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Severe unilateral bronchospasm mimicking inadvertent endobronchial intubation: a complication of the use of a topical lidocaine Laryngojet injector

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A healthy young woman is described in whom the left chest was unable to be inflated after intubation. The differential diagnosis and management are discussed. Severe unilateral bronchospasm was probably caused by topical lidocaine injected at the vocal cords and, inadvertently, into the left main bronchus with a Laryngojet device.

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A healthy 19-yr-old woman, weighing 64 kg, was scheduled for angiography and alcohol sclerosis of a facial arteriovenous malformation. She had no history of chest disease but smoked 10 cigarettes per day.

She received no premedication. Anaesthesia was induced slowly with midazolam 1.5 mg and propofol 110 mg, during which time she breathed oxygen. Anaesthesia was maintained with an infusion of propofol at 660 mg h⁻¹ initially, reducing to 500 and 400 mg h⁻¹ at 15 and 25 min respectively. Neuromuscular block was obtained with vecuronium. Before laryngoscopy, 40 mg of lignocaine was given i.v. A clear view of the larynx was obtained. The tip of a Laryngojet lidocaine (4%) injector was passed about 2 cm through the glottis, so that about half of the side-holes were above and half were below the cords. Five millilitres of solution were injected and the trachea was intubated with a

7.5 mm reinforced tracheal tube which was secured at 21 cm at the lips. On immediate inspection, both sides of the chest appeared to inflate equally and capnography revealed a normal waveform. The patient was transferred to the adjacent x-ray table and was ventilated with oxygen and air ($F_{I_{O_2}} = 0.35$) via a Bain system and a Nuffield 200 ventilator with a tidal volume of approximately 600 ml. Within 2 min of intubation the Sp_{O_2} had fallen from 100 to 90%. The inspired oxygen fraction was increased to 1 and the chest was re-examined. The right chest was expanding and breath sounds were vesicular with no wheeze. On the left, there was no expansion and there were no breath sounds or added sounds at all. Inadvertent right-sided endobronchial intubation was suspected. The tube position was rechecked. It remained at 21 cm at the lips. The tube tip was checked immediately by dynamic x-ray screening and was

seen 2 cm above the carina. A suction catheter was passed blindly down the tracheal tube. No secretions were aspirated and the left chest remained silent. The whole chest was then screened on X-ray. The right lung was seen to expand and accommodate the entire tidal volume. On the left there was no inflation of any lobe. The left hemidiaphragm was motionless and there was some basal collapse. Pneumothorax was excluded. Because the ventilatory abnormality was unclear, the procedure was discontinued, and a bronchoscopy was done to exclude an obstruction in the left main bronchus. This showed a normal bronchial tree on both sides to the level of the segmental bronchi, and no appreciable secretions.

The left chest remained static. The Sp_{O_2} 20 min after intubation remained at 90% with an FI_{O_2} of 1, and the basal collapse had increased.

Fifty micrograms of salbutamol was given intravenously. The PE'_{CO_2} rapidly increased from 5.2 to 6.3 kPa after 2 min. The alveolar plateau of the capnogram changed from virtually flat, to markedly up-sloping. The pulse rate increased from 85 to 110 min^{-1} . There was now very slight expansion of the left chest and a marked expiratory wheeze. A further 50 μg of salbutamol was given. Within 3 min, expansion of the left and right chest was equal. The PE'_{CO_2} decreased to 5.0 kPa within a further 3 min and the capnogram plateau became almost flat again. The inspired oxygen fraction was reduced to 0.35 and the Sp_{O_2} remained at 98%. Given this marked improvement, the angiography proceeded and sclerosis of the arteriovenous malformation was done, taking approximately 1 h. After this, repeat screening of the chest showed both lungs expanding equally and complete resolution of the previous left basal collapse. The patient was extubated uneventfully and returned to the ward, where she was monitored closely. She displayed no wheeze or gas exchange deficit.

Discussion

Failure to ventilate one side of the chest after intubation of the trachea occurs commonly. Routine examination of the chest by inspection and auscultation after intubation will identify this and allow treatment before complications result. The most likely cause is unilateral endobronchial intubation, which is easily remedied by withdrawal of the tip of the tube into the trachea.

In this case, it was unlikely that the tip of the tube was beyond the carina because its position at the lips seemed to be normal for the size of the patient. If attempts had been made to withdraw the tube until the left chest began to expand, this could have caused inadvertent extubation at a time when this could be least afforded. We had a unique radiographic opportunity to examine the causes and consequences of this incident without altering the tube position. Screening the tube tip position confirmed the initial clinical belief that it was in the trachea. Screening the whole chest

showed no air entry to the left lung. If this were caused by large airway obstruction, say a large mucous plug, then it would have to be very proximal indeed. Blind passage of a suction catheter was predictably unhelpful and bronchoscopy excluded any airway obstruction as far down as the segmental bronchi.

The patient's oxyhaemoglobin saturation remained at 90% with an FI_{O_2} of 1. This is consistent with venous admixture of the order of 40–50%.¹ The development of the basal atelectasis is of interest. Atelectasis in dependent dorsal regions is well known to occur during anaesthesia.^{2,3} It can occur very rapidly (within minutes) when patients have been preoxygenated⁴ or when lungs containing regions with a very low ventilation/perfusion ratio (V/Q) are ventilated with a high FI_{O_2} . Atelectasis is not easily visible on conventional X-ray imaging, but is detected with high sensitivity by CT scanning.⁵ This patient was only partially pre-oxygenated. However, when the oxyhaemoglobin saturation fell after intubation, the FI_{O_2} was increased to 1. The whole of the left lung probably had a low V/Q, and the dependent dorsal regions more so. Even with screening that was relatively insensitive, atelectasis was noted and evolved over 20 min. In this case, the ventilatory abnormality was gross and unlikely to be missed. However, with a less obvious abnormality, if an increase in FI_{O_2} restored a nearly normal Sp_{O_2} , a prolonged anaesthetic might be permitted to proceed. Indeed, some anaesthetists use an FI_{O_2} of 1 routinely. In such cases, the progressive development of atelectasis might impair postoperative lung function and gas exchange.⁶

There are three cases of 'unilateral bronchospasm' listed in Medline (1966–2000). Two occurred after surgical procedures of the pleura (interpleural analgesia and pleurodesis)^{7,8} and one after subclavian vein puncture. Shantha⁷ reviews the likely mechanisms for unilateral bronchospasm. Bronchomotor tone depends on the balance of β -adrenergic and vagal parasympathetic outflow. Unilateral bronchospasm could result from (i) disproportionate sympathetic blockade, or (ii) stimulation of superficial airway receptors supplied by vagal fibres by any irritant.⁷ A direct sympathetic nerve supply to the bronchial smooth muscle has been questioned⁹ and it is unlikely that sympathetic blockade occurred in this case. The lignocaine given i.v. would be unlikely to cause significantly large plasma concentrations, nor would it act unilaterally, and the topical lignocaine is unlikely to have penetrated such a deep and distant structure as the hilar plexus. The topical lignocaine may have stimulated airway receptors to produce vagally mediated bronchoconstriction. Atelectasis by a local neural bronchoconstrictor reflex can occur after a variety of lung insults, including pulmonary embolism and trauma.¹⁰ Constriction is not limited to the conducting airways, but is thought to be effected within the alveolar acinus by specific contractile interstitial cells, which are

thought also to control local pulmonary capillary blood flow and hence exert an effect on V/Q matching.¹¹

Downes and colleagues^{12 13} showed that, in dogs, both aerosolized and i.v. lignocaine prevented the increase in airway resistance produced by an irritant citric acid aerosol, suggesting that lignocaine by either route blocked the reflex irritant response. They also showed that aerosolized lignocaine did not prevent allergic bronchospasm produced by inhalation of *Ascaris* antigen in previously sensitized animals. By contrast, Fish and Peterman¹⁴ showed that, in eight asthmatic subjects, rather than blocking reflex bronchoconstriction, aerosolized lignocaine (4%) actually caused it. They conclude that whilst topical lignocaine is theoretically capable of blocking neurogenic reflexes, it may produce reflex bronchoconstriction in patients with asthma or hyperirritable airways.

This patient was not asthmatic and had no history of recent upper respiratory tract infection. She was, however, a smoker and could have had hyperirritable airways. In addition, the drug was administered not as aerosolized mist but as a jet of liquid, which itself is likely to be more irritant. This case demonstrates a problem of a topical lignocaine delivery device which is capable of depositing a relatively large dose of the drug below the cords, and sending a jet of drug down either main bronchus or even both bronchi. It also demonstrates a risk of high inspired fractions of oxygen (in this case obligatorily) when the lung has significant areas with very low V/Q ratios. The fortuitous use of instant X-ray screening allowed the tube position to be exonerated, and the value of fibre-optic bronchoscopy in the differential diagnosis is highlighted.

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Anaesthesia for Kartagener's syndrome

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Kartagener's syndrome is a hereditary syndrome involving a combination of dextrocardia (situs inversus), bronchiectasis and sinusitis, transmitted as an autosomal recessive trait. We describe a patient who had three anaesthetics over a period of a few months. Discussion relates to anaesthetic considerations in the syndrome and to recent findings relating to the molecular mechanisms of left-right development.