una toracotomia, se estudiaron el índice cardiaco, las presiones arteriales pulmonar y sistémica, así como la eliminación del anhidrido carbónico y la ventilación de cada pulmón. Con el objeto de moderar la hipocapnia, se ventilaron a diez y siete pacientes colocados en postura lateral total y mecánicamente ventilados mediante un tubo de Carlen. El índice cardiaco promedio aumentó en un 12% cuando se abrió la pleura (P < 0,05) sin ningún otro cambio durante la operación en el pulmón superior todavía bajo ventilación. La presión arterial promedio no cambió después de la abertura de la pleura, pero disminuyó de 114 ± 15 mm Hg (Promedio ± 1 SD) hasta 104 ± 18 mm Hg durante la operación del pulmón (P < 0,01). La presión promedia de la arteria pulmonar no sufrió cambio alguno. Se produjo un aumento significante (P < 0,01) de la eliminación del anhidrido carbónico del pulmón superior, una vez que la pleura se hallaba abierta. Además, la ventilación de dicho pulmón aumentó de manera significante (P < 0,05). El Pco2 terminal del pulmón inferior aumentó de 4,1 a 4,2 kPa después de la abertura de la pleura, mientras que el del pulmón superior aumentaba de 3,0 a 3,6 kPa (P < 0,01). El Vb/Vt disminuyó de 43 a 38% cuando se abrió la pleura (P < 0,01). Durante el manejo quirúrgico del pulmón, se observaron en el pulmón superior descensos significativos de la ventilación, de la elasticidad pulmonar, de la eliminación del anhidrido carbónico y del Pco2 terminal. Concluimos que la discrepancia ventilación-perfusión disminuyó al abrir la pleura y que la abertura de la pleura ni tampoco la cirugía subsecuente del pulmón (ambos pulmones encontrándose bajo ventilación) no causaron disturbios clínicamente importantes en la hemodinámica o la oxigenación.