

Early recognition of pulmonary dysfunction during intramedullary orthopaedic surgery

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Summary

Peri-operative partial arterial oxygen tension (P_{aO_2}) was monitored in 20 patients undergoing total hip replacement. Three distinct groups emerged on analysis of the arterial oxygen tensions.

In group I there was no initial decrease in P_{aO_2} but it rose above baseline level towards the end of the procedure. In group II there was a distinct fall in P_{aO_2} during the acetabular and femoral stages of the operation. On termination of the procedure P_{aO_2} had returned to baseline levels. In group III P_{aO_2} values were significantly lower than those in group II during the acetabular and femoral stages and decreased further at the end of the procedure. One patient in group II and 6 in group III developed postoperative pulmonary dysfunction.

A progressive deterioration in P_{aO_2} during hip replacement without recovery at end of the procedure indicates that the patient is likely to develop postoperative pulmonary dysfunction.

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A report by Sproule *et al.*¹ in 1964 was the first to elucidate some aspects of the fat embolism syndrome. In 1966 Ashbaugh and Petty² demonstrated the pathological changes which take place in the respiratory distress syndrome, which has since been shown to be the common denominator in conditions such as shock lung and micropulmonary embolism.

It is not universally agreed that the presence of fat in the pulmonary circulation is of clinical significance in the fat embolism syndrome. Many investigators believe that the syndrome is caused by platelet aggregation and fibrin deposition in the lungs, initiated by tissue thromboplastin release.³

Peltier⁴ reported a 5,5% mortality directly related to fat embolism in a large series of trauma patients.⁴ At autopsies carried out in the Birmingham Accident Hospital, pulmonary fat embolism was observed in 44% of patients who had died of severe skeletal trauma.⁵

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Patients undergoing intramedullary prosthetic hip surgery are prone to develop the fat embolism syndrome.⁶ We investigated such a group of patients to gain further information about this disorder.

Patients and methods

Patients

Twenty patients with no history of major systemic illness and undergoing total hip replacement for osteo-arthritis under general anaesthesia were studied. The mean age of the group ($\pm$ SD) was 69 ± 9 years (range 53-94 years).

Anaesthetic management

Patients were given premedication of lorazepam or a combination of pethidine hydrochloride and promethazine hydrochloride, the former being favoured for older patients. After they had been given oxygen for 4 minutes anaesthesia was induced with thiopentone 1-2 mg/kg, followed by alcuronium 0,25 mg/kg. They were ventilated with an inspired oxygen fraction of 0,4 in nitrous oxide. The trachea was sprayed with 4% lignocaine and a number 8 or 9 cuffed endotracheal tube was inserted. Appropriate doses of diazepam and/or fentanyl and a low concentration of enflurane ($< 1,0\%$) was used for maintenance of anaesthesia. Ventilation was with a Bird Mk II-Ventviva combination incorporating a warm-water humidifier. Expired carbon dioxide was monitored with a Godart infant capnograph and kept at normal levels. At the end of the operation curarization was reversed with atropine and prostigmine.

Postoperatively the patients were exposed to an inspired oxygen fraction of 0,4 for 24 hours, using a Ventimask.

Monitoring

A 20G radial artery catheter was inserted and arterial and mean arterial pressures were monitored continuously, using an AE 840 pressure transducer and Simonsen and Weel oscilloscope and digital display.

A central venous line was inserted via a basilic vein. Routine chest radiography to confirm the position of the catheter was not carried out. A swing of at least 1 cm H_2O with respiration was taken as reasonable evidence of central placement. Pulse, ECG and temperature were also monitored continuously.

Surgical procedure

Routine total hip replacement by the Charnley method, with partial excision of the joint capsule and trochanteric osteotomy, was performed. After preparation of the acetabulum, acrylic cement was inserted and the acetabular prosthesis fixed in position. Care was taken not to insert the acrylic cement until it was pliable, to minimize absorption of free acrylic monomers into the

circulation.⁷ The medullary cavity of the femur was reamed and vented before insertion of the cement. The femoral prosthesis was fixed in position in the medullary cavity. The trochanter of the femur was reattached with steel wires. The mean duration of the operation ($\pm$ SD) was 125 ± 34 minutes and mean blood loss was 900 ± 400 ml.

Measurement times

Blood samples were withdrawn at measurement times 1-7 (Fig. 1). The results for the sample taken at measurement time 6 were not used because of the effects of opiates on partial arterial carbon dioxide tension (P_{aCO_2}) and inadequate control of the inspired oxygen fraction. Note that the inspired oxygen fraction varied at the different times.

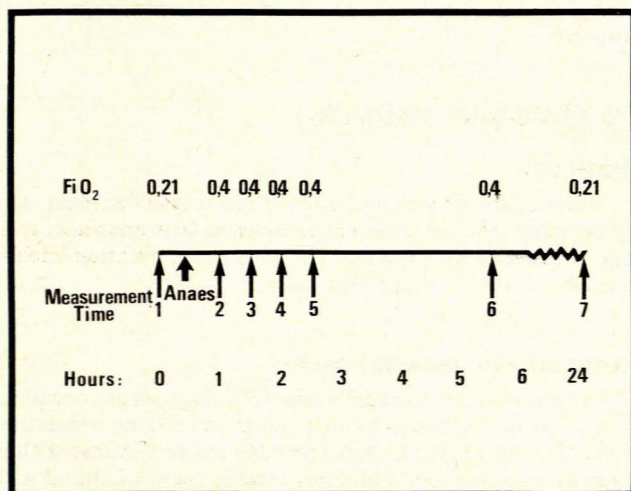


Fig. 1. Measurement times: 1 — pre-operative; 2 — ± 30 minutes stable anaesthesia; 3 — 5 minutes after fixation of the acetabular prosthesis; 4 — 5 minutes after fixation of the femoral prosthesis; 5 — end of operation; 6 — ± 3 hours after the operation; 7 — ± 24 hours after the operation.

Measurements performed

All estimations were on arterial blood samples. Arterial oxygen tension (P_{aO_2}), P_{aCO_2} and pH were determined with a Gas-Check AVL apparatus.

Results

P_{aO_2}

Because the patients were elderly and there was a wide range in alveolar-arterial oxygen tension differences,⁸ there was a large scatter of results for the individual patients. It was therefore decided to express changes in P_{aO_2} as a percentage from control values at measurement time 2, which reflected 'steady-state' anaesthesia. Utilizing the results of measuring times 2-5, i.e. all anaesthetic results, three distinct groups emerged (Fig. 2).

Group I. This group consisted of 6 patients. No fall in P_{aO_2} at measuring times 3 and 4 (when the acetabulum and femur respectively were operated on) and 5 (end of operation) was demonstrated. On the contrary, at points 3-5 the P_{aO_2} was higher than the baseline level at point 2. No patient developed pulmonary dysfunction⁹ or required treatment; these served as the control group.

Group II. This group consisted of 7 patients. At measuring times 3 and 4 (depicting acetabular and femoral procedures respectively) there was a distinct fall in P_{aO_2} , which was more severe at point 4. At both these points there was a highly significant

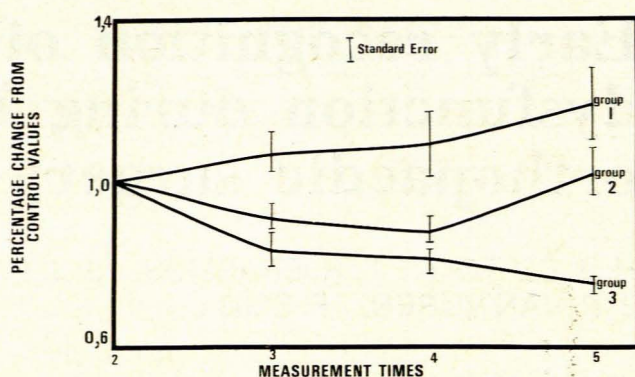


Fig. 2. Measurement times: 2 — ± 30 minutes stable anaesthesia; 3 — ± 5 minutes after fixation of the acetabular prosthesis; 4 — ± 5 minutes after fixation of the femoral prosthesis; 5 — end of the operation.

difference ($P < 0.01$) in comparison with group I. At point 5 the P_{aO_2} reached the baseline level. One patient developed pulmonary dysfunction.

Group III. This group consisted of 7 patients. At measuring times 3 and 4 the P_{aO_2} was even lower than in group II. The P_{aO_2} at position 5 was markedly lower, the difference from the group II value being highly significant ($P > 0.0025$). In this group 6 of the 7 patients developed pulmonary dysfunction.

In the 20 patients there was therefore a 35% incidence of pulmonary dysfunction.

P_{aCO_2} and pH

There was no significant difference in these parameters at any of the measuring times in the different groups, indicating a stable anaesthetic state and adequate pulmonary ventilation.

Other haemodynamic parameters

The arterial pressure remained constant except for occasional minor fluctuations (5-15 mmHg) after the insertion of the cement. This is in agreement with other findings.^{10,11} It appears that the use of methylmethacrylate only gives rise to minor haemodynamic changes which are of short duration.

In our patients there was a tendency for the central venous pressure (CVP) to fall while the bones were operated on, and more intravenous fluid than appeared necessary was required to keep the CVP stable. This is in agreement with the haemodynamic changes demonstrated by Modig and Malmberg.⁶

Discussion

Postoperatively the patients were given an inspired oxygen fraction of 0.4 for 24 hours. Blood pressure, pulse, temperature and colour were carefully monitored. Patients were examined twice daily.

Clinical features of the fat embolism syndrome (possibly synonymous with pulmonary dysfunction) usually present on the 2nd postoperative day. Gurd and Wilson¹² presented elaborate criteria for detecting the fat embolism syndrome. We preferred our simpler criteria to detect pulmonary dysfunction.

The usual pattern is that the patient appears well on a post-operative visit. On the next visit he or she complains of shortness of breath and 'feeling out of sorts'. Examination reveals the following: (i) tachypnoea (> 20 /min); (ii) tachycardia (> 110 /min); (iii) P_{aO_2} significantly lower than the pre-operative level ($P > 0.0125$) — in our patients those who did not develop pulmonary dysfunction showed no significant difference between

pre- and postoperative P_{aO_2} values; and (iv) P_{aCO_2} values at the lower limits of normal, but not significantly different from the pre-operative level. A raised temperature is not a feature, clinical examination of the lungs is negative, and chest radiography reveals no abnormalities.

These patients were then subjected to a 24-hour period of oxygen inhalation at an inspired oxygen fraction of 0.4, vigorous chest physiotherapy and frequent blood gas estimations. In 6 of the 7 patients who developed pulmonary dysfunction the P_{aO_2} had returned to normal after 24 hours. In 1 patient this result was achieved in 48 hours.

We know that the fat embolism syndrome progresses to 'shock lung', and owing to early diagnosis and prompt treatment none of our patients died and morbidity was very low.

Studies by Modig's group^{3,13,14} have shown that major trauma involving the femoral bone marrow results in immediate release of tissue thromboplastin products. These thromboplastin products generate platelet and fibrin deposits on the venous side of the systemic circulation and on the precapillary side of the pulmonary circulation. The micro-emboli are then trapped in the pulmonary microcirculation, which acts as a filter. It was also demonstrated that the magnitude of tissue thromboplastin release and pulmonary micro-embolism correlates well with the severity of pulmonary dysfunction and hypoxaemia.^{3,9,14}

In the presence of a normally functioning fibrinolytic system there is rapid resolution of these micro-emboli, and if there are pulmonary changes they will be transient.⁶ Our group I results conform to this category, since none of these patients developed hypoxaemia.

Modig and his co-workers^{6,13} found that 6 out of 7 patients in their general anaesthetic group developed hypoxaemia which was completely reversed within 30 minutes after impaction of the femoral prosthesis. These results correlate well with those in our group II patients.

A prolonged release of thromboplastic products, such as occurs in severe tissue damage, leads to a continuous deposition of micro-emboli in the lungs. If at the same time the fibrinolytic process is compromised, micro-emboli are retained in the pulmonary microvasculature.^{15,16} Our group III patients appear to fall into this category.

Modig and Malmberg⁶ found no correlation between the reduction in P_{aO_2} after insertion of the femoral prosthesis and the postoperative P_{aO_2} ; our 6 patients in group III who developed pulmonary dysfunction, however, showed a significant difference ($P > 0.0125$) between the pre-operative and postoperative P_{aO_2} (measuring times 1 and 7), as well as a positive correlation between P_{aO_2} at insertion of the femoral prosthesis and after the operation (points 4 and 7).

Modig and Malmberg⁶ have demonstrated a ventilation/perfusion defect in patients developing the fat embolism syndrome,

and state that at an early stage of pulmonary dysfunction determination of the P_{aO_2} is the most valuable investigation. The very significant difference between the mean P_{aO_2} ($P < 0.0025$) in group II and group III at measuring time 5 implies that this single measurement can be used as a criterion to determine which patients are at risk of developing postoperative pulmonary dysfunction. From the P_{aO_2} data at measurement time 5 (Table I) for the patients in group III who developed pulmonary dysfunction, the highest P_{aO_2} , seen in patient 5, was 81% of the mean control value. Although our series is small, we conclude that if the P_{aO_2} at measurement time 5 is 18% or more lower than the value during a stable anaesthetic state at measurement time 2, the patient is at high risk of developing pulmonary dysfunction.

Conclusion

From our studies, the baseline P_{aO_2} at measurement time 2 and the P_{aO_2} at the end of the operation (measurement time 5) appear to have prognostic implications. In relation to control values (point 2) a reduction in P_{aO_2} of 18% or more at point 5 indicates that the patient is at high risk of developing pulmonary dysfunction. These patients should be carefully monitored and treated at the earliest indication of lung dysfunction. When following such a regimen we experienced very low morbidity and no mortality.

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TABLE I. PERCENTAGE CHANGE FROM CONTROL VALUES AT MEASUREMENT TIME 5 IN GROUP III PATIENTS WHO DEVELOPED PULMONARY DYSFUNCTION

Patient No.	Change from control values (%)
3	69
5	81
13	79
14	67
15	73
16	79