Survivors of childhood cancers: implications for obstetric anaesthesia

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Modern, aggressive treatment of paediatric malignancies has enabled survival into adulthood. There is a small but increasing cohort of women of reproductive age who were treated with chemotherapy, radiotherapy and surgery in childhood. Although infertility and pregnancy loss are more common in such women, a significant number of them have successful pregnancies.1 The consequences of chemotherapy, radiation and surgery will differ according to the site of the malignancy treated, but presentations in adulthood have common features. We present two women who survived childhood cancer and illustrate some of the difficulties that were encountered. The infants of both women were delivered by elective Caesarean section and both women requested regional anaesthesia.

Case 1

A 25-yr-old primipara presented to the anaesthesia clinic before elective Caesarean section at 34 weeks of gestation. At the age of 10 months, a mass around the right adrenal had been diagnosed as stage IV neuroblastoma. She had been treated with nephrectomy, adrenalectomy, radiotherapy and chemotherapy (vincristine, cyclophosphamide and doxorubicin). She was short in stature (145 cm, 4 ft 10 inches), and had a deformity of her right hip and a significant thoracolumbar scoliosis, which was not severe enough to limit respiratory function; pulmonary function tests had not been performed. She had suffered anthracycline cardiotoxicity, a dose-dependent complication of therapy with doxorubicin and daunorubicin. There was myocyte damage resulting from the production of free radicals, and cardiac growth was impaired.2 Echocardiography 3 days before the operation demonstrated an ejection fraction of 45% and fractional shortening of 22%. Increasing breathlessness during pregnancy was ascribed to worsening cardiac function, for which she was taking lisinopril 7.5 mg daily. She was hypertensive (130/100 mm Hg), and methyldopa 250 mg twice daily had been added during pregnancy as the hypertension worsened. Renal function was normal. Because of her problems, her obstetrician had already chosen Caesarean delivery, and she requested regional anaesthesia. Two doses of dexamethasone 4 mg were given before delivery in an attempt to improve fetal lung maturity.

From the history and examination in the clinic, it was felt that reduced cardiac reserve posed the greatest anaesthetic problem, and that technical difficulty with regional block arising from scoliosis was a secondary concern. No respiratory problems were anticipated, and further investigations were not thought to be necessary. The plan agreed upon for anaesthesia was to use an incremental subarachnoid technique, with invasive arterial monitoring but without a pulmonary artery flotation catheter.
Before operation, a 20-gauge cannula was inserted into the left radial artery, and a 16-gauge i.v. cannula was inserted into a forearm vein. An infusion of Gelofusine (B. Braun Medical Ltd, Aylesbury, UK) 500 ml was commenced. Further monitoring included ECG and pulse oximetry. In the sitting position, a 26-gauge Quincke spinal needle was inserted with difficulty into the L3/4 interspace. A 32-gauge catheter (TFX Medical, Lurgan, N. Ireland) was then inserted and the needle removed. Two centimetres of catheter was placed in the subarachnoid space. Initially, 0.5% w/v hyperbaric bupivacaine 1 ml and diamorphine 0.4 mg were injected whilst the patient was sitting. A further 500 ml of Gelofusine containing ephedrine 30 mg was commenced. The patient was then moved into a tilted left supine position. After 10 min, plain bupivacaine 2.5 mg was injected. Sensation was blocked up to T6 on the right and T4 on the left. Caesarean section was performed without incident. Transient hypotension (systolic pressure of 60 mm Hg) occurred on administration of a test dose of Syntocinon 1 IU, and no further oxytocin was given. A live female baby weighing 2 kg was delivered. Apgar scores at 1 and 5 min were 8 and 9 respectively, and the umbilical arterial pH was 7.36.

For postoperative analgesia, the patient received diclofenac 100 mg per rectum, and thereafter up to 150 mg orally per day, and paracetamol 1000 mg/codeine 60 mg as required to a maximum of six doses per day. The intrathecal catheter was removed in the recovery ward 2 h after operation. We continued invasive monitoring for 24 h after delivery and the patient was stable throughout this period. Blood loss at delivery was estimated at 500 ml, but the haemoglobin had decreased to 8.1 g dl\(^{-1}\) on the first postoperative day and 3 units of blood was transfused. The patient was seen by an anaesthetist for three consecutive days to identify complications of regional block. No such complications were apparent. The mother and baby were discharged home after 5 days. Antihypertensive medication was continued on discharge.

Case 2

A 24-yr-old primipara presented at 33 weeks for urgent Caesarean section with only a few hours’ notice. The membranes had ruptured prematurely and markers of infection, especially raised C-reactive protein (11 mg litre\(^{-1}\)), were causing increasing concern about chorioamnionitis. The patient was apyrexial and had no other clinical signs of systemic infection. The patient had been treated successfully with radiotherapy and chemotherapy for rhabdomyosarcoma of the pharynx as a child. She had been informed that the treatment would result in infertility, so did not use contraception and the pregnancy was unplanned. She requested regional anaesthesia.

On examination she was of short stature (147 cm, 4 ft 11 inches) and had mandibular hypoplasia, which was considered likely to cause difficulty with intubation (Mallampati grade 2). There was little opportunity for further investigation. Cardiomyopathy secondary to an unknown anthracycline had been diagnosed previously and mid-trimester echocardiography demonstrated an ejection fraction of 60% and fractional shortening of 23%. Her medical history included fractured lumbar spine, for which no details were available. She was taking thyroxine because treatment had resulted in thyroid hypoplasia.

An incremental subarachnoid technique was chosen. In case regional anaesthesia was not successful, facilities for awake fibre-optic intubation were available. Before the block, monitoring with ECG, non-invasive arterial blood pressure and pulse oximeter were established. An i.v. cannula was placed in a forearm vein and Gelofusine 500 ml was given. In the absence of signs and symptoms of heart failure, invasive monitoring was considered unnecessary. Aseptic techniques were used, including gown, hat and mask. The skin was prepared with alcohol solution. We used a needle-through-needle technique with a Spinocath kit (B. Braun Medical, Melsungen, Germany). An 18-gauge Tuohy needle was placed in the epidural space and a 24-gauge catheter was threaded over a 29-gauge Quincke spinal needle passed intrathecally. Both needles were then withdrawn. An i.v. infusion of ephedrine 30 mg in Gelofusine 500 ml was begun with the first intrathecal injection. Three increments of 0.5% w/v hyperbaric bupivacaine were given (10 mg in total), together with 0.5% w/v plain bupivacaine 4 mg, and diamorphine 0.4 mg, without achieving a satisfactory block. At this point it was decided that the tip of the catheter might be positioned in a caudad direction and the catheter was withdrawn 2 cm. Hyperbaric bupivacaine 0.5% w/v 2.5 mg and 0.5% w/v plain bupivacaine 5 mg were then given, producing excellent anaesthesia with abolition of sensation bilaterally to T5. There was no haemodynamic disturbance, and the patient was stable throughout. A female baby was delivered, and the umbilical arterial pH was 7.28. Syntocinon 10 units was given slowly via the i.v. infusion on delivery, without detectable hypotension. Prophylactic cefuroxime 750 mg and metronidazole 500 mg were given i.v. in the operating theatre and continued orally for 5 days. Postoperative analgesia consisted of diclofenac and paracetamol/codeine. The estimated blood loss at delivery was 700 ml. The intrathecal catheter was removed in the recovery ward. The next day, haemoglobin was recorded as 9.8 g dl\(^{-1}\) and 3 units of blood was transfused. An anaesthetist visited the patient for 3 days to identify complications of regional blockade. The mother and baby were discharged home after 5 days.

Discussion

Despite different original pathologies, these two survivors of paediatric malignancy have common features.
Anthracycline cardiotoxicity

Anthracyclines are a family of cytotoxic antibiotics. Anthracycline cardiomyopathy is a dose-dependent complication of therapy with doxorubicin and daunorubicin, which damages myocytes and impairs cardiac growth. If given in childhood, normal cardiac growth is impaired, leading to permanent cardiac dysfunction. The toxicity has been attributed to the production of free radicals. The incidence of cardiomyopathy after chemotherapy is unknown, and whether a safe dose exists has not been clarified. Changes are irreversible and are characterized by a poorly compliant myocardium that is likely to respond poorly to changes in preload and afterload. Diagnosis is made clinically, using echocardiography or cardiac catheterization, but may be confirmed histologically, showing hypertrophy and fibrosis of myocytes. Both our patients had impairment of left ventricular function, but one woman was symptomatic with breathlessness, indicating a degree of cardiac failure. Her limited cardiac reserve presumably accounted for the hypotension after administration of 1 unit of Syntocinon. We did not think it advisable to give a further dose. Our second patient, despite a fractional shortening of 23%, was asymptomatic and tolerated Syntocinon well. Clinical assessment correctly identified which woman posed the greater risk. The long-term prognosis of this condition may be poor, and transplantation has been required.

Scoliosis and short stature

Scoliosis can be a cause of short stature. In our first case, the scoliosis was presumed to be secondary to radiation-induced hip deformity. The spine may also be affected during the irradiation of midline tumours, and deformity, white matter injury, osteoporosis and epidural space scarring has been documented. Norris recommended one dose of single-shot intrathecal local anaesthetic for regional block, assuming normal anatomy, but of the 50 women included in that study only one was less than 150 cm tall. Scoliosis and short stature can cause uncertainty about the dose of intrathecal local anaesthetic required. Our solution to potential problems within the epidural space is to use an intrathecal approach, and because of uncertainties over dose we titrated the local anaesthetic through an indwelling catheter. This had the added advantage of a slow onset of block, avoiding cardiovascular upset. An additional factor in our choice was that we have significant experience with Caesarean delivery in scoliotics using continuous intrathecal anaesthesia.

Oxytocics

Oxytocics are used to provide uterine contraction and haemostasis after delivery. Two drugs are commonly used for this purpose: ergometrine and Syntocinon, singly or in combination. Ergometrine, an ergotamine alkaloid, is a powerful oxytocic but is known to raise systemic vascular resistance significantly and increase afterload. Syntocinon, a synthetic form of the naturally occurring oxytocin, has greater cardiac stability, but is known to cause hypotension with bolus administration. The mechanism for this is debatable. Possible causes are a reduction in venous pressure, a decrease in vascular resistance, and negative inotropy, or a combination of these. Death at Caesarean section has occurred after bolus administration of Syntocinon in a woman with congenital heart disease. Our experience demonstrates that even minute doses of Syntocinon can cause worrying hypotension in the presence of poor myocardial function. It has been suggested that slow infusion of Syntocinon may reduce the incidence of hypotension. Cardiac output monitoring is justified when effects are predicted to be severe.

Survivors of childhood cancers will present with increasing frequency for obstetric anaesthesia both to regional centres and to district general hospitals. It is likely that these women will have abnormalities affecting anaesthesia and a management plan should be made in the antenatal period. Adult survivors with lifelong cardiac impairment secondary to anthracyclines are an emerging problem. The first exposure of young adult cancer survivors to anaesthesia may occur with their first pregnancy, and the obstetric anaesthetist may be the first to test cardiac performance and to deal with the sequelae of their treatments.

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Prolonged vecuronium neuromuscular blockade associated with Charcot Marie Tooth neuropathy

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Charcot Marie Tooth (CMT) disease comprises a group of disorders characterized by progressive distal muscle weakness and wasting. Review of the anaesthetic literature produced conflicting reports concerning the responses to neuromuscular blocking drugs in these patients. We describe a case in which vecuronium 0.11 mg kg⁻¹ produced prolonged neuromuscular blockade lasting 115 min in a patient with the condition. Conduction velocity in the facial nerve is usually less affected than the ulnar or peroneal nerve in CMT patients. This nerve may be more useful in monitoring neuromuscular blockade, both in titrating the dose of neuromuscular blocking drug and ensuring adequate reversal at the end of a procedure. Recent advances in molecular biology have enabled identification of the underlying genetic abnormalities and pathophysiology of CMT. These advances are reviewed and implications of CMT for the anaesthetist discussed.

Keywords: neuromuscular block, vecuronium; complications, Charcot Marie Tooth disease

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Case report

A 59-yr-old, 74 kg male with CMT neuropathy presented with a fractured right proximal fibula and distal tibia, requiring open reduction and internal fixation with a tibial nail. The patient had been diagnosed as having CMT in May 1987 in a medical report for retirement on the grounds of ill health. The diagnosis was made on clinical grounds, with examination revealing high foot arches and distal muscle wasting involving the forearms, interossei in the hands, and all muscles below the knee. His arm tendon reflexes were brisk and symmetrical but knee and ankle jerks were absent with flexor plantar responses. Sensation was normal and there was no ataxia. He described himself as always having been weak. He had no siblings but described his father as having been of the same build. Electromyography had not been performed.

The patient underwent L5-S1 discectomy in 1983, receiving thiopental 225 mg, tubocurarine 40 mg and halothane. The operation lasted 75 min. There was no comment in the anaesthetic record regarding reversal of residual neuromuscular blockade, nor were any problems recorded. In 1996, he underwent insertion of a dynamic hip screw of his left femur and received midazolam 2 mg, propofol 100 mg, and atracurium 30 mg at
induction followed by atracurium 10 mg after 30 min. This operation lasted 50 min. Acetylcholinesterase inhibitors were not given. On neither occasion was any neuromuscular monitoring used. No adverse events or clinical evidence of prolonged neuromuscular block were noted.

After his hip fracture, a diagnosis of osteoporosis was made using bone density studies. He received alendronate 10 mg day⁻¹ and calcium supplementation. His corrected serum calcium was 2.59 mmol litre⁻¹ with an inorganic phosphate of 1.00 mmol litre⁻¹ prior to this operation. Serum potassium was 4.6 mmol litre⁻¹.

On this occasion the patient was unpremedicated. He received morphine 10 mg and thiopental 350 mg at induction. Vecuronium 0.11 mg kg⁻¹ was administered and after 2 min the trachea was intubated easily with a size 9 mm cuffed oral tracheal tube. Anaesthesia was maintained with isoflurane 1% in nitrous oxide/oxygen 2:1. No further opiates were given. A peripheral nerve stimulator was not used at this stage because no problems had been identified from the previous anaesthetic records.

The operation lasted approximately 115 min, during which time no further drugs were given. At the end of the procedure the lungs were ventilated with FIO₂ 1.0 and the patient received neostigmine 2.5 mg with glycopyrrolate 500 μg. A peripheral nerve stimulator was not used at this stage. It was felt that sufficient time had passed since induction for recovery from neuromuscular blockade to occur. Spontaneous diaphragmatic breathing with an adequate tidal volume followed and the tracheal tube was removed uneventfully although the patient was not fully conscious. He was then transferred to the recovery room with oxygen administered by facemask. In recovery, the patient became anxious and distressed, with signs of incomplete reversal of neuromuscular block including twitching of the limbs and uncoordinated movements. Oxygenation and ventilation remained normal.

A peripheral nerve stimulator was applied first to the right ulnar nerve. Trains of four stimuli were applied. There was a palpable twitch response to the first stimulus only. As clinically obvious muscle wasting was present in the forearm muscles the nerve stimulator was applied to the right facial nerve. Again, a twitch response was only visible to the first stimulus of the train of four. Double burst stimulation was not used as partial reversal was clinically obvious and it was felt this would subject the patient to further distress.

A second dose of neostigmine 2.5 mg was given, resulting in all four twitches of the train of four response in the facial muscles being visible within 2 min. The patient’s condition improved and he was transferred to the high dependency unit for overnight observation and returned to the ward on the next day.

Discussion
Charcot Marie Tooth neuropathy comprises a heterogeneous group of peripheral nerve diseases affecting adults and children.⁷ There is a typical clinical picture with distal muscle weakness and atrophy affecting the intrinsic muscles of the foot, the peronei and tibialis anterior. Both motor and sensory function may be affected. Deep tendon reflexes are diminished or absent. There is a spectrum of clinical presentation from severe atrophy with limb abnormality to pes cavus. A significant proportion of CMT patients are not identified because their symptoms do not cause them to consult a doctor. Most patients with the condition have manifestations by their second decade, seldom presenting after 30 yr. Upper limb involvement tends to occur later.

Clinical studies in CMT families using nerve biopsies and nerve conduction studies enabled a separation of these patients into two main groups.⁸ In CMT1 there is a marked reduction in nerve conduction velocity with demyelination; nerve conduction velocity in the ulnar, median and peroneal nerves is commonly half that in normal subjects.⁷ The muscle action potential amplitude is half normal, and the nerve latency three times as long on average; 25% of patients have clinically thickened peripheral nerves. Those adolescents with the slowest conduction velocities tended to have a worse neurological deficit in later life.

In CMT2 the nerve conduction velocity is normal or low normal.⁹ There is axonal loss but no prominent demyelination. In this type the clinical symptoms may present later, even in middle age. Although the features are similar to CMT1, there is said to be less upper limb involvement and the peripheral nerves are less enlarged.

The two types cannot be distinguished solely on clinical grounds because there is such a range of severity of symptoms. Nerve conduction studies are often performed in the median or ulnar nerves because distal nerve degeneration is often complete in the lower limbs. In fact, motor nerve conduction velocity is commonly less than 60% normal in all nerves studied, including the facial nerve.

Advances in molecular biology and gene mapping have enabled the underlying genetic abnormalities in CMT to be identified. In CMT1a and CMT2 the inheritance is autosomal dominant.¹⁰ There are also much rarer X-linked and autosomal recessive variants. CMT1 can be subdivided on the basis of the gene defect. In CMT1a there is a duplication at the 17p11, 2–12 locus. This area codes for the PMP22 gene resulting in multiple expression. PMP22 is a major component of peripheral nerve myelin. The result may be a new Schwann cell phenotype that has defective myelin stability and increased turnover.¹⁰ In CMT1b there are point mutations in the myelin protein P0 gene.⁶ This codes for the major myelin protein in peripheral nerves, accounting for 50% of the mass. It has been suggested that the neurological disability in CMT1b is worse than in CMT1a. In X-linked CMT (CMTX), which is clinically similar to CMT1, there are mutations in the
connexin 32 gene. This encodes a gap junction channel protein that may play a role in transmission at nodes of Ranvier. Dejerine Sottas disease, or CMT3, is a severe variant with onset in infancy. There may be point mutations in the P0 or PMP22 genes. The clinical features overlap with severe CMT1. CMT2 accounts for 25% of cases. There does not appear to be a unifying genetic defect.

As 1 in 2500 people has some form of CMT, it is surprising that case reports concerning anaesthesia in CMT sufferers are rare. In one report, the notes of seven patients under 16 yr who were anaesthetised over a 10 yr period were reviewed.11 These patients received both depolarizing and nondepolarizing neuromuscular blocking drugs. There were no recorded adverse effects of succinylcholine and no cases of prolonged response to a range of non-depolarising neuromuscular blocking drugs. However, the authors conceded that the methods used to assess neuromuscular function varied widely.

Antognini reviewed the operative charts of 86 patients with CMT identified by postal questionnaire to members of a CMT help group.12 In 161 surgical procedures on these patients anaesthetic complications were few, although the study is necessarily biased because non-responders are excluded. In addition, anaesthetic management and inclusion criteria for CMT could not be standardized. 48% of patients received succinylcholine with no recorded adverse effect. A paralysing dose of non-depolarizing neuromuscular blocking drug was used in 45% of patients. How neuromuscular block was monitored and reversed is not known. There were no descriptions of objective weakness or prolonged intubation. Despite the absence of adverse effects after succinylcholine, it would seem prudent to avoid this drug in CMT if possible as hyperkalaemia is well reported in other similar polyneuropathies and denervation injuries. There is no evidence to suggest that patients with CMT are susceptible to malignant hyperthermia.

Another report studied 20 patients with CMT presenting for orthopaedic procedures.2 The authors found that CMT sufferers required less thiopental for induction than controls. The report is of interest because one female patient received vecuronium 0.07 mg kg$^{-1}$ at induction and subsequently did not exhibit a normal twitch response or spontaneous ventilation for 280 min. In addition, the ulnar nerve twitch response was absent in six of the 20 patients. In these six patients, the facial nerve was used for evaluation of neuromuscular block.

A review of the literature concerning anaesthesia for CMT revealed few detailed case reports. One describes prolonged artificial ventilation due to respiratory muscle involvement in pregnancy.13 Another describes a 17-yr-old who underwent anaesthesia on two occasions and showed no prolonged response to either mivacurium or atracurium.3 Baraka described a 16-yr-old girl who underwent a tendon transfer under general anaesthesia using vecuronium. In this case, the T4/T1 ratio recovered to 25% after 20 min and further supplementation was required. Residual neuromuscular blockade was readily reversed after 90 min. The author suggested that upregulation of acetylcholine receptors at the neuromuscular junction due to the generalized polyneuropathy might account for the resistance to vecuronium even in the presence of muscle weakness and atrophy.5

One case report demonstrated a potential difficulty in monitoring neuromuscular block in CMT patients. In a 17-yr-old neurosurgical patient, it was not possible to elicit responses from either tibial nerve following vecuronium 0.1 mg kg$^{-1}$ after 100 min. The muscle response was either too delayed or too small in amplitude to be recorded.5 This is perhaps unsurprising as the tibial nerves are among the worst affected by demyelination in CMT. The use of peripheral nerve stimulators in CMT has been shown to be misleading if a nerve with delayed conduction or increased latency is chosen. One solution would be to monitor relaxation after induction using a less affected peripheral nerve such as the facial nerve. A suitable site for neuromuscular monitoring should be identified before a neuromuscular blocking drug is administered. The drug can then be titrated to effect and adequate reversal identified.

Case reports have been described in the literature of patients with CMT undergoing central neural blockade without neurological sequelae.14 15 Posterior column demyelination has been noted in autopsies of some CMT patients, however. A balanced risk assessment in considering central blockade would seem prudent lest any deterioration be blamed on the anaesthetic technique. Autonomic neuropathy is not a major finding in CMT. It is usually limited to decreased sweating in the extremities. Autonomic neuropathy does not feature as a clinical problem in any anaesthetic case reports.

Clinically palpable nerve enlargement occurs in 25% of patients with CMT 1. These patients may be at greater risk of positional nerve injuries due to pressure during prolonged procedures. In addition, the use of regional blockade in areas such as the elbow may carry additional risk.

Given the incidence of CMT and the fact that many cases go unrecognized, it would seem that anaesthesia for these patients must be generally uneventful. The cases presenting for corrective limb surgery must be the worst affected and yet adverse events are seldom described.

References

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.


Key words: complications, bronchospasm; anaesthetics, local, lidocaine

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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 (FIO₂ = 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 SpO₂ 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
The inspired oxygen fraction was reduced to 0.35 and 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 $SpO_2$ 20 min after intubation remained at 90% with an $FIO_2$ of 1, and the basal collapse had increased.

Fifty micrograms of salbutamol was given intravenously. The $PFCO_2$ 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 $PFCO_2$ 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 $SpO_2$ 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%. The development of the basal atelectasis is of interest. Atelectasis in dependent dorsal regions is well known to occur during anaesthesia. It can occur very rapidly (within minutes) when patients have been preoxygenated 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. 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 $SpO_2$, 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.

There are three cases of ‘unilateral bronchospasm’ listed in Medline (1966–2000). Two occurred after surgical procedures of the pleura (interpleural analgesia and pleurodesis) and one after subclavian vein puncture. Shantha 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. A direct sympathetic nerve supply to the bronchial smooth muscle has been questioned 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. 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.\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 bronchospasm 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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A 27-yr-old female presented for surgery three times. She had Kartagener’s syndrome. 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. 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). 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 anticlockwise 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?
The anaesthetic implications of Kartagener’s syndrome are varied. The anaesthetist might be involved with patients who have sinus surgery, pulmonary surgery, infertility investigations or possibly cardiac surgery. Of primary importance will be assessment of pulmonary and cardiac structure and function, and also prevention of pulmonary complications in the bronchiectatic patient.

Physiotherapy, postural drainage, antibiotics, bronchodilators and incentive spirometry all have a role perioperatively. Where possible, local or regional anaesthesia is to be preferred to general anaesthesia. In thoracic surgery, the anatomy of the bronchi should be considered before selecting a double lumen tube.

Knowledge of the position of the abdominal organs and of the branching pattern of the main stem bronchi is important in categorizing malpositions; and these must be borne in mind. When dextrocardia occurs with situs inversus, the heart is most frequently normal. When dextrocardia occurs without situs inversus, the visceral situs is indeterminate (situs ambiguus), or if isolated levocardia is present, associated, often complex, multiple anomalies are usually present. Dextrocardia with complete situs inversus occurs in approximately 2 per 10 000 births. The incidence of congenital heart disease is low being about 3%. Kartagener’s syndrome will occur in about 20% of patients. Thus, the incidence being approximately 1 in 50 000 births. Our patient did not have an echocardiogram and on clinical grounds was assumed to have a totally normal heart.

In contrast, dextrocardia with situs solitus or situs ambiguus is less common (1 per 20 000 births) and the incidence of congenital heart disease is extremely high, probably 90% or greater. Dextrocardia with situs solitus usually, although not invariably, associates with severe 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 ambiguus, polyosplenia 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 maldevelopment of the great arteries.

The incidence of isolated levo-cardia (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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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
Shunting of blood across a defect between the two sides of the heart is usually directed by the left-to-right pressure gradient. A large and persistent left-to-right shunt can increase pulmonary blood flow, which over months or years leads to microvascular changes, increased pulmonary vascular resistance and elevated pulmonary artery pressure. When the pulmonary arterial pressure approaches the systemic arterial pressure, the direction of the shunt reverses (the Eisenmenger syndrome). This chronic development of right-to-left shunt (RLIAS) is accompanied by electrocardiographic changes of atrial enlargement and right ventricular hypertrophy. Chest radiography reveals prominent pulmonary arteries.\(^1\)

In contrast, the adult patient with an asymptomatic inter-atrial defect may develop an acute RLIAS following a cardiac or respiratory insult. Acute RLIAS is a separate clinical entity from the Eisenmenger syndrome. Acute RLIAS is often overlooked as a cause of hypoxia\(^2\) but early diagnosis can direct therapy and avoid unnecessary invasive investigations. The three cases reported here illustrate the need to consider RLIAS in the differential diagnosis and investigation of profound hypoxia.

**Case reports**

**Patient 1**

A 67-yr-old man presented for elective repair of an abdominal aortic aneurysm. He had a past history of hypertension, transient ischaemic attacks and a left hemiparesis from which he had made a full recovery. His exercise tolerance was good and a preoperative multiple uptake-gated acquisition scan demonstrated a left ventricular ejection fraction of 67%. Anaesthesia and surgery were uneventful. The perioperative cardiac index was 3-4 litres min\(^{-1}\) m\(^{-2}\) and pulmonary arterial pressures ranged between 28/8 and 52/24 mm Hg. He required admission to intensive care unit (ICU) 3 days postoperatively with hypoxia (\(P_{aO_2}=5\) kPa) on room air, pulmonary oedema, blood pressure of 90/60 mm Hg and oliguria. He required tracheal intubation, positive pressure ventilation, diuretics and inotropes. His oxygenation improved, allowing tracheal extubation after 3 days but after 10 days his \(P_{aO_2}\) was 5.65 kPa and \(P_{acO_2}\) 2.7 kPa on a \(F_{o_2}\) 0.95. He also developed platypleoae; that is he was unable to sit upright because of severe breathlessness but he was less breathless when lying flat, particularly in the left lateral decubitus position.

Initially the underlying cause of this patient’s hypoxia was not clear. Transthoracic echocardiography (TTE) demonstrated good left ventricular function, mild mitral regurgitation and septal hypertrophy. A lung perfusion scintigraphic scan showed no evidence of pulmonary embolism. Thoracic computerized tomography revealed non-specific bilateral shadowing of the lung fields consistent with adult respiratory distress syndrome (ARDS) or infection. Despite antibiotic therapy followed by a trial of prednisolone 70 mg day\(^{-1}\) for possible ARDS, he continued to deteriorate and required reintubation and positive pressure ventilation. A pulmonary artery flotation catheter demonstrated a cardiac index of 2.1 litres min\(^{-1}\) m\(^{-2}\), mean right atrial pressure of 8 mm Hg, mean pulmonary arterial pressure of 37 mm Hg and pulmonary artery occlusion pressure of 3 mm Hg. During inspired nitric oxide therapy at 20 p.p.m. the cardiac index increased to 3.8 litres min\(^{-1}\) m\(^{-2}\), the mean right atrial pressure was 4 mm Hg, mean pulmonary arterial pressure 34 mm Hg and pulmonary artery occlusion pressure 5 mm Hg. The \(P_{aO_2}\) increased from 10.5 to 24.5 kPa on a \(F_{o_2}\) 1.0.

A cardiology opinion was sought concerning the possibility of an inter-atrial shunt, but this diagnosis was thought to be excluded by the absence of a persistent right-to-left pressure gradient; right atrial pressure range of 4-8 mm Hg with a pulmonary artery occlusion pressure range of 3-5 mm Hg. However, an inter-atrial defect was discovered fortuitously during pulmonary angiography when a catheter passed from the right atrium into the left atrium. Bubble contrast transoesophageal echocardiogram (TOE) demonstrated RLIAS with severe impairment of right ventricular function (Fig. 1).

The patient became septicaemic and died on the 26th postoperative day. Post-mortem examination revealed a secundum ASD, absent superior vena cava and anomalous venous drainage via a dilated coronary sinus. The right ventricle appeared normal and the lungs were oedematous. On histological examination, there was mild pulmonary fibrosis but no features of ARDS, organizing pneumonia, or pulmonary hypertension.
Acute right-to-left interatrial shunt

**Patient 2**

A 38-yr-old woman presented with platypnoea and cyanosis that developed over a period of 4 days. Sitting upright exacerbated her breathlessness and she was more comfortable when lying flat despite being profoundly hypoxic. She had a past history of chronic renal impairment and aortic incompetence. At presentation she was inpatient recuperating from an episode of infective endocarditis and gastrointestinal bleeding for which she had required treatment in the ICU.

On physical examination she was apyrexial, heart rate 110 beats min⁻¹, arterial pressure 120/80 mm Hg and the jugular venous pressure was not elevated. An aortic murmur was present, but unchanged from previous examinations; respiratory rate was 20 b.p.m. and chest auscultation was normal. Arterial blood gases on a room air were: pH 7.6, \( P_{aO2} \) 5.6 kPa, \( P_{aco2} \) 3.8 kPa, base excess 6.3 mmol litre⁻¹. A chest radiograph showed a small right pleural effusion. Lung perfusion scintographic scan demonstrated a single segment perfusion defect in the right mid-zone. A preliminary decision to anticoagulate was deferred until a pulmonary angiogram could be performed in view of her recent history of gastrointestinal haemorrhage. During pulmonary angiography, the catheter passed through a patent foramen ovale into the left atrium and left upper lobe pulmonary vein. Radio-opaque dye flowed from the right to the left atrium. Pulmonary arteriography demonstrated no emboli. Mean pulmonary arterial pressure was 12 mm Hg and mean right atrial pressure was 13 mm Hg compared with 8 mm Hg in the left atrium. Right atrial \( P_{O2} \) was 2.8 kPa and the left atrial \( P_{O2} \) was 5.5 kPa. Transthoracic echocardiogram revealed a right-sided pericardial effusion, 4.5 cm in depth, compressing the right ventricle. Left ventricular function was good. Pericardial tap brought immediate relief of the patient’s hypoxia; the \( P_{aO2} \) rising from 5.8 to 30.4 kPa, on oxygen 10 litres min⁻¹ by facemask.

**Patient 3**

A 46-yr-old man presented with breathlessness 4 days after right hemicolectomy for caecal carcinoma. Axillary temperature was 38.5°C, he had a sinus tachycardia 135 beat min⁻¹, arterial pressure 90/60 mm Hg and a respiratory rate of 32 breath min⁻¹. \( P_{aO2} \) was 4.1 kPa and \( P_{aco2} \) 4.2 kPa on room air. Oxygen at maximum flow via a facemask did not improve his hypoxia and he was transferred to ICU. \( F_{O2} \) 0.9 with CPAP 5 cm H₂O produced a \( P_{aO2} \) 7.5 kPa. The patient was not distressed despite his hypoxia and was able to tolerate lying flat for a pulmonary angiogram. Pulmonary angiogram revealed a large embolus in the left-upper lobe pulmonary artery with smaller emboli in the right middle lobe pulmonary artery and right pulmonary vessels peripherally. The catheter passed from the right atrium to the left atrium. Mean right atrial pressure was 13 mm Hg, left atrial pressure 11 mm Hg and mean pulmonary arterial pressure 27 mm Hg. Left pulmonary venous \( P_{O2} \) was 32.3 kPa but the radial arterial \( P_{aO2} \) was 6.24 kPa. A Greenfield inferior vena cava filter was deployed with its base at the level of the L3-L4 disc space. A bolus of tissue plasminogen activator 5 mg i.v. plus an infusion of 5 mg h⁻¹ was instituted. The patient’s oxygenation improved over the next 3 days and thereafter his recovery was uneventful.

**Discussion**

Acute RLIAS is a rare but important cause of profound hypoxia. The pathophysiology of acute RLIAS arises from an inter-atrial defect coupled with a secondary cardiac or pulmonary insult. Patent foramen ovale, which occurs in approximately 30% of the adult population, is present in the majority of reported cases of acute RLIAS. ASD is present in the remainder. A rise in right atrial pressure above left atrial pressure may precipitate RLIAS. This can be the result of obstruction of pulmonary blood flow by pulmonary embolus (Patient 3), increased transmural pressures in asthma, right ventricular hypokinesis following right ventricular infarction or coronary artery bypass grafting, or as a result of elevated \( P_{aco2} \) in hemidiaphragmatic paresis. However, in similar cases right heart pressures can be normal, which indicates that other factors can induce RLIAS. Mechanical distortion of the heart can alter the relative positions of the atrial septum and the inferior vena cava (IVC) which then directs the flow of blood from the IVC towards the inter-atrial defect and into the left atrium. Mediastinal shift following lung resection is the most commonly reported cause of RLIAS. Other causes of distortion of cardiac anatomy with no apparent right-to-left pressure gradient include thoracic trauma, rupture of the papillary muscles, after tuberculosis, thoracic aortic...
aneurysm,\textsuperscript{15} pericardial effusion (Patient 2),\textsuperscript{16} and metastatic cardiac disease.\textsuperscript{17} The mechanism of RLIAS is unclear in cases of morbid obesity,\textsuperscript{18} cystic fibrosis,\textsuperscript{19} adult respiratory distress syndrome\textsuperscript{20} and exposure to low atmospheric pressure.\textsuperscript{21 22}

Platypnoea is a sign that is strongly associated with RLIAS. This sign, first described in 1949 and given the name platypnoea in 1969, occurs in approximately one quarter of reported cases of acute RLIAS.\textsuperscript{23 24} A corresponding postural change in oxygenation is known as orthodeoxia.\textsuperscript{25}

RLIAS is often diagnosed unexpectedly during investigation for other causes of acute hypoxia.\textsuperscript{26} A pulmonary angiography catheter may pass through an inter-atrial defect, as occurred in our patients, but the angiogram may be reported as normal despite the presence of a significant RLIAS.\textsuperscript{4 13} Lung perfusion scintigraphy indicates RLIAS when early activity is detected in the brain, myocardium and kidneys.\textsuperscript{27} It has been advocated as the investigation of choice\textsuperscript{28} but failed to detect RLIAS in our first and second patients and in several other reported cases.\textsuperscript{2 4 29} Injection of radiolabelled microaggregates into the arm has resulted in a normal perfusion scan whereas injection into the foot has resulted in a positive scan.\textsuperscript{30} The use of bubble contrast during the patient’s first echocardiogram might have revealed the RLIAS early enough for surgery to be feasible.

Patient 1 developed acute RLIAS and platypnoea. The nature of the precipitating cause remains uncertain. However, the raised perioperative pulmonary arterial pressures and the subsequent beneficial effect of nitric oxide suggests that pulmonary vasoconstriction was a contributing factor. The diagnosis of RLIAS was discounted because of the absence of a significant right-to-left pressure gradient between the atria. By the time RLIAS was diagnosed, the patient’s condition precluded surgical closure of his ASD. The use of bubble contrast during the patient’s first echocardiogram might have revealed the RLIAS early enough for surgery to be feasible.

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Air embolism during anaesthesia for shoulder arthroscopy

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We report a case of venous air embolism during an elective shoulder arthroscopy in which air was used as a joint distending agent. Venous air embolism was diagnosed by the sudden decrease in the end-tidal carbon dioxide concentration. The patient suffered no serious complications of venous air embolism and made a full recovery. We present this case to make surgeons and anaesthetists aware of the possibility of gas/air embolism during elective arthroscopy, when gas/air is used to distend the joint. This case also illustrates that the end-tidal carbon dioxide monitor, which is part of the standard anaesthetic monitoring system, is very sensitive in detecting venous air embolism.

Keywords: surgery, arthroscopy; complications, air embolism; monitoring

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Venous air embolism is a known complication during anaesthesia for hip replacements, laparoscopies and operative procedures in which the operative site is above the level of the heart, as in neurosurgical and major head and neck procedures, especially in the sitting position. Although rare, venous gas embolism can occur during arthroscopy, when air or another gas is used to distend the joint. We are aware of only one report where air embolism occurred during shoulder arthroscopy. We report an additional case of air embolism during a diagnostic arthroscopy of a shoulder joint, during which the surgeon had injected 50 ml of air into the joint.

Case report

A 28-yr-old man was scheduled for elective arthroscopy of the left shoulder and subacromial decompression. He had suffered left shoulder pain for 2 yr but there was no history of trauma to the joint. Two preoperative MRI scans had not shown any abnormalities in the joint. His health status was unremarkable, apart from a history of mild asthma for which he had not taken any medication in the previous 2 yr. He had undergone an uneventful general anaesthetic in the past for inguinal hernia repair.

Anaesthesia was induced with propofol 200 mg, fentanyl 0.1 mg, and vecuronium bromide 10 mg. The trachea was intubated and ventilation of the lungs was controlled. Anaesthesia was maintained with sevoflurane and nitrous oxide in oxygen. Morphine 10 mg and ondansetron 4 mg was administered. Routine monitoring included end tidal carbon dioxide measurements. The patient was placed in the beach chair position. After a stab incision in the skin, the arthroscope with a pointed trochar was introduced into the left shoulder joint. The joint was seen using the arthroscope and 50 ml of air was injected into the joint by attaching a syringe with air to the inflow portal on the arthroscope. Within 1 min after the injection, a marked decrease in the end tidal carbon dioxide (ETCO₂) from 4.0 to 1.5 kPa was noted. The electrocardiogram showed a 1-mm rise in the ST segment, and the heart rate increased from 56 to 90 beats min⁻¹. There were no changes either in the oxygen saturation or in the non-invasive blood pressure. An air embolism was suspected and the patient was placed horizontal and nitrous oxide discontinued. The end tidal carbon dioxide concentration returned to 4.0 kPa after 4 min. The surgical procedure was allowed to continue after ensuring the condition of the patient was stable and the ETCO₂ concentration had returned to normal. There was an episode of bradycardia 15 min following the incident during which the heart rate decreased to 38 beats min⁻¹. This responded to 0.6 mg of i.v. atropine.

The patient made a full recovery from the anaesthetic at the end of the procedure, without any sequelae. He remained stable for the next 48 h and was discharged home.

Discussion

Venous air embolism may occur during any operative procedure in which the operative site is above the level of the heart and non-collapsible veins are exposed to atmospheric pressure, or when air or any other gas is introduced under pressure into a body cavity. The clinical presentation and complications of venous air embolism depend on the dose and the speed of air entry into the vein and the filtering capacity of the lungs.

Although venous air embolism of some degree is detected in all patients undergoing neurosurgical procedures in the sitting position,¹ it is very rare in shoulder arthroscopy. Air
and other gases have been used to distend the joint in arthroscopy since 1921. Fatal pulmonary air embolism has been reported during arthroscopy of injured knee joints. It is presumed that air entered the circulation through the exposed venous sinuses in these cases. We are aware of only one case of venous air embolism during a shoulder arthroscopy. There are, however, theoretical risks of embolism during this procedure. In the beach chair position, used for shoulder operations, the operative site is higher than the right atrium. In addition, during arthroscopy the pressure inside the joint may be 5–10 times higher than the venous pressure and if a vein is punctured during insertion of the arthroscope, any agent used to distend the joint may enter the venous system.

Because of the low morbidity and anaesthetic complication rate associated with shoulder arthroscopy, the procedure is performed as a day case in most hospitals. Air is commonly used as the joint distending agent because of the better view obtained, when compared with normal saline.

The possible mechanisms of developing a venous air embolism during arthroscopy are: injection of air into an injured joint, where the air can get into the exposed venous sinuses because the intra-articular pressure during arthroscopy is high and may reach peak values of 300 mm Hg with sudden manipulation of the joint; or accidental injection of air into a vein in the joint.

In our case there was no history of trauma, preoperative MRI scans showed no abnormalities in the joint and arthroscopy did not show any disruption in the joint surface. It is presumed that a vein was accidentally punctured during the stab incision or by the introduction of the arthroscope into the joint. The raised intra-articular pressure because of the injected air and the manipulation of the joint resulted in air entering an open vein.

There are several reports in the anaesthetic literature where the use of sophisticated monitors to detect venous air embolism early in operations, with a high risk of development of venous air embolism, has resulted in a significant reduction in morbidity and mortality.

Transoesophageal echocardiography is the most sensitive monitor for early detection of venous air embolism (detects as little as 0.02 ml kg⁻¹ of air). Others include pulmonary artery catheters, pre-cordial Doppler probe and ETCO₂, and nitrogen measurements.

For day case arthroscopies an inexpensive, non-invasive, sensitive and easy-to-use monitor is essential. The pre-cordial Doppler probe meets the above criteria. It is very sensitive, but picks up interference from other sources. Before it becomes a useful tool for early detection of venous air embolism, familiarity with its use is essential. In most district general hospitals, however, capnography remains the main monitor for the early diagnosis of venous air embolism. It has fewer sources of interference than echocardiography and pre-cordial Doppler. It is widely used and is sensitive (sensitivity of 0.4 ml kg min⁻¹), but is non-specific for air embolism. Above all, awareness of the possibility of air embolism, a high index of suspicion and vigilance are most important in arthroscopy when air or other gases are used to distend the joint. Early detection of venous air embolism will allow for prompt treatment before serious clinical complications develop.

A significant venous air embolism will manifest as bronchospasm, hypoxaemia, hypercapnia, decreased ETCO₂, hypotension, cardiac dysrhythmias and cardiovascular collapse, depending on the size of the embolus and the rate of entry of air into the veins.

Management of clinical venous air embolism includes measures to prevent expansion of the embolus and further entry of air into the venous system. The operative site should be lowered below the level of the heart, if possible and flooded with fluids while haemostasis is obtained. Nitrous oxide should be discontinued and oxygen increased to 100%. Blood pressure and central venous pressure should be supported with fluids, vasopressors and inotropes. If a large amount of air is entrained, the left lateral decubitus position may improve right ventricular outflow. Further treatment includes insertion of right atrial catheter and aspiration of air from the right side of the heart. In the event of cardiovascular collapse, cardiovascular resuscitation should be commenced.

Prevention is the key to the management of venous air embolism. Patients at risk should be well hydrated in order to reduce the pressure gradient between the surgical site and the right atrium. Surgical haemostasis is essential at all times. Intravenous air is five times more dangerous than carbon dioxide. Therefore, carbon dioxide or normal saline are safer agents for distension of joints during arthroscopy.

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