CHANGES IN FUNCTIONAL RESIDUAL CAPACITY DURING CARDIAC SURGERY

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The induction of general anaesthesia causes a decrease in functional residual capacity (FRC) of around 20% (Rehder, Sessler and Marsh, 1975; Nunn, 1977). However, what happens to FRC during the subsequent operative procedure is less clear. During cardiac surgery, several factors such as sternotomy and cardiopulmonary bypass (CPB) might affect FRC. We studied this problem in adults undergoing aortic valve replacement or coronary artery bypass grafting (CABG). In view of the marked changes in lung volume that were observed, arterial oxygenation was assessed in another group of patients.

PATIENTS AND METHODS

Eight men were studied. Informed consent was obtained from all patients and the study was approved by the local Human Investigations Committee. None of the patients had any history of pulmonary disease. Five patients were ex-smokers and three were non-smokers. Recent measurements of vital capacity and forced expired volume over 1 s were available for all patients (table I). Anaesthesia and surgery followed the routine practice of our hospital: anaesthesia was induced with droperidol 0.12–0.21 mg kg\(^{-1}\) and fentanyl 0.008–0.018 mg kg\(^{-1}\) and maintained with droperidol, fentanyl and 50–65% nitrous oxide in oxygen. Alcuronium 0.12–0.21 mg kg\(^{-1}\) was administered at induction, and supplementary doses (0.05–0.15 mg kg\(^{-1}\)) were given to maintain neuromuscular blockade during the measurements of FRC.

Volume controlled ventilation was provided by a ventilator (Servo Ventilator 900 B) set to deliver 10 b.p.m., and the tidal volume was adjusted to keep the end-tidal \(P_{CO_2}\) at around 4.5 kPa.

SUMMARY

A gas washout technique was used to measure the functional residual capacity (FRC) in eight patients during anaesthesia for cardiac surgery. The patients were anaesthetized with droperidol, fentanyl and nitrous oxide, alcuronium was given and the lungs were ventilated with a volume controlled ventilator. FRC was measured at three stages: before skin incision, after sternotomy but before cardiopulmonary bypass, and after closure of the sternum. The pleural cavities were intact in all patients during the operation. FRC before skin incision was 1.7±0.5 litre (mean±1 SD). A 55% mean increase in volume was noted after sternotomy and placement of the sternal retractor (\(P < 0.001\)). Mean FRC after sternal closure was 16% lower than the preincision value (\(P < 0.05\)). Arterial \(P_{O_2}\) was measured in 22 other patients who underwent coronary artery bypass surgery and in whom \(F_{O_2}\) was 0.5. \(P_{O_2}\) increased significantly when the sternum was opened, but decreased after cardiopulmonary bypass. There was a further significant decrease on closure of the sternum.

Insufflation time was 25% and the end-inspiratory pause 10% of the ventilatory cycle. End-expiratory airway pressure was zero. During CPB the tidal volume was reduced to 100–200 ml. Otherwise, the settings on the ventilator were changed only to give a few large breaths at the end of the tracer gas washout (see below).

The chest was opened via a median sternotomy. A self-retaining sternal retractor was used. The pleura was not opened in any patient. Moderate systemic hypothermia (minimum blood temperature 28 °C) and cold cardioplegia were used during CPB. The heart–lung machine (Stöckert, Munich) with a membrane blood oxygenator (Optiflo II; Cobe, Lakewood, Colorado) was
primed with Ringer’s solution 2 litre and 15% mannitol 0.25 litre. Before terminating CPB, the patients were rewarmed to a rectal temperature of 34 °C. After CPB an anaesthetic bag was used to expand the lungs with a few large breaths. This was repeated on sternal closure. One 28-F drain and one 16-F drain were placed in the mediastinum and in the pericardium, respectively. At the end of operation the fluid balance was calculated by adding the amount of crystalloids, colloids and blood given and subtracting the residual volume in the heart–lung machine, the urine output and the estimated blood loss.

Before each measurement of FRC, the absence of any leaks was verified and the tracheal and stomach tubes were suctioned. Measurements were obtained using a tracer gas washout technique which used sulphur hexafluoride (SF₆) as the tracer gas. A brief account of the technique is given here: SF₆ was washed in until a stable alveolar concentration of approximately 0.5% was reached. The concentration was measured by an infrared SF₆ analyser (Jonmarker, Castor et al., 1985). The transducer of the analyser was placed between the patient and the Y-piece of the ventilator tubing. The high sensitivity and rapid response of the analyser made it possible to calculate the expired volume of SF₆ in each breath from signals representing SF₆ concentration and expired flow (obtained from the expiratory flowmeter of the ventilator). Washout was considered complete when the mean expired SF₆ concentration was less than 0.001%. The total volume of SF₆ washed out was calculated by adding the expired SF₆ volume of all breaths during washout. Since the alveolar concentration at the end of washin was known, the lung volume could be calculated. The technique has been tested in a model lung and in patients without obstructive lung disease (Jonmarker, Jansson et al., 1985). In the present study the completeness of washout was tested on 14 occasions in six patients by giving four to eight breaths of double the ordinary tidal volume at the end of washout.

The first measurement of FRC was obtained within 1 h of induction, while the patient was being prepared for surgery. The next measurement was undertaken shortly before CPB with the sternal retractor in place. This measurement was timed so that the work of the surgeon would not interfere with the movement of the lungs. The last determination was carried out following the closure of the sternum and during skin suture. Since the SF₆ analyser is disturbed by electrocautery, which was needed for haemostasis after CPB, FRC was not measured in the period between CPB and sternal closure. All measurements were undertaken with the patient in the supine position on a horizontal operation table, and most measurements were determined in duplicate. The obtained value was corrected for rebreathing (Jonmarker, Jansson et al., 1985), converted to BTPS and apparatus deadspace (75–120 ml, depending on the size of the heat–moisture exchanger) was subtracted.

Arterial blood was analysed for Po₂, Pco₂ and pH. Samples were analysed within 5 min of sampling at 37 °C on an ABL2 blood-gas analyser (Radiometer, Copenhagen). Blood-gas tensions were measured in all patients after the induction of anaesthesia and at the end of surgery. Additional measurements of blood-gas tensions were obtained in seven of the patients immediately before releasing the sternal retractors, and 5–10 min after sternal closure. However, because of varying FIo₂ (table I) and the small number of patients, the effect of sternal closure on arterial oxygenation was difficult to evaluate. Therefore, the changes in arterial oxygenation during sternal opening and closure were subsequently studied in 22 other patients undergoing coronary bypass surgery in whom the pleura was intact. Otherwise, they were unselected. In these patients, FIo₂ was set at 0.5 during the whole procedure.

Statistics

Student’s two-sided paired t test was used. Probability values less than 0.05 were considered to indicate statistical significance.

RESULTS

The results of the FRC measurements are shown in figure 1. Individual values for fluid balance and arterial Po₂ are shown in table I. Values of FRC at the three stages were 1.7 ±0.5, 2.6 ±0.7 and 1.4 ±0.5 litre (mean ±1 SD), respectively. The mean increase in FRC, between the measurement taken before skin incision and the one taken after sternotomy was 55% (P < 0.001) (fig. 1). The mean decrease between the pre-incision value and that taken after sternal closure was 16% (P < 0.05). Twenty of the 24 (3 × 8) measurements were done in duplicate with a mean difference between the two determinations of 4.5% (range 0.6–12.9%). The time needed to complete
TABLE I. Patient characteristics and results. FVC = forced vital capacity; FEV₁ = forced expired volume (1 s); CABG = coronary artery bypass grafting; Aort. valve = aortic valve replacement. *F₁O₂ = 0.5; all other blood samples were taken with F₁O₂ = 0.35

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (m)</th>
<th>FVC (litre, BTPS)</th>
<th>FEV₁/FVC</th>
<th>Operation</th>
<th>Fluid balance (litre)</th>
<th>Before sternal closure</th>
<th>After sternal closure</th>
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<tbody>
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<td>1</td>
<td>56</td>
<td>95</td>
<td>1.80</td>
<td>4.5</td>
<td>0.78</td>
<td>CABG</td>
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<td>—</td>
<td>15.5</td>
</tr>
<tr>
<td>2</td>
<td>61</td>
<td>80</td>
<td>1.75</td>
<td>4.2</td>
<td>0.93</td>
<td>CABG</td>
<td>+0.1</td>
<td>17.3</td>
<td>13.9</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>81</td>
<td>1.85</td>
<td>4.9</td>
<td>0.75</td>
<td>Aort. valve</td>
<td>+2.5</td>
<td>23.6</td>
<td>18.5</td>
</tr>
<tr>
<td>4</td>
<td>53</td>
<td>92</td>
<td>1.69</td>
<td>3.9</td>
<td>0.77</td>
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<td>+0.1</td>
<td>12.1</td>
<td>9.7</td>
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<td>5</td>
<td>49</td>
<td>98</td>
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<td>3.7</td>
<td>0.74</td>
<td>CABG</td>
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<td>16.7</td>
<td>18.6</td>
</tr>
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<td>49</td>
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<td>1.74</td>
<td>4.0</td>
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<td>28.0</td>
<td>15.8</td>
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<td>70</td>
<td>1.76</td>
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<td>12.6*</td>
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<td>8</td>
<td>67</td>
<td>67</td>
<td>1.69</td>
<td>3.3</td>
<td>0.81</td>
<td>Aort. valve</td>
<td>+1.3</td>
<td>18.4</td>
<td>22.7*</td>
</tr>
</tbody>
</table>

Fig. 1. Changes in FRC in eight patients, Nos: 1 (●); 2 (x); 3 (∆); 4 (■); 5 (□); 6 (○); 7 (∇); 8 (○).

washout varied from 2 to 8 min. Four to eight large tidal volume breaths towards the end of washout yielded at most 0.19 ml SF₆, corresponding to an increment in measured FRC of less than 3 ml on each occasion (mean value 19 ml). The two patients (Nos 6 and 7 in table I) with the most marked decreases in FRC between the pre-incision measurement and the one taken after sternal closure (0.5–0.6 litre) both accumulated more than 2 litre of fluid during the operation. However, when values from all patients were considered, the correlation between fluid balance and change in FRC was not statistically significant. There was no statistically significant correlation between the change in FRC and the duration of CPB or the duration of anaesthesia.

In the eight patients in whom FRC was measured, P_aO₂ was 17.1 ± 6.5 kPa (range 11.3–30.0) after the induction of anaesthesia. There was no statistically significant change in P_aO₂ on sternal closure in the six patients in whom the F₁O₂ was unchanged (table I). P_aO₂ values in the 22 patients studied subsequently are shown in table II. In these, mean P_aO₂ increased slightly during sternal opening (P < 0.05) and decreased during sternal closure (P < 0.05). Moreover, P_aO₂ after CPB was significantly less than before CPB (P < 0.001). Mean P_aCO₂ values at the four measurements were 5.0, 5.1, 5.0, and 5.0 kPa, respectively.

TABLE II. Arterial oxygen tension (P_aO₂) in 22 patients undergoing coronary artery bypass grafting. F₁O₂ was 0.5. Mean values ± 1SD. *P < 0.05, ***P < 0.001

<table>
<thead>
<tr>
<th></th>
<th>Before surgery</th>
<th>Open sternum, before CPB</th>
<th>Open sternum, after CPB</th>
<th>Closed sternum</th>
</tr>
</thead>
<tbody>
<tr>
<td>P_aO₂ (kPa)</td>
<td>26.3 ± 6.6</td>
<td>28.1 ± 5.2</td>
<td>21.7 ± 9.2</td>
<td>18.7 ± 6.5</td>
</tr>
<tr>
<td></td>
<td>*</td>
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</table>
A new method to measure FRC was used in the present study. The system has given accurate and reproducible results in studies on a model lung, and in human studies during both air and nitrous oxide in oxygen ventilation. However, as with other gas dilution methods, SF₆ washout will not measure the volume of gas in pulmonary units which do not communicate with the airway during the period of measurement. An underestimation of the lung volume may occur either because the tracer gas fails to reach pulmonary units during washout, or because it fails to leave pulmonary units during washout. Anaesthesia has been reported to promote closing of small airways (Gilmour, Burnham and Craig, 1976; Hedenstierna, McCarthy and Bergström, 1976) and would thus increase the time needed for both wash in and washout of tracer gas. In patients with healthy lungs, however, the underestimation with gas dilution methods seems to be fairly small. Hedenstierna, Järnberg and Gottlieb (1981) found that the thoracic gas volume (TGV), measured by body plethysmography was 240 ml greater, on average, than the communicating gas volume (CGV), measured by nitrogen washout to the 2% level in a study of anaesthetized patients of similar age to those in the present study. The real difference between TGV and CGV may have been even smaller, as these authors used a fairly high correction value for nitrogen evolved from body tissues when calculating CGV. Washout seems to have been complete in the present study, as very little SF₆ was recovered when a number of large breaths were given at end of washout. Of course, an underestimation of the true lung volume, as a result of incomplete washin, cannot be ruled out, but it appears unlikely that this would explain the marked changes in lung volume that were observed.

The increase in lung volume on sternal retraction was obviously a result of the change in the configuration of the chest. It seems likely that the retraction of the sternum increases the trans-pulmonary pressure. Incidentally, the increase in end-expiratory lung volume may be inferred from a simple observation: if volume-controlled ventilation is used, a transient decrease in expired tidal volume will be seen in most patients when the sternal edges are separated. The reverse is seen as the sternum is closed. We can only speculate about how the lung expansion occurring during sternal retraction was distributed. There are studies which suggest that changing the shape of the pleural cavities has rather little effect on the regional distribution of alveolar distending forces (Vawter, Matthews and West, 1975). Furthermore, the effect on the two pleural cavities was probably symmetrical. Therefore, we believe that the effects on the alveoli, brought about by sternal retraction, were similar to the effects of another intervention, which increases lung volume, namely the application of a positive end-expiratory pressure (PEEP). The resultant shape of the lung may not be the same, however. Also, the circulatory effects of PEEP and of sternal retraction are different (see below).

An increase in FRC can be expected to improve arterial oxygenation only if it is associated with an improvement in V/Q balance in regions with a low V/Q and if cardiac output does not decrease markedly. Thus, an increase in FRC improves oxygenation in some patients, but not in others (Craig et al., 1971; Falke et al., 1972; Wyche et al., 1973; Heneghan, Bergman and Jones, 1984). Unstable circulatory conditions and varying $F_{1O_2}$ make it difficult to interpret the effect of sternal opening and closure on arterial oxygenation in the eight patients in whom we measured FRC. In the 22 patients whom we studied subsequently, however, $P_{aO_2}$ increased significantly after sternal opening and decreased after sternal closure. This agrees well with the previously observed changes in FRC. However, opening the sternum tends to increase cardiac output (Matsumoto et al., 1980) and sternal closure results in a significant decrease in cardiac output (Matsumoto et al., 1980; Jögi and Werner, 1986). Therefore, the observed changes in oxygenation may have been secondary to changes in pulmonary perfusion. We measured rectal and not oesophageal temperature. However, since rectal temperature is not always the same as the temperature of the blood in the lung capillaries, particularly not during the first h after CPB, we preferred not to correct the $P_{aO_2}$ values for temperature. This has probably not introduced any systematic errors in the assessment of blood-gas tensions as oesophageal temperature, which probably more closely reflects the temperature in the lung capillaries, does not seem to change much between the stages during which we measured $P_{aO_2}$. Thus, in 24 adult patients previously operated on in our hospital, mean oesophageal temperature was 35.8, 35.5, 35.6, and 35.5 °C, at the four stages depicted in table II
(R. Fletcher, personal communication). Our findings, in respect of arterial oxygenation, do not support those of Norlander, Bernhoff and Norden (1969) who found no significant changes in venous admixture on sternal opening or on sternal closure. However, patients in whom the pleura was opened were included in their study, so the data may not be directly comparable with ours.

There are a number of possible explanations for the 16% mean decrease in FRC between the measurement before skin incision and the one after sternal closure. First, the fluid load during and after CPB may have increased the extravascular fluid volume in the lungs as well as the intrathoracic circulating blood volume. Second, the drains and minor bleeding may have decreased the available space inside the thorax. Finally, the reduction of ventilation during CPB and the possible respiratory effects of CPB, for example, on alveolar surfactant (Mandelbaum and Giammona, 1964), may have induced alveolar collapse. In fact, our experience is that postoperative atelectasis is a frequent finding after cardiac surgery.

We conclude that sternal opening and retraction causes a significant increase in FRC in patients with intact pleural cavities. If one of the pleural cavities is opened, one may expect marked asymmetry between the lungs in respect of volume during sternal retraction. A slight decrease in FRC can be expected after cardiac surgery.

The results may have some bearing on the use of PEEP during cardiac surgery. Although we have not specifically studied the effects of PEEP, the increased FRC and $P_aO_2$ at sternal retraction suggest that end-expiratory pressure need not be increased routinely when the heart is exposed before CPB.

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REFERENCES


