Mediastinal Emphysema following Extradural Analgesia in a Ventilated Patient

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SUMMARY: A case of mediastinal emphysema developing in a patient under general anaesthesia supplemented with epidural analgesia is described. A possible cause is discussed and its prevention considered.

Introduction
Surgical emphysema and pneumomediastinum as complications of general anaesthesia have been reported, as have cases of subcutaneous supraclavicular emphysema following epidural analgesia in obstetrics. A case of mediastinal emphysema in a surgical patient under general anaesthesia supplemented with epidural analgesia is described.

Clinical details
A 58 year old woman was scheduled for sigmoid colectomy for diverticular disease. She had no past history of any respiratory disease. Physical examination and routine investigations including lung function tests were normal and there was no other evidence of chronic obstructive airway disease. She was premedicated with lorazepam 2mg orally six hours pre-operatively and anaesthesia was induced with midazolam 2mg followed by alcuronium 20mg intravenously. Intubation was easy and atraumatic. The lungs were ventilated manually with nitrous oxide in oxygen 33% with the maximum peak inspiratory pressure of 30cm H₂O. Catheterisation of the epidural space was attempted at the 2-3 lumbar vertebral interspace in the left lateral position using the loss of resistance to air to identify the epidural space. The ligamentum flavum was easily felt and entered with the Tuohy needle and there was no suggestion of injection of air into the neck from the mediastinum and subcutaneous emphysema (Fig 1). Spontaneous breathing restarted and the trachea was extubated. The patient was returned to the ward for further observation, breathing 40% oxygen by means of a mask.

Twelve hours later there was no evidence of subcutaneous emphysema and the previous X-ray changes had disappeared (Fig 2).

Discussion
Pneumomediastinum under general anaesthesia can occur following damage to the trachea, bronchi, alveoli or oesophagus. In this patient there was no evidence of damage to the respiratory tract. Tracheal intubation had been uncomplicated and atraumatic. There had been no history of respiratory disease. A nasogastric tube had not been passed and this made the possibility of oesophageal damage unlikely. If, however, the pneumomediastinum had been caused by damage to the trachea, bronchial mucosa or alveoli, then tension pneumomediastinum would have been likely with the continued positive pressure ventilation. Pneumothorax invariably accompanies alveolar damage associated with positive pressure ventilation. In this patient the pneumomediastinum was self limiting. Inadvertent transient hyperinflation in a lung with congenital bullae or air trapping behind secretions cannot be excluded. Nevertheless, in this patient a considerable volume of air had been injected into the epidural space.
Evans et al. demonstrated radiologically that there was communication between the epidural space and the retroperitoneal area via the intervertebral foramina. Air and fluid from the retroperitoneal space can spread to the mediastinum. In this case, some of the large volume of air injected into the epidural space may have found its way into the retroperitoneal space via the intervertebral foramina and then into the mediastinum. Positive pressure ventilation could have assisted this spread by the transmission of a positive pressure to the epidural space. This may be a rare complication of injecting large volumes of air into the epidural space. The danger of air emboli from the injection of air into the valveless venous plexus should also be borne in mind.

This case report shows a possible alternative cause for pneumomediastinum and it is suggested that if air is used for the identification of the epidural space, minimum volumes should be used.

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REFERENCES