CHANGES IN COLLOID OSMOTIC PRESSURE AND PLASMA ALBUMIN CONCENTRATION ASSOCIATED WITH EXTRACORPOREAL CIRCULATION

R. SANCHEZ DE LÉON, J. L. PATERSON AND M. K. SYKES

SUMMARY
Colloid osmotic pressure (COP), plasma albumin concentration, haematocrit, and blood-gas tensions were measured in 16 patients undergoing open-heart surgery with cardiopulmonary bypass. The use of crystalloid priming and cardioplegia solutions resulted in a 60% decrease in COP, a 48% decrease in plasma albumin concentration and a 35% decrease in haematocrit. These measurements had returned to pre-perfusion values 6 h after the end of surgery. The alveolar–arterial $\bar{P}_{O_2}$ difference increased significantly after bypass and returned to pre-perfusion values within the same time scale. Right-to-left shunt increased from 7.9 to 10.3% 30 min after the end of bypass. It is concluded that, in the absence of an increase in left atrial pressure, marked decreases in COP can be tolerated without the occurrence of pulmonary oedema.

In the early days of open-heart surgery the priming volumes of the extracorporeal circuits were large and the primes consisted mainly of whole blood. The subsequent introduction of bubble and membrane oxygenators with small priming volumes permitted crystalloid or colloid primes to be used, thereby minimizing the hazards associated with the use of homologous blood.

Studies of respiratory function after perfusion with a rotating-disc oxygenator primed with two-thirds whole blood and one-third lactated Ringer's solution showed that there was no significant increase in alveolar–arterial oxygen tension difference ($\bar{P}_{A_{O_2}} - \bar{P}_{A_{O_2}}$) or total venous admixture in the period immediately after bypass (Seed, Sykes and Finlay, 1970; Sykes et al., 1970). Routine blood-gas monitoring indicated also that the introduction of bubble oxygenators primed with 1–3 litre of lactated Ringer's solution had no significant effect on pulmonary function.

In recent years myocardial preservation has been achieved by the perfusion of 1–2 litre of iced cardioplegia solution into the coronary circulation. This fluid is usually allowed to drain into the perfusion circuit so that further haemodilution occurs. Patients subjected to this technique commonly display signs of widespread peripheral oedema. In addition, some patients have developed severe arterial hypoxaemia during the period after bypass despite the administration of inspired oxygen concentrations of 50–60% and mechanical ventilation with a positive end-expiratory pressure of 0.5–1.0 kPa. Although there have been no obvious signs of pulmonary oedema on the chest radiograph after surgery, and there have been no gross changes in airway resistance or total thoracic compliance, it seemed possible that the arterial hypoxaemia might have been caused by an accumulation of lung water. Since the arterial hypoxaemia had not been associated with a significant increase in left atrial pressure we decided to study the changes in colloid osmotic pressure and plasma albumin concentration associated with this technique of cardiopulmonary bypass.

PATIENTS AND METHODS
Studies were performed on 16 patients aged between 43 and 63 yr, who were being subjected to open-heart surgery with cardiopulmonary bypass. Fourteen patients were male and two female. Twelve patients underwent coronary artery vein bypass grafts, two had mitral valve replacements and two had aortic valve replacements. The duration of operation varied between 185 and 390 min and the duration of bypass between 50 and 130 min. The surface area of the patients varied between 1.55 m$^2$ and 2.10 m$^2$ (mean 1.99 m$^2$).

The patients were premedicated with papaveretum and hyoscine supplemented, where
necessary, with small doses of droperidol or lorazepam. The anaesthetic consisted of thiopentone, pancuronium, phenoperidine, nitrous oxide and oxygen, the inspired oxygen concentrations being adjusted to produce an arterial $P_{O_2}$ ($P_{aO_2}$) in the range 13–20 kPa. Ventilation was controlled with an Engström 300 ventilator at frequencies of 12–14 b.p.m. Tidal volumes of 500–700 ml were required to maintain an arterial $P_{CO_2}$ ($P_{aCO_2}$) in the range of 4–5 kPa, and an end-expiratory positive pressure of 0.5 kPa was added to maintain a normal functional residual capacity (FRC) when the chest was open. Ventilation was continued during the bypass period using a mixture of oxygen 1–2 litre and air 2 litre. The lungs were hyperinflated several times at the end of bypass. Controlled ventilation was maintained for 3–18 h after operation and discontinued when the cardiovascular system was stable.

The operation was performed through a median sternotomy, the pleura being opened accidentally on only one occasion. The patients were cooled to 28–30 °C during cardiopulmonary bypass with a Rygg-Kyvsgaard bubble oxygenator. The circuit was primed with 1.5–2.0 litre of lactated Ringer's solution and a small quantity of sodium bicarbonate solution. The basal flow rate was 2.4 litre m$^{-2}$ body surface area, but this was increased by 30–40% when the haematocrit was low. Additional quantities of lactated Ringer's solution (1–2 litre) were added during bypass to maintain the circulating volume and if the haematocrit decreased to less than 20–25%, homologous blood (stored for 1–2 days in citrate-phosphate-dextrose anticoagulant) was added to the circuit. When the oesophageal temperature had been decreased to 30 °C, 1 litre of cardioplegia solution (1 litre of lactated Ringer's containing MgCl$_2$.6H$_2$O 16 mmol, KCl 16 mmol and procaine HCl 1 mmol) was injected to the coronary circulation to cool the heart to less than 10 °C. Further increments of 0.5–1.0 litre were required in nine patients. The solution drained into the left ventricular vent and coronary sinus and so mixed with the other fluid in the perfusion circuit. Urine output was measured every 30 min and furosemide administered whenever the output decreased to less than 1 ml min$^{-1}$.

The volume of blood transfused in the period after bypass and in the intensive care unit was determined by measured blood loss and right atrial pressures. Arterial pressure was maintained close to preoperative values by the use of vasodilator or inotrop drugs.

**Measurements.** Arterial blood-gas tensions and pH, serum potassium concentration and haematocrit were monitored throughout the procedure. Measurements of colloid osmotic pressure (COP), serum albumin concentration and haematocrit were made on blood samples drawn at the induction of anaesthesia, just before bypass, 10 min after commencing bypass and every 30 min thereafter until the end of bypass. Additional samples were taken just before the patient was returned to the Intensive Care Unit (usually 1–2 h after the end of bypass) and 3, 6 and 12 h later. The samples were centrifuged immediately and all measurements completed within a few hours of sampling.

COP was measured with a pressure transducer system which was a modification of the osmometer described by Aukland and Johnsen (1974). The unit consisted of two cylindrical metal blocks with a semipermeable membrane sandwiched between them (fig. 1). The lower block replaced the threaded dome of a standard Consolidated Dynamics differential pressure transducer (4-327-L222) and had a central channel which was filled with boiled saline (to exclude air bubbles). The membrane was sealed between the two metal blocks by means of rubber O-rings. The upper block contained a central channel with a volume of 0.3 ml. This served as a reservoir for the solution under test. When a solution with a greater osmotic pressure than saline was placed in the reservoir it created an osmotic pressure gradient across the membrane which forced the
transducer diaphragm upwards. The movement of the diaphragm was detected by the strain gauge in the transducer and the signal from the transducer was then amplified and displayed on a Devices heated-stylist recorder.

The membrane chosen for these measurements was Amicon PM 10. This had a typical rejection of >98% for albumin whilst the typical maximal deionized water flow was 1–4 ml cm$^{-2}$ min$^{-1}$. The membrane had a rapid response and readings quickly returned to zero when physiological saline was placed in the reservoir (fig. 2).

The zero was adjusted before each measurement when the cuvette contained saline. The gain control was also adjusted before each measurement by applying step increases in pressure of 1, 2 and 2.5 kPa to the back of the transducer diaphragm through the reference pressure inlet. This pressure was generated by injecting air into a reservoir connected both to the transducer and to a saline manometer. This method of calibration stressed the diaphragm in the same direction as that produced by a hyperosmolar solution and confirmed that the output of the transducer was linear to within ±0.01 kPa. Hysteresis was also within these limits. Assessments of the functioning of the oncometer and on the reproducibility of the measurements were performed with a reference solution containing albumin 50 g litre$^{-1}$ and globulin 33 g litre$^{-1}$ (Sigma Laboratories), and with a second albumin solution containing 45 g litre$^{-1}$ in saline. The former solution read 3.6 (±0.1) kPa and the latter 2.4 (±0.1) kPa on our oncometers. These solutions were split into samples of 1 ml and kept in a refrigerator at −10°C, one tube of each solution being used for reproducibility checks twice a day. All the measurements of COP were made at room temperature (23–26°C) and the values were corrected to the patient’s temperature from the equation:

$$\text{COP}_{t_2} = \frac{273 + t_2}{273 + t_1} \times \text{COP}_{t_1}$$

where $t_1$ and $t_2$ were the respective temperatures of the room and the patient in degrees Celsius.

No correction was made for the pH or $P_{CO_2}$ of the plasma. The sample was covered by a plastic cover during the measurement and duplicate measurements were obtained on each sample by running two oncometers in parallel. Duplicates always agreed within ±0.1 kPa throughout the study.

Albumin concentrations were measured in duplicate by the Bromocresol green method (Sigma Chemical Co. P.O. Box 14508, St Louis, Missouri

![Figure 2](https://example.com/figure2.png)

**Fig 2.** Recordings from oncometers A and B. C = pressure calibration: -1.5 and -2.5 kPa. Three to four aliquots of sample were inserted into the well on each oncometer between the arrows. Oncometer B (with a new membrane) responded more quickly than oncometer A, but both reached the same final value (-2.2 kPa) after 20–25 min. Both wells were then flushed with saline to check that there had been no zero drift.
and haematocrit was determined by the standard method using a microcentrifuge. Inspired oxygen tension ($P_{O_2}$) was measured by an oxygen electrode or paramagnetic analyser and blood-gas tensions and pH by a Radiometer ABL1 automated system which was checked daily by tonometered blood samples (Selman and Tait, 1976). In six patients in this group and 11 other patients measurements of total venous admixture were made using arterial and pulmonary artery samples obtained by direct puncture at operation. These were analysed for oxygen content ($C_{aO_2}$ and $C_{vO_2}$) by the Lex-O2-Con analyser (Selman, White and Tait, 1975) and the values used to provide the arteriovenous oxygen content difference in the modified shunt equation:

$$\frac{Q_s}{Q_t} = \frac{(C_{cO_2} - C_{aO_2})}{(C_{cO_2} - C_{aO_2}) + (C_{aO_2} - C_{vO_2})}$$

The difference between the end-pulmonary capillary and arterial oxygen contents ($C_{cO_2}$ and $C_{aO_2}$) in both numerator and denominator was calculated from the ideal alveolar $PO_2$ ($P_{A02}$), the arterial $PO_2$ and the arterial pH. These values were used to derive end-pulmonary capillary and arterial saturations by reference to the standard dissociation curve (Severinghaus, 1966). Content was calculated from the haemoglobin, saturation and $PO_2$ assuming that each gram of haemoglobin combined with 1.34 ml of oxygen. Ideal alveolar $PO_2$ was calculated from the simplified equation:

$$P_{A02} = P_{102} - P_{aCO_2} \frac{0.8}{0.8}$$

The statistical analysis was performed using an analysis of variance and Student's paired t tests.

RESULTS

The average volume of Ringer's lactate solution administered during bypass was 2.56 litre (range 2.0–3.8 litre) and the average volume of cardioplegia solution was 1.5 litre. The total quantity of crystalloid administered during bypass therefore ranged from 2.5–5.8 litre. One patient required 2 units of blood during bypass because of low haematocrit. An average of 2.52 litre (range 1.5–5.5 litre) of blood was infused during operation and a further 2.27 litre (range 0.99–2.36 litre) was given in the 24 h after operation. Crystalloid administration in the latter period averaged 0.60 litre. Urine output averaged 1.07 litre (range 0.67–1.85 litre) during and after bypass and 1.42 litre (range 0.2–2.82 litre) in the 24 h after operation.

The patients remained in the intensive care unit for 20–28 h and were mechanically ventilated for 3–18 h (mean 14 h) after operation. There was no gross radiological evidence of pulmonary oedema in the x-rays taken in the supine position immediately after operation. Four patients required dopamine after operation. Two patients died after the period of study, one from intractible arterial hypertension and another from cerebral embolic problems.

There were no significant differences between the induction and pre-bypass COP measurements. There was a significant decrease in COP ($P<0.05$) from a mean of 2.69 kPa before bypass to 1.08 kPa 10 min after instituting bypass (table I and fig. 3). There was little change during bypass. In the 13
patients studied 10, 40 and 70 min after commencing bypass the means were 1.11, 1.13, and 1.21 kPa respectively. In the four patients in whom samples were obtained after 100 min on bypass the mean was 1.04 kPa compared with 1.25 kPa at the start of bypass. However, for the group as a whole the COP had increased to 1.75 kPa at the end of bypass and had reached pre-bypass values by the 6th hour after operation. The pattern of change of albumin concentration was similar. Haematocrit decreased from 40.4% before bypass to 26.3% on bypass but had returned to pre-bypass values by the 3rd hour following operation.

The alveolar–arterial $P_O_2$ difference ($P_{A_O_2} - P_{A_O_2}$) increased from a mean of 14.1 kPa before bypass to 20.3 ± 4.1 kPa about 30 min after the end of bypass ($P < 0.05$). Mean ($P_{A_O_2} - P_{A_O_2}$) was 17.1 kPa at the end of operation, 15.8 kPa 3 h later and had returned to 12.5 kPa 6 h after the end of operation. In the 17 patients in whom intrapulmonary shunt was measured before and 30 min after bypass (table II) there was increase from a mean of 7.9 ± 3.2% to 10.3 ± 4.5% ($P < 0.05$). The majority of the patients showed only a small increase in shunt, but in one patient the shunt increased from 9 to 23%. This patient required inspired oxygen concentrations of more than 50% and the application of a positive end-expiratory pressure of 5–10 cm H₂O for a period of 10 h after bypass.

**DISCUSSION**

These studies have shown that cardiopulmonary bypass with a crystalloid prime and additional cardioplegic solution decreased COP to 40%, albumin concentration to 53% and haematocrit to 65% of the pre-perfusion values. The addition of further crystalloid solution during bypass tended to maintain those values throughout bypass periods of up to 100 min, but after bypass the values returned towards normal. In the majority of patients there was an increase in ($P_{A_O_2} - P_{A_O_2}$) and only a small increase in intrapulmonary shunt in the period immediately after bypass, but one patient developed a marked increase in shunt for a period of 10 h after operation.

Ladegaard-Pedersen (1967) studied some of the factors which affect the measurement of COP in patients. He found that there was a daily variation of ±10.5% in patients who were confined to bed, and he also showed that the COP of such patients was 0.4–0.5 kPa less than when they were ambulant. Our initial values were usually taken a few minutes

### Table I. Changes in colloid osmotic pressure, plasma albumin concentration and haematocrit during operation and the period after operation (mean ± SEM). A = induction; B = before bypass; C = 10 min after starting bypass; D = 30 min later; E = end of bypass; F = end of operation; G, H, and I = 3, 6, and 12 h after end of operation

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>G</th>
<th>H</th>
<th>I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colloid osmotic pressure (kPa)</td>
<td>2.73</td>
<td>2.69</td>
<td>1.08</td>
<td>1.19</td>
<td>1.75</td>
<td>2.20</td>
<td>2.44</td>
<td>2.68</td>
<td>2.87</td>
</tr>
<tr>
<td></td>
<td>±0.25</td>
<td>±0.15</td>
<td>±0.16</td>
<td>±0.15</td>
<td>±0.17</td>
<td>±0.19</td>
<td>±0.20</td>
<td>±0.30</td>
<td>±0.15</td>
</tr>
<tr>
<td>Haematocrit (%)</td>
<td>40.4</td>
<td>40.1</td>
<td>26.3</td>
<td>26.4</td>
<td>33.1</td>
<td>36.6</td>
<td>39.1</td>
<td>39.6</td>
<td>41.5</td>
</tr>
<tr>
<td></td>
<td>±3.9</td>
<td>±4.0</td>
<td>±3.8</td>
<td>±3.6</td>
<td>±3.4</td>
<td>±4.0</td>
<td>±3.7</td>
<td>±2.5</td>
<td></td>
</tr>
<tr>
<td>Albumin (g litre⁻¹)</td>
<td>37.0</td>
<td>34.0</td>
<td>18.0</td>
<td>19.0</td>
<td>26.0</td>
<td>31.0</td>
<td>31.0</td>
<td>34.0</td>
<td>37.0</td>
</tr>
<tr>
<td></td>
<td>±1.0</td>
<td>±1.2</td>
<td>±1.1</td>
<td>±1.0</td>
<td>±1.1</td>
<td>±1.2</td>
<td>±1.3</td>
<td>±1.2</td>
<td>±1.2</td>
</tr>
<tr>
<td>($P_{A_O_2} - P_{A_O_2}$) (kPa)</td>
<td>—</td>
<td>14.1</td>
<td>—</td>
<td>—</td>
<td>20.3</td>
<td>17.1</td>
<td>15.8</td>
<td>12.5</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>±2.8</td>
<td>±1.1</td>
<td>—</td>
<td>—</td>
<td>±4.1</td>
<td>±4.7</td>
<td>±4.9</td>
<td>±4.7</td>
<td></td>
</tr>
</tbody>
</table>

### Table II. Arterial and mixed venous blood-gas tensions, haemoglobin (Hb) concentration, oxygen contents and percentage shunt before and 30 min after bypass in 17 patients

<table>
<thead>
<tr>
<th></th>
<th>$P_{A_O_2}$</th>
<th>$P_{A_CO_2}$</th>
<th>$P_{A_O_2} - P_{A_CO_2}$</th>
<th>$P_{V_O_2}$</th>
<th>$P_{V_CO_2}$</th>
<th>Hb (g dl⁻¹)</th>
<th>$C_{A_O_2}$</th>
<th>$C_{V_O_2}$</th>
<th>($C_{A_O_2} - C_{V_O_2}$)</th>
<th>Shunt (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>25.35</td>
<td>4.38</td>
<td>16.38</td>
<td>5.67</td>
<td>5.04</td>
<td>13.64</td>
<td>8.43</td>
<td>6.43</td>
<td>2.00</td>
<td>7.90</td>
</tr>
<tr>
<td></td>
<td>±6.04</td>
<td>±0.81</td>
<td>±5.87</td>
<td>±0.65</td>
<td>±0.83</td>
<td>±1.33</td>
<td>±0.83</td>
<td>±0.78</td>
<td>±0.43</td>
<td>±3.23</td>
</tr>
<tr>
<td>After</td>
<td>24.89</td>
<td>4.95</td>
<td>16.41</td>
<td>6.23</td>
<td>5.92</td>
<td>11.13</td>
<td>6.96</td>
<td>5.35</td>
<td>1.61</td>
<td>10.3</td>
</tr>
<tr>
<td></td>
<td>±5.84</td>
<td>±1.17</td>
<td>±5.77</td>
<td>±0.65</td>
<td>±1.56</td>
<td>±1.37</td>
<td>±0.93</td>
<td>±0.80</td>
<td>±0.34</td>
<td>±5.4</td>
</tr>
</tbody>
</table>
after the induction of anaesthesia and the pre-perfusion values about 1 h later, after the administration of 100–200 ml of Ringer’s lactate solution. Both means were smaller than the means of 3.9, 3.2 and 4.0 kPa reported by Losowsky, Alltree and Atkinson (1962), Ladegaard-Pedersen (1967) and English, Digerness and Kirklin (1971). However, our values were close to those reported by Marty and colleagues (1973). Since the latter authors used a membrane similar to ours it seems likely that the disparities were a result of differing membrane characteristics.

English, Digerness and Kirklin (1971) reported that COP decreased from a mean of 4.0 kPa before bypass to a mean of 2.5 kPa during bypass. Perfusion was carried out with a rotating-disc oxygenator and a prime of 4 units of acid–citrate–dextrose blood, 750 ml of 5% dextrose in 0.45% sodium chloride and 1250 ml of balanced salt solution (Normosol). The initial haematocrit of their perfusate was 30% and alternate units of blood and salt solution were given during the remainder of the bypass period. They observed that COP increased to a mean of 2.7 kPa 3 h after the end of bypass and then remained unchanged for 24 h. However, they noted that in the single patient with a bypass time of 180 min both the COP and total protein concentration remained less than that observed in the other patients during the period after bypass.

Further studies on patients subjected to cardiopulmonary bypass were reported by Marty and co-workers (1973). These authors investigated two primes. One contained 150 ml of 5% dextrose solution and 500 ml of acid–citrate–dextrose whole blood with a further 2500–3000 ml of balanced salt solution (Normosol) and 75 g of salt-poor albumin. Variable amounts of ACD blood (500–400 ml) were added during bypass. With the first prime mean oncotic pressure decreased by 41% from 2.7 kPa to 1.6 kPa and with the second it decreased by 42% from 3.1 kPa to 1.8 kPa. By the 4th hour after bypass COP was more than 90% of the pre-bypass value in both groups. Thus the intraoperative administration of an average of 155 g of salt-poor albumin did not significantly affect the average increase in oncotic pressure after operation.

English, Digerness and Kirklin (1971) found that total protein concentration decreased from 75 g litre$^{-1}$ to 48 g litre$^{-1}$ whilst Marty and colleagues (1973) found a similar decrease from a mean of 63 g litre$^{-1}$ to 40 g litre$^{-1}$. Although the pattern of change of COP is similar to the pattern of change in albumin concentration, the relationship between the two is not necessarily linear and in chronic situations it is not always possible to predict one from the other (Losowsky, Alltree and Atkinson, 1962). However, in our own studies a close correlation was observed between COP and both haematocrit and plasma albumin concentration (fig. 4). Thus, it appears that haemodilution with crystalloid solutions decreases the plasma protein concentration and COP and so may alter the Starling equilibrium across the capillary wall.

Although most of the patients studied had obvious peripheral oedema with swollen eyelids, distension of the tissues of the face, neck and limbs and pitting oedema over the sacrum, there was remarkably little evidence of alveolar or interstitial oedema. $(P_{A_{2}} - P_{F_{2}})$ increased after bypass but returned to pre-bypass values during the ensuing 6 h. An increase in $(P_{A_{2}} - P_{F_{2}})$ during ventilation with 50% oxygen could be the result of an increase in intrapul-

![Figure 4. Correlation (regression lines with 95% tolerance limits) of haematocrit (Hct) and plasma albumin concentration against colloid osmotic pressure (COP).](https://academic.oup.com/bja/article-abstract/54/4/465/264628)
COLLOID OSMOTIC PRESSURE AND PLASMA ALBUMIN WITH BYPASS

monary shunt, but could also be caused by a decrease in mixed venous $P_{O_2}$, an alteration in haemoglobin concentration or a shift of the oxygen dissociation curve. In order to clarify the significance of the increase in $(P_{A_o} - P_{a_o})$ we made measurements of intrapulmonary shunt using direct measurements of arterial and mixed venous oxygen contents. Only one of the 17 patients so studied showed an increase in shunt of more than 4%. This patient had received 2 litre of cardioplegia solution and developed a shunt of 23% 30 min after bypass. The impairment in gas exchange persisted for 10 h after the end of the operation but responded to treatment with 50% oxygen and a positive end-expiratory pressure of 5–10 cm H$_2$O. Although this patient showed no clinical or radiological evidence of pulmonary oedema, it is believed that the increased shunt was caused by terminal airway closure resulting from interstitial oedema.

Muir and colleagues (1975) infused 2 litre of normal saline into normal seated subjects at a rate of 100 ml min$^{-1}$ and found that these subjects had an increase in closing volume without any change in their lung compliance or flow–volume characteristics. Furthermore, in the two subjects in whom closing volume exceeded the functional residual capacity after infusion there was also a decrease in arterial $P_{O_2}$. Narrowing of the small terminal airways has little effect on airway resistance since the total cross sectional area of these airways is large and the velocity of airflow small. Terminal airway narrowing or closure may therefore occur without obvious changes in airway resistance (Hogg et al., 1972; Hauge, Bø and Waaler, 1975). Thus there is experimental evidence to support the suggestion that the decrease in colloid osmotic pressure might have increased fluid transudation into the interstitial space, many investigators have found that the application of PEEP has rapidly corrected the arterial hypoxaemia in most of the patients who have developed a low arterial $P_{O_2}$ after bypass.

The mechanisms which protect the lung from pulmonary oedema when COP is decreased have been studied in baboons subjected to acute isovolaemic decreases in plasma oncotic pressure by plasmapheresis (Zarins et al., 1978). In these experiments plasma COP was decreased by 76% (from 2.6 to 0.6 kPa) whilst pulmonary capillary pressure was maintained at a normal value. This resulted in a seven-fold increase in pulmonary lymph flow and, although there was marked peripheral oedema, there was no increase in the wet lung/dry lung weight ratio. The increased lymph flow in these experiments was accompanied by a reduction in the lymph albumin concentration and by a decrease in lymph/plasma COP ratio which would have further protected against pulmonary oedema formation. It is possible that a similar change in the lymph/plasma COP ratio would have been observed in the experiments of Zarins and co-workers (1978) if the measurements had been prolonged further.

An additional factor which must be considered is tissue pressure. In most tissues the interstitial space is easily distended and can therefore accommodate relatively large volumes of fluid with little change in pressure (Guyton and Lindsey, 1959). In the lung the interstitial space around the capillaries is bounded by tight epithelial junctions and a collagen framework so that a relatively small increase in interstitial fluid volume probably leads to a significant increase in interstitial pressure (Fung, 1974; Guyton, Taylor and Granger, 1975). This in turn would tend to decrease fluid transfer from the capillary to the interstitial space.

Two other factors probably minimized the occurrence of pulmonary oedema in these patients. First, the lung was not perfused during the period of maximal haemodilution. During the period immediately after bypass COP rapidly increased so that the period during which the lung was exposed to a significant decrease in COP was limited. Second, a positive end-expiratory pressure was maintained during anaesthesia.

The mechanism by which PEEP improves oxygen transfer in patients with pulmonary oedema is still not known. Although Barach, Martin and Eckman (1938) and Ashbaugh and colleagues (1969) originally postulated that an increase in alveolar pressure might alter the pressure gradients across the alveolar capillary membrane and so decrease the transudation of fluid into the interstitial space, many investigators have found that the application of PEEP results in no change or even an increase in lung water (Wagner et al., 1961; Mellins et al., 1969; Caldini,


Ashbaugh, D G , Petty, T. L , Bigelow, D. B., and Hams, T

MODIFICATIONS DE LA PRESSION COLLOIDO-OSMOTIQUE ET DE LA CONCENTRATION PLASMATIQUE D'ALBUMINE INDUITES PAR LA CIRCULATION EXTRACORPORELLE

Nous avons mesuré la pression colloïdo-osmotique (PCO), la concentration plasmatique d'albumine, l'hématocrite et les gaz du sang chez 16 patients subissant un acte de chirurgie à cœur ouvert sous circulation extracorporelle (CEC). L'utilisation de solutions cristalloïdes de commencement et de cardioplegie a entraîné une diminution de 60% de la PCO, une diminution de 48% de la concentration plasmatique d'albumine et une diminution de 35% de l'hématocrite. Ces paramètres avaient retrouvé leur valeur de départ 6 h après la fin de l'acte chirurgical, de façon significative après la CEC et étaient de retour à leurs valeurs de départ dans le même délai. Le shunt droit gauche passait de 7,9% à 10,3%, 30 min après la fin de la CEC. Nous en concluons que, en l'absence d'augmentation de la pression dans l'oreillette gauche, des diminutions importantes de la PCO peuvent être tolérées sans que l'on voit apparaître d’œdème pulmonaire.

RESUME

ZUSAMMENFASSUNG


SUMARIO

Se midieron la presión osmótica coloidal (POC), la concentración de albúmina en el plasma y las tensiones hemotocritas y gaseosas en 16 pacientes sometidos a intervención quirúrgica a pecho abierto con desviación cardiopulmonar. El uso de imprimación cristaloidea y de soluciones cardiopílegicas tuvieron como resultado una disminución del 60% en la POC, del 48% en la concentración de albúmina en el plasma y de un 35% en la tensión hematocritica. Las mediciones efectuadas volvieron a adquirir los valores previos a la perfusión seis horas después de la intervención quirúrgica. La diferencia de $P_{O2}$ alveolo-arterial aumentaron de forma significativa después de la desviación y volvieron a adquirir los valores previos a la perfusión dentro de la misma escala de tiempos. El transvase de derecha a izquierda aumentó desde el 7,9 al 10,3% a los 30 minutos después de terminada la desviación. La conclusión es que, si no existe un incremento de la presión auricular izquierda, pueden tolerarse incrementos notables de la POC sin que tenga lugar edema pulmonar.