Re-expansion pulmonary oedema: slow decompression?

Sir,—Re-expansion pulmonary oedema is a recognized and potentially lethal complication of the treatment of a pneumothorax by the rapid intercostal drainage of air. We would like to report a patient in whom recollapse of the lung following re-expansion pulmonary oedema may have prevented such an outcome.

A 23-yr-old man presented with chronic relapsing pancreatitis. A Whipple's pancreatico-duodenectomy was performed. The patient made a good recovery. Cannulation of the right subclavian vein by the infraclavicular approach was made and i.v. feeding was commenced. A chest radiograph demonstrated the tip of the catheter to be situated in the right superior vena cava and both lungs were expanded. However, on the 4th day of feeding, physical signs and a chest radiograph confirmed the presence of a right-sided pneumothorax.

Under local anaesthesia a Mallecot drain was inserted anteriorly to the right second intercostal space. When air was released it was observed to be under pressure. The catheter was connected to an underwater seal drain. Immediately the patient had a bout of non-productive coughing and became distressed, dyspnoeic and tachypnoeic, with central cyanosis. Examination revealed pale, clammy skin, a tachycardia of 130 beat min⁻¹ and an arterial pressure of 140/80 mm Hg. Inspiratory crepitations were heard over the whole of the right lung field. A provisional diagnosis of re-expansion pulmonary oedema was made.

The patient was given 100% oxygen, frusemide 40 mg and hydrocortisone hemisuccinate 100 mg i.v. Radiography confirmed that the right lung was almost fully expanded, but with the appearance of pulmonary oedema (fig 1.). The patient's arterial blood-gas tensions ($FiO_2$ = 1.0) after re-expansion showed pH 7.53, standard bicarbonate 28.4 mmol litre⁻¹, base excess +5, $Pa_{CO_2}$ 4.7 and $Pa_O_2$ 4.7 kPa.

During transfer of the patient to the Intensive Care Unit, air accidentally re-entered the right pleural cavity. Immediately, the patient's condition improved. Dyspnoea decreased, although air entry to the right lung decreased. The arterial blood-gas tensions were pH 7.54, standard bicarbonate 31.5 mmol litre⁻¹, base excess +8.5, $Pa_{CO_2}$ 4.74 and $Pa_O_2$ 10.2 kPa. Radiography showed re-collapse of the right lung. The patient's condition improved over the next 2 days as the right lung was allowed to expand slowly. Serial radiographs (over the next 5 days) showed that the pulmonary infiltration was resolving. Thereafter, postoperative recovery was uneventful and the patient was later discharged from hospital.

Re-expansion pulmonary oedema as a complication of the treatment of spontaneous pneumothorax has been described often [1–5]. Various factors appear to be implicated in the pathogenesis of the oedema. Lung collapse for three or more days [2], large pneumothoraces [3] alterations in surfactant concentration [1, 3, 4, 6] and the rapidity with which air is removed from the pleural cavity [2, 5], may be predetermining factors. Pulmonary capillary endothelial cells suffer anoxic damage during lung collapse [1]. When the pulmonary circulation is restored by expansion of the collapsed lung, the capillaries become excessively permeable to protein and fluid [7]. Management of re-expansion pulmonary oedema by slow re-expansion of the affected hemithorax does not always bring about improvement of the patient's condition [8]. However, the use of formal chest drainage and the sudden expansion of the lung in this patient was followed by pulmonary oedema. Since diuretics and increased $FiO_2$ did not improve the patient's condition, it may be reasonable to suggest that, when all else fails, re-collapse of the lung be attempted when re-expansion pulmonary oedema occurs. Later, the lung is allowed to re-expand slowly. Although the improvement in our patient's condition may have been coincidental with the re-collapse of the lung, we are unable to explain fully such a change.

A search of the literature has not revealed any similar cases. We would be pleased to hear from any of your readers who may be able to offer an explanation, or who may have had a similar experience.

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Continuous pump infusions for extradural analgesia during labour

Sir,—The use of pump infusions for continuous extradural analgesia during labour is gaining in popularity. Several excellent articles have outlined the basic pharmacological principles and clinical guidelines of the technique [1-3]. I would like to point out, however, that patient studies generally follow a rigid regimen, whereas clinical work should be more flexible. For example, Abboud and colleagues [2] used 0.125% bupivacaine 14 ml h⁻¹ alone, while Li, Rees and Rosen [3] used 10 ml h⁻¹ and 8-10 ml “top-ups” of 0.125% bupivacaine if analgesia was inadequate. Both induced excellent analgesia. However, Li, Rees and Rosen [3] had an exceptionally (and to me unacceptably) high incidence of muscle weakness, with almost 80% able to flex the ankle only or nothing at all. Such an incidence of muscle weakness is unavoidable if one uses a fixed regimen as in the above studies, but is almost entirely avoidable if a flexible schedule, suited individually to each patient, is used. Our clinical guidelines for infusion pump extradurals are as follows:

1. Establish a good block at the onset. The pump infusion will not make up for a poor extradural.
2. Patients vary. Start with rates of 10-14 ml h⁻¹ (if using 0.125% bupivacaine) and vary the rate to suit patient response. One patient (my wife) had excellent analgesia with minimal motor block with 30(!) ml h⁻¹.
3. If motor block is significant, stop the infusion until motor power returns, then restart the infusion.
4. If pain returns, add a reasonable top-up then continue the infusion.
5. Be flexible. Only journal articles have to follow a rigid regimen!

With these guidelines we can provide excellent analgesia with minimal motor weakness. As already stated in this forum [4], fixed regimens do not satisfy all clinicians. They also do not satisfy all patients.

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References

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Extradural test doses in labour

Sir.—The extradural administration of local anaesthetic agents is undertaken frequently in labouring mothers and various techniques, using test doses of local anaesthetic, are recommended to confirm that the catheter has not passed into the subarachnoid space before the full dose of local anaesthetic is given.

Test doses are usually assessed by detecting alterations in maternal arterial pressure, or the loss of motor power in the legs [1]. However, a confirmatory sign that is useful in labouring mothers is to wait for 5 min after the administration of the test dose and to observe the mother during the following contraction. If the pain from the contraction has not altered, it is unlikely that the drug has been deposited in the subarachnoid space. This sign should not be relied on alone as a useful basis for them to assess test doses—a subject that is controversial [1].

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References


Continuous flow ventilators in the ICU

Sir,—We were delighted to read the paper by Mecklenburgh and colleagues [1], which is of great clinical interest to...