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

Downes and colleagues\textsuperscript{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 bronchoospasm produced by inhalation of \textit{Ascaris} antigen in previously sensitized animals. By contrast, Fish and Peterman\textsuperscript{14} 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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\textbf{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.
A 27-yr-old female presented for surgery three times. She had Kartagener’s syndrome.1 The first operation was a left middle lobectomy for bronchiectasis. Some months later she complained of abdominal pain and her second operation revealed an ovarian adenocarcinoma. This operation was complicated by an episode of postoperative pneumonia. Two weeks later she had a total abdominal hysterectomy with bilateral salpingo-oophorectomy.

Case report

The first operation—left middle lobectomy

The patient’s main complaint was one of recurrent episodes of pneumonia. She also admitted to chronic sinusitis and volunteered the information that ‘her heart was on the wrong side but normal’. Examination revealed no clubbing; her apex beat was on the right side; auscultation of the heart sounds was normal and there were some crepitations and rhonchi mainly in the left axilla.

Chest x-ray revealed dextrocardia and a right-sided stomach bubble. ECG demonstrated sinus rhythm and inversion of the P wave in lead 1 with a deep Q wave and inversion of the T wave. CT scan showed bronchiectasis that was most prominent in the left middle lobe. Lung function tests were normal and it was not considered necessary to do a blood gas study. She was admitted to the clinic for physiotherapy and scheduled for left middle lobectomy.

Anaesthesia for thoracotomy and lobectomy was unremarkable apart from it seemed logical to use a right-sided double lumen tube as she had three lobes on the left and two on the right with the longer main bronchus on the right. General anaesthesia was induced with propofol and maintained with isoflurane and sufentanil. For neuromuscular block we used vecuronium. Routine monitoring was enhanced by inserting a radial arterial line. The trachea was intubated with a right-sided 37 FG Mallinkrodt double-lumen tube and the lungs ventilated with a Servo Elema 900C ventilator.

After surgery, the trachea was extubated and the patient returned to the ward. Her postoperative course was uneventful and she was sent home after a few days.

The second operation—diagnostic laparoscopy

A few months later after the lobectomy the patient presented to a gynaecologist with abdominal pain. We were not involved with her management at this time. She had a general anaesthetic for laparoscopy that revealed adenocarcinoma of the ovary. Her postoperative course was complicated by an episode of left lower lobe pneumonia.

The third operation—total abdominal hysterectomy and bilateral salpingo-oophorectomy

Two weeks after the laparoscopic examination, the patient presented for total abdominal hysterectomy and bilateral salpingo-oophorectomy. In view of the fact that her last procedure had been complicated by pneumonia, spinal anaesthesia was chosen. She was sedated with midazolam 5 mg i.v. Spinal anaesthesia was given through a 25-gauge pencil point needle using 12.5 µg of hyperbaric bupivacaine. A continuous propofol infusion was used for sedation. Surgery was performed through a Pfannenstiel incision. The operation and postoperative period was uneventful.

Discussion

Kartagener’s syndrome is a variant of the immotile cilia syndrome.2 This has also been called primary ciliary dyskinesia and is a result of an autosomal recessive disorder of the microtubules of ciliated cells. Symptoms include male sterility, chronic or recurrent respiratory tract infection, and bronchiectasis because of the absence of mucociliary clearance. In 50% of patients situs inversus occurs and hence Kartagener’s syndrome.

It may become possible to link the occurrence of abnormal ciliary function and abnormal position of the body organs. It seems that genes determine the structure, function, and time of appearance of proteins in the embryo that influence normal development and siting of the internal organs asymmetrically (situs solitus). On the left side of the embryo, near Hensen’s node, a variety of proteins are secreted (Sonic Hedgehog, Nodal, Lefty and Pitx2). On the right side are others (Activin betaB, Snail and Fibroblast Growth factor-8).3 These normally influence rotations, that lead to a left-sided heart and the usual position and structure of the lungs and abdominal organs. Abnormal genes and so abnormal proteins would lead to malpositions. Normal ciliary motion in the mouse node is anteclockwise and conceivably influences the flow of the proteins to their correct sites in the embryo. In the immotile cilia syndrome it is possible that abnormal distribution of the proteins occurs. Is this the reason that the dextrocardia of Kartagener’s syndrome links with the immotile cilia syndrome, and also situs inversus?
Acute right-to-left inter-atrial shunt; an important cause of profound hypoxia

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Three patients presented to our intensive care unit over a 3-yr period with profound hypoxia resulting from acute right-to-left inter-atrial shunt (RLIAS). Patient 1 was a 67-yr-old male with an atrial septal defect who became hypoxic and developed the rare sign of platypnoea following elective repair of an abdominal aortic aneurysm (breathlessness made worse when upright and relieved by lying flat). Patient 2 was a 38-yr-old female who developed platypnoea and complex cardiac abnormalities. They are most commonly transposition of the great arteries, double outlet right ventricle, ventricular septal defect, single ventricle and pulmonary stenosis or artresia. In patients with dextrocardia and situs ambiguous, polyspleenia or asplenia may be present in association with complex multiple cardiac abnormalities. These include a combination of systemic and pulmonary venous abnormalities, defects in the ventricular and atrial septa and endocardial cushion defects.

There may be pulmonary artery obstruction and mal-development of the great arteries.

The incidence of isolated levocardia (a left-sided heart) with situs inversus is about 0.6% per 10 000 births and more than 90% have serious heart disease.

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