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Epidural anaesthesia for caesarian section in a parturient with a single ventricle

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Editor,

Heart disease is a leading cause of maternal death. Management of pregnant women with preexisting cardiac problems should be undertaken by multidisciplinary teams in tertiary centres [1]. Single ventricle anomalies occur in only 1.5% of patients with congenital heart disease [2]. The primary anaesthetic goal is to avoid any haemodynamic change that might increase the right to left shunt and, thereby, increase hypoxaemia [3]. With this in mind, many practitioners have avoided regional anaesthetic techniques in favour of general anaesthesia. We report a case of a pregnant woman with a functionally univentricular heart and double outlet right ventricle who underwent caesarean section using an epidural technique.

Case report

A 32-year-old woman was referred at 30 weeks gestation to our antenatal anaesthetic clinic. Her past medical history included cyanotic congenital heart disease. At the age of 2, she was diagnosed as having congenital heart disease, the exact nature of which was not known. She had a right thoracotomy and a R. Blalock shunt.

Her obstetric history included a caesarean section for partial placental abruption 8 years previously, which had been performed under general anaesthesia without complications.

On admission, the patient was 165 cm, 75 kg and she was New York Heart Association (NYHA) class IIA. Her physical examination showed moderate central cyanosis, marked digital clubbing, a grade 3/6 systolic ejection murmur heard maximally at the upper left sternal border radiating over the entire precordium into her neck. On room air, arterial blood gas analysis revealed a PaO₂ of 68 mmHg. Oxygen saturation by pulse oximetry (SpO₂) was 89%. Haematocrit was 50%. Echo revealed mitral atresia, double outlet right ventricle, pulmonary valve stenosis and severe hypoplastic left ventricle (Fig. 1). The ECG showed normal sinus rhythm with right ventricular hypertrophy.

Because of increasing fetal hypoxia (as shown by fetal bradycardia, pathological blood flow in ductus venous and umbilical vein on Doppler) a caesarean section was electively performed at the 34th week of gestation.

The patient was given ranitidine and metoclopramide intravenously (i.v.) 1 h before the operation. The heart rate and ECG were monitored continuously using a three-lead system, the arterial pressure was monitored directly through left radial artery cannulation and a central line was inserted into the right jugular vein. Endocarditis prophylaxis was given. Pulmonary artery catheterization was considered. However, it was felt that, because of the anatomy, interpretation of values would be extremely difficult.

With the patient in the sitting position, a lumbar epidural catheter was inserted through a standard 18-gauge Tuohy needle at the L3/L4 intervertebral space. Preservative free normal saline was used to test for the loss of resistance. Following a test dose of 3 ml lidocaine 2%, the patient was placed in the supine position with a left lateral tilt.

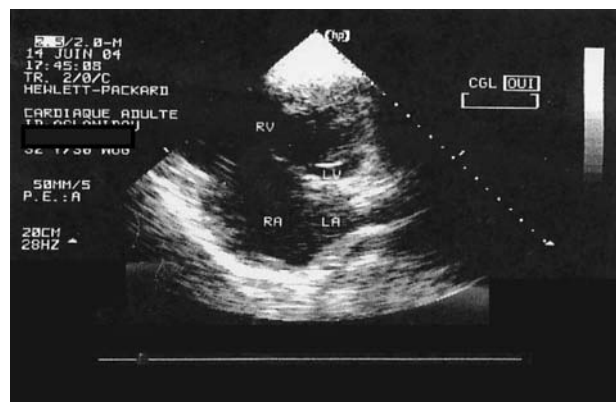
Throughout the procedure, her central venous pressure (CVP) was maintained between 6 and 10 mmHg with i.v. Ringer lactate.

Epidural boluses of 5 ml of ropivacaine 0.75% were given 5, 15 and 30 min after the test dose (with the addition of fentanyl 50 µg to the first two boluses). The choice of ropivacaine was made because of its safer haemodynamic profile [4]. Sensory blockade to touch and cold was achieved at the 6th and 4th thoracic dermatomes, respectively. Cardiovascular parameters remained stable during the development of the block. Blood pressure (BP) ranged from 110/65 mmHg to 125/75 mmHg.

Just after delivery, 5 mg of furosemide was given to the patient and an oxytocin infusion was commenced. Following a fall in BP to 80/40 mmHg, she became nauseous. Treatment consisted of 50 µg increments of phenylephrine to a total dose of 100 µg and another litre of Ringer's solution was infused. Thereafter, BP was kept stable using 5 mg increments of ephedrine to a total of 20 mg.

After the operation, the patient was transferred to the ICU and a continuous infusion of ropivacaine 4 mg h⁻¹ and morphine was commenced. She remained in the ICU for 24 h and in the coronary care unit for another 5 days.

Fig. 1



Two-dimensional echo-apical four-chamber view. Mitral atresia, severely hypoplastic left cavities.

Her postoperative course was complicated by deep vein thrombosis (DVT) in the left leg, even though low molecular weight heparin had been given. The patient was treated conservatively and she was discharged from the hospital 2 weeks later.

Discussion

Congenital heart disease in pregnancy is increasingly common because of the advances in surgery and medical therapy which have taken place over the last 30 years, which means that more affected women are surviving to reproductive age [5]. Single ventricle anomalies are rarely seen in parturients [6].

The management of anaesthesia in these patients should be aimed at providing anaesthesia while maintaining a favourable balance between systemic and pulmonary vascular resistance. This would ensure that there would be minimal change in the amount of right to left shunt [7].

Traditionally in Greece, general anaesthesia has been recommended for these patients because the relative sympathectomy that occurs with regional anaesthesia would tend to decrease systemic vascular resistance (SVR) and, therefore, promote increased right to left shunting.

General anaesthesia, however, carries some risks. These include the potential for tachycardia and increasing BP in response to catecholamine release after laryngoscopy, during anaesthesia and in recovery given the relatively poor pain control achievable with systemic opioids. Intermittent positive ventilation increases intrathoracic pressure, reduces venous return and increases pulmonary arterial pressure.

In addition, general anaesthesia can be associated with difficult or failed intubation and aspiration of gastric contents. The use of regional anaesthetic techniques in high-risk cardiorespiratory patients undergoing elective caesarean section is a new trend, as an increasing number of case reports demonstrate satisfactory cardiovascular stability. Epidural anaesthesia because of its slow onset has a reduced chance of precipitating haemodynamic deterioration and has been used successfully in similar cases [8].

Although epidural anaesthesia can result in patchy or incomplete block, which in turn may lead to undesirable sympathetic stimulation, it was adequate for this patient.

The patient received an oxytocin infusion. Oxytocin can cause direct vasodilatation and reduction of the SVR with a compensatory increase in heart rate and cardiac output. These effects can be decreased by a slow i.v. infusion.

The importance of maintaining SVR has already been emphasized. The prophylactic and therapeutic use of vasoconstrictor drugs, therefore, seems logical and attractive. We initially used phenylephrine because the patient was tachycardic. But later, when the heart rate was within normal values, we chose ephedrine, as it is the most commonly used vasoconstrictor in obstetric practice [9].

Patients with cyanotic congenital heart disease are at increased risk of developing air embolism and thromboembolism. Therefore, the i.v. lines should be carefully checked; the patient should also receive heparin [10].

Although epidural anaesthesia decreases the likelihood of postoperative DVT formation, the patient developed a DVT postoperatively in her left leg despite being on low molecular weight heparin.

Invasive monitoring has been used, except for a pulmonary artery catheter. It has been suggested that the use of pulmonary artery catheters has not reduced the peripartum mortality; also insertion can be difficult owing to low cardiac output and anatomy. Echocardiography represents another possibility for monitoring these patients and could be used.

The risk of congenital heart disease in the fetus is increased from twice to 20-fold, depending on the nature of the mother's lesion [11].

As haemodynamics do not return to normal for several days, continued close monitoring in a high-dependency unit is necessary for a minimum of 72 h.

Women with complex congenital heart disease rarely achieve successful pregnancy. For optimum outcome these patients should be managed by multidisciplinary

teams. The clinical team should include obstetricians, cardiologists, anaesthetists and midwives with experience in such cases. This case adds to the total experience of a subject where, due to considerable individual variations, every such case tends to be unique.

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Topical airway anaesthesia for awake fiberoptic intubation

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Securing the airway during general anaesthesia in patients with difficult airways poses a risk to the patient and presents challenges for the anaesthesiologist [1–3].

Therefore, awake fiberoptic intubation is the safest approach to airway management. It is usually carried out under topical anaesthesia or light sedation. Upper

airway anaesthesia should be administered to a level sufficient to suppress the gag, swallow and cough reflexes, and to avoid pain [1–3]. The more cooperative the patient is, the easier the procedure [4].

There are multiple ways of administering anaesthesia for fiberoptic intubation via the oral or nasal route.

We describe the use of an ultrasonic nebulizer (Multi-sonic Schill GmbH, Probstzella, Germany) that has been designed to deliver liquid medication in the form of droplets with an average diameter of just 3.5 µm to the airways. Ultrasonic nebulizers are often used in the treatment of pulmonary hypertension [5]. They can be easily integrated into the breathing circuit using an adapter. This arrangement provides the delivery of a sufficient dose of local anaesthetic only during inspiration as well as an optimum preoxygenation. Because of the particularly fine mist of vaporized anaesthetic, a remarkably lower dose of lidocaine is needed.

Two case examples are described in the following.

The first case involves an awake fiberoptic intubation in a setting documented by experienced anaesthesiologists as being a 'cannot ventilate – cannot intubate' situation during previous general anaesthesia. A patient undergoing elective aortic valve replacement and aortocoronary bypass surgery for severe aortic stenosis and three-vessel coronary artery disease required tracheal intubation. The patient had concomitant chronic obstructive pulmonary disease and used nasal continuous positive airway pressure every night to treat obstructive sleep apnoea. After a detailed explanation of the procedure on the day preceding surgery, the patient was premedicated with 7.5 mg of oral midazolam and was administered 100 mg of lidocaine (2%) via the ultrasonic nebulizer described above. Awake oral fiberoptic intubation was successfully performed. The cooperative patient did not exhibit signs of stress and, consequently, did not require additional analgesedation. General anaesthesia was induced subsequently by using intravenous etomidate (0.3 mg kg⁻¹), fentanyl (0.01 mg kg⁻¹) and pancuronium (0.1 mg kg⁻¹).

The second case describes an awake fiberoptic intubation in emergency transnasal surgery for optic nerve decompression because the patient manifested acute amaurosis that was induced by a known tumour. The patient had undergone several previous surgeries for carcinoma of the base of the tongue (mouth floor resection and resection of parts of the tongue, flap for closure of the defect, and neck dissection). Evaluation of anamnestic data of previous operative interventions revealed that the only possibility was to fiberoptically intubate this patient. Painful experience with previous awake fiberoptic intubations resulted in the patient exhibiting signs of anxiety, accompanied by a stress reaction. After a detailed explanation of the

procedure, the patient was premedicated with 2 mg of intravenous midazolam. Although being treated with continuous infusion of remifentanyl $0.1 \mu\text{g kg}^{-1}$ body weight min^{-1} , the patient received a 10 min treatment of 100 mg lidocaine (2%) that was aerosolized by the ultrasonic nebulizer. Awake fibreoptic orotracheal intubation was performed without any problems. The patient was cooperative during the entire process. General anaesthesia was then induced with propofol (2 mg kg^{-1}), remifentanyl ($0.3 \mu\text{g kg}^{-1}$ body weight min^{-1}) and atracurium (0.5 mg kg^{-1}).

Preparations for nasotracheal intubations should include instilling a vasoconstrictor into the nasal cavities in order to reduce mucosal oedema [1].

Local anaesthesia can be applied to the upper airway in multiple ways.

Glossopharyngeal and superior laryngeal nerve blocks are often difficult to perform and may involve damage to nerves. If the agents are administered topically in the form of gargles, sprays, lozenges, creams or impregnated swabs, high doses [6,7] up to the maximum dose [8] of topical anaesthetics may be necessary. Neither of these two approaches results in an anaesthetized trachea distal beyond the glottis. Topical administration of anaesthetic agents makes it difficult to say whether or not the larynx was adequately anaesthetized. Laryngeal anaesthesia may be provided by translaryngeal injection, via the working channel of the flexible bronchoscope or via a (e.g. epidural) catheter inserted [9] at end-expiration and under the anaesthesiologist's direct vision. All these methods are likely to induce cough in the patient.

Nebulizing the topically applied inhaled anaesthetic is a possible solution to this problem. Customary compressed air-driven jet nebulizers atomize only highly concentrated anaesthetics (e.g. lidocaine 4–10%), so that the maximum dose of local anaesthetic (lidocaine 9 mg kg^{-1} body weight) can be exceeded within a short period of time [6]. Woodall *et al.* [10] recently published the experiences of anaesthesiologists undergoing awake fibreoptic intubation, one-third of whom found the side effects produced by lidocaine to be the most common complication.

In the two cases described above, the use of an ultrasonic nebulizer effected sufficient airway anaesthesia with considerably lower doses of lidocaine. Upper airway anaesthesia and suppression of gag and cough reflexes was obtained by using only 100 mg of vaporized lidocaine. Thanks to cooperative patients, that is, they stuck out their tongues and raised the floor of their mouths, the pharyngeal lumen was fully opened. This manoeuvre essentially improved the view during endoscopy and made it so much easier to perform intubation.

We believe that this procedure, involving the use of an ultrasonic nebulizer, offers important advantages compared with the other procedures mentioned as follows.

Considerably smaller doses of local anaesthetics are required [2], and adequate preoxygenation and anaesthesia of the airway are achieved rapidly, easily and simultaneously. The ultrasonic nebulizer can be easily integrated into any system, it is easy to clean and sterilize and it reduces the number of devices that are needed in such clinical situations.

Ultrasonic nebulizers are tailored to meet the patients' needs and enable them to cooperate with the anaesthesiologist. These findings may influence clinical practice in the management of the difficult airway during anaesthesia.

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Severe vancomycin-induced drug rash with eosinophilia and systemic symptoms syndrome imitating septic shock

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Editor,

Drug rash with eosinophilia and systemic symptoms (DRESS) syndrome is a rare adverse reaction to drugs [1,2]. This syndrome is well described with anticonvulsants, sulfonamides, dapsone, allopurinol, and minocycline [1,2]. Few cases of vancomycin-induced DRESS syndrome have been recently reported [3], but none in its severe presentation, which mimics septic shock. A prompt diagnosis is most important, as the mortality rate can be as high as 10% [4]. In addition, high dose of steroids as a specific therapy of DRESS syndrome may be necessary, which is especially challenging in the context of septic shock. Skin biopsy is of major interest in supporting the diagnosis of severe DRESS syndrome.

Case report

A 38-year-old white woman, with no medical history or known drug allergies, was referred to our hospital for an emergency aortic valve replacement by allograft because of an acute endocarditis of native valves with acute pulmonary oedema for *Streptococcus oralis*. The patient gradually improved with amoxicillin and gentamicin therapy.

On day 16 of hospitalization, vancomycin (500 mg three times daily) was added because of fever, and a second surgical procedure was performed on day 23 to replace the previous allograft because of the association of fever, unstable haemodynamic and new vegetations and septal abscess on repeat echocardiography. The patient gradually recovered with amoxicillin–gentamicin–vancomycin treatment and was weaned from ventilator at day 25. On day 43, vancomycin administration was discontinued because of rapid-onset neutropenia (2.5 g l^{-1}). The next day, fever recurred but was associated with a facial and thoracic rash, and at day 47, rifampicin and targocid were introduced in order to broaden the antibiotic spectrum. On day 50, the patient developed a new, persistent high fever associated with upper body erythema and renal failure. On the basis of echocardiographic results, the patient immediately underwent a third aortic allograft change. All cultures were subsequently negative for any growth. At day 52, rifampicin and teicoplanin treatments were stopped, whereas amoxicillin and gentamicin treatments continued. The patient presented a severely compromised multiorgan failure requiring catecholamine support, mechanical ventilation, and dialysis. In addition, fever, nodal enlargement, upper body erythroderma, and facial oedema were apparent on physical examination.

Blood tests showed a major hyperleucocytosis (50 g l^{-1}) with hypereosinophilia (2.82 g l^{-1}).

A skin biopsy was performed, which revealed basal and suprabasal lymphocytosis and exocytosis with vacuolization and keratinocytosis necrosis eosinophilia in the epiderm. The superficial layer of derma presented a lymphocytic infiltrate with perivascular polynuclear eosinophilia. A severe form of DRESS syndrome was diagnosed, as supported by the skin biopsy and clinical signs, including oedema of the face, erythroderma, and visceral involvement (nodal, hepatic, and renal). According to the 'causality-assessment methods', which determine the likelihood of drug involvement in a suspected episode of adverse event [5], every drug administered to the patient during her stay at the hospital was considered. Vancomycin and teicoplanin both appeared as the drugs most likely responsible for this severe DRESS syndrome.

Because of the life-threatening multiorgan system failure, high-dose intravenous corticotherapy (methylprednisolone $1 \text{ mg kg}^{-1} \text{ day}^{-1}$) was started on day 61. The patient rapidly improved and was discharged home within a few weeks.

Discussion

DRESS syndrome is a well recognized disease in dermatology, but severe forms resulting in multiple organ failures are very rare [1], and only very few have been previously reported. In its typical form, DRESS syndrome usually appears within 2–8 weeks after the first drug use [2]. It is characterized by fever, erythrodermia and oedema, which are usually predominant in the periorbital region. Superficial lymphadenopathy is very frequent, and hepatic impairment is the most common sign of visceral involvement, as demonstrated by cholestasis, hepatitis, or fulminant liver failure [6]. White blood count may rise as high as 50 g l^{-1} with hypereosinophilia over 20 g l^{-1} [4].

The DRESS severity stems from the visceral involvement that can result in clinical syndromes consistent with multiple organ dysfunction involving myocarditis, respiratory failure, hepatitis, renal failure, pancreatitis, thyroiditis, blood dyscrasia, and haemolytic anaemia. This is particularly important because the presentation can easily be mistaken for septic shock.

However, delay in diagnosis and suspension of the offending responsible drug can lead to a mortality of up to 10% [4]. Moreover, in these severe forms, corticosteroid therapy is recommended, and steroid-resistant cases might even require immunosuppressive therapy with agents such as cyclophosphamide or cyclosporine [2,3]. Clinical suspicion of the DRESS syndrome and subsequent timely diagnosis are particularly important

because immune suppression with high-dose corticosteroids or other immunosuppressive therapies is the mainstay of the treatment of DRESS. Skin biopsy provides the key supporting evidence to conclusively diagnose DRESS syndrome in these situations.

In clinical practice, it may be difficult to identify the specific offending agent responsible for the DRESS syndrome. Often, many agents are used simultaneously, and questions about other potential causes may be inadvertently omitted. 'Causality-assessment methods' provide a uniform approach to determine the likelihood of drug involvement in a suspected episode of adverse event [5]. These methods provide convenient, practical tools for assessing the probability that a given reaction can be attributed to a specific drug. Every medication should be considered as a suspected drug. Regarding hypersensitivity reaction to vancomycin, allergic cross-reactivity between teicoplanin and vancomycin has been reported, but its occurrence appears infrequent; thus, hypersensitivity to vancomycin is not a contraindication to the use of teicoplanin if needed [7].

We described a severe vancomycin-induced DRESS syndrome mimicking septic shock. Moreover, the disease carried on, even after interruption of vancomycin and its replacement with teicoplanin, possibly because of cross-reactivity between teicoplanin and vancomycin. The main point in the current case lies in the severity of the multiorgan system failure that could easily be attributed to septic shock. However, several points are inconsistent with septic shock and should help the astute clinician make the correct diagnosis; erythrodermia and facial oedema, lymphadenopathy, negative bacteriological samples, hypereosinophilia, and failure of the usual treatment of septic shock all point towards DRESS syndrome. Skin biopsy is of major interest in this situation and should be performed early as treatment of severe antibiotic-induced DRESS syndrome involves immunosuppressive therapy and antibiotics.

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Cranial subdural haematoma: a rare complication of spinal anaesthesia

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Editor,

Postdural puncture headache (PDPH) is the most frequent major complication after spinal anaesthesia. In the majority of cases, the symptoms subside within a few days when treated with analgesics and bed rest. However, careful postoperative follow-up of the patient with PDPH is crucial. Intracranial subdural haematomas are rare but could be a lethal complication after epidural or spinal anaesthesia, as well as following myelography [1–3].

This report describes the occurrence and late diagnosis of a cerebral subdural haematoma in a 39-year-old female patient following spinal anaesthesia. Her only complaint was a headache, which was assumed to be PDPH.

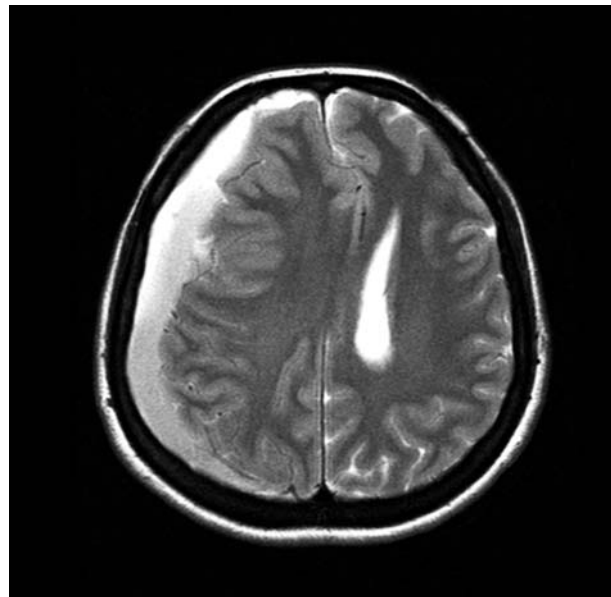
A 39-year-old woman was admitted to our Algology Department with a complaint of a severe headache. She had undergone an abdominal scar revision operation 21 days previously under spinal, and then general anaesthesia. She had no history of trauma, headache, or coagulation abnormalities before the surgery. The preoperative laboratory blood tests, including platelet count, prothrombin time (PT), and activated partial thromboplastin time (APTT), were normal. The patient received no anticoagulants. Spinal anaesthesia was performed in the sitting position at the L3–4 interspace with a midline approach, using a 25-gauge Quincke needle (Spinocan; B. Braun, Melsungen, Germany). Subarachnoid puncture was successful on the first attempt, and 15 mg of 0.5% heavy bupivacaine and 25 µg fentanyl were administered.

Twenty-five minutes after spinal anaesthesia, the sensory level was inadequate; thus, general anaesthesia was administered. The intraoperative course was uneventful, and the haemodynamic parameters were stable throughout. The immediate postoperative recovery was uncomplicated. On the first postoperative day, the patient experienced a mild frontal headache, which was assumed to be PDPH by the surgeons, and intravenous fluids and 100 mg of oral diclofenac sodium were administered. The headache improved partially following hydration and bed rest. The patient was discharged on the second postoperative day with prescriptions for anti-inflammatory analgesics, caffeinated drinks, and bed rest. At home, her headache improved partially 1 or 2 h after analgesics, but worsened again. On the 7th postoperative day, she was admitted to the hospital for suture removal and she repeated her complaint to the surgeons. Again, they concluded that the complaint was PDPH and suggested the same prescriptions. During the course of the following days, she was also evaluated in the Emergency Department on several occasions and was treated with parenteral analgesics without further consultation for other causes of severe headache. On the 21st postoperative day, she was admitted to our Algology Department with a complaint of persistent headache which did not resolve with bed rest and analgesics. The characteristics of the headache changed from postural to nonpostural, and she described the headache as severe and throbbing. The headache was consistently in the left retroorbital and frontal area. The physical and neurological examinations were normal. There were no meningeal signs or papilloedema. The laboratory tests, including haemoglobin, leucocyte and platelet counts, bleeding time, clotting time, PT, APTT, and liver and renal functions, were normal. She underwent an urgent cerebral magnetic resonance imaging (MRI) scan to exclude intracranial disorder. The MRI scan revealed a subdural haematoma (1.5–2 cm) overlying the right fronto-parietal lobe causing compression of the underlying brain, obliteration of the sulci in the right frontal region, and a 12 mm intracranial shift with subfalcine and uncal herniation (Fig. 1).

The patient was transferred to the Neurosurgery Department. Emergent evacuation of the haematoma by the burr hole drainage method was performed. The headache improved immediately after surgery. She received regular neurological follow-up. On the 3rd postoperative day, she was discharged from the hospital without any problems.

Subdural haematomas are a rare, but serious complication of dural puncture. Cases have been reported following accidental dural puncture with an epidural needle and even after spinal anaesthesia, myelography, discography, and diagnostic lumbar puncture [4,5]. The true incidence of subdural haematomas following dural puncture is unknown.

Fig. 1



MRI showing a 1.5–2 cm subdural haematoma overlying the right fronto-parietal lobe and displacement of the midline structures.

The presumed mechanism for the development of an intracranial haematoma is the loss of cerebrospinal fluid (CSF) with low CSF pressure, leading to traction and tearing of the intracranial subdural veins [6,7]. Excessive leakage of CSF through the dural puncture (≥ 250 ml) may cause caudal displacement of the intracranial structures, which may result in subdural haematoma formation [8,9]. Cerebral atrophy, cerebral aneurysm, arteriovenous malformations, pregnancy, dehydration, anticoagulant use, and excessive CSF leakage associated with multiple dural punctures and large dural holes are thought to be contributing factors in the pathogenesis of subdural haematomas. As our patient received general anaesthesia after spinal failure, the positive pressure ventilation was associated with a Valsalva phenomenon which likely decreased the epidural venous pressure and subsequent intraspinal pressure leading to an increase of the CSF dural leak. Therefore, excessive CSF leakage may have been the contributing factor in the present case, though the possible rupture of a preexisting A-V aneurysm malformation is conceivable, especially considering that cranial angiography was not performed.

Headache is a frequent major complication after lumbar puncture. PDPH occurs or worsens shortly after assuming the upright position and disappears or improves after resuming the recumbent position. Additional symptoms may include neck pain, nausea, emesis, interscapular pain, photophobia, diplopia, dizziness, hearing changes, visual blurring, cranial nerve palsies, and radicular upper extremity symptoms [10]. The occurrence of a subdural

haematoma increases the intracranial pressure, which can be associated with nonpostural headache, convulsions, hemiplegia, disorientation, and more serious neurologic symptoms. Differentiation between the neurologic symptoms of PDPH and subdural haematoma can be difficult. A change in headache characteristics from postural to nonpostural should be a warning sign. Macon *et al.* [11] reported that the headaches associated with subdural haematomas are more severe than PDPH and persistent. Suess *et al.* [3] stated that headache lasting for more than 5 days was the chief complaint in 17 reported cases of intracranial haemorrhage after myelography. In our patient, the headache developed on the first postoperative day and lasted for 21 days. It was nonpostural, persistent, and located in the frontal and left retroorbital area. In our patient, the only warning sign was a severe headache, despite the dimension and intracranial effects of the subdural haematoma. In our case, further evaluation for severe nonpostural headache was not performed, despite several admissions to the Emergency Department.

A CT scan of the skull usually leads to the correct diagnosis. However, a 7–21-day-old intracranial haematoma may have the same radiological density as the brain, so MRI or CT scan with contrast may be more reliable [1]. Thus, MRI was chosen to exclude subdural haematoma on the 21st postoperative day.

The delayed diagnosis in our case implied that the subdural haematoma was not taken into consideration by the surgeons as a complication of spinal anaesthesia. In our case, there was a major delay between the onset of symptoms and the diagnosis. The surgeons considered the prolonged postoperative headache as a benign complication of spinal anaesthesia, and they did not consult us until the 21st postoperative day. The consideration in our department now is that every patient is instructed regarding this possible complication and asked to contact the anaesthesiologist in the case of a change in the characteristics of the headache, that is, from postural to nonpostural.

In conclusion, a prolonged, nonpostural, severe headache may be the only warning sign of a cranial subdural haematoma following spinal anaesthesia. Any patient experiencing a nonpostural headache following spinal anaesthesia must be investigated for other causes of headache.

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Brain stem tension pneumocephalus leading to respiratory distress after subdural haematoma evacuation

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Editor,

Pneumocephalus, or air within the intracranial cavity, is a common occurrence after the cranial surgery [1]. Tension pneumocephalus in the posterior fossa compressing brain stem is previously reported in the sitting [2] or prone position [3]. To the best of our knowledge, it has not been reported in the supine position. We report a case of brain stem tension pneumocephalus after craniotomy for chronic subdural haematoma (SDH) evacuation performed in supine position. Increase in the intracranial pressure due to pneumocephalus leads to postoperative respiratory distress requiring mechanical ventilation.

A 54-year-old, 70-kg man admitted with bilateral chronic SDH was scheduled for left craniotomy and haematoma evacuation, followed by burr hole evacuation of haematoma on the right side under general anaesthesia. All preoperative investigations including pulmonary function tests were within normal limits.

After informed consent, routine monitoring was instituted, and anaesthesia was induced. Anaesthesia was

maintained with 60% air in oxygen and propofol infusion with intermittent doses of fentanyl and vecuronium. Arterial blood gas tension was normal (pH 7.43, p_{aO_2} 104.2 mmHg, p_{aCO_2} 38 mmHg) following intubation. After the frontal craniotomy and haematoma evacuation on the left side, the head was turned to the right for burr hole and haematoma evacuation. During the burr hole evacuation, the head end of the table was tilted about 45° above heart level and then backwards to facilitate evacuation of haematoma. The table was tilted two more times within the next 10 min again at the surgeon's request. Total duration of surgery was 90 min. The intraoperative period was uneventful. At the end of surgery, neuromuscular block was reversed with neostigmine and glycopyrrolate. The trachea was extubated, and the patient was fully awake and following commands. Head lift and handgrip were adequate. Thereafter, the patient was shifted to the ICU. Within 10 min of shifting to the ICU, the patient became restless and irritable, and respiration became irregular and shallow. At that time, pulse was 100 beats min^{-1} , blood pressure 140/86 mmHg, respiratory rate 30 breaths min^{-1} and Sp_{O_2} 100%. Auscultation of the chest was unremarkable. Arterial blood gas tension showed pH 7.23, p_{aO_2} 260.6 mmHg and p_{aCO_2} 67.8 mmHg. In view of the high p_{aCO_2} , the respiration was supported by bag and mask. However, even after 5 min, the patient's clinical condition did not improve. He was drowsy and responding only to painful stimulus. It was then decided to initiate mechanical ventilation. The trachea was intubated after giving propofol 100 mg intravenously (i.v.) and rocuronium 70 mg i.v. An urgent computed tomography (CT) of the head was done that revealed air around the brain stem (Fig. 1). The patient was sedated with propofol infusion and ventilated overnight. The following morning, a repeat CT was done that showed that the air around the brain stem had resolved. Neurological response was assessed after stopping sedation. He was conscious following verbal commands and moving all four limbs. The patient was thereafter weaned off the ventilator and extubated. He was discharged from hospital on the third postoperative day with intact neurological status.

Tension pneumocephalus is not a rare complication of neurosurgical procedures. Tension pneumocephalus may present as deterioration in the level of consciousness, with or without focal signs, restlessness, generalized convulsions or even cardiac arrest [4]. Posterior fossa tension pneumocephalus has been reported previously after surgery performed in sitting [2], prone [3] and even lateral position [5].

In our case, posterior fossa tension pneumocephalus occurred in supine position. The probable mechanism causing air collection near the brain stem could have been as follows – initially, a craniotomy for left-sided haematoma evacuation was carried out. In spite of all precautions

Fig. 1



Noncontrast computed tomography of head showing air around the brain stem (arrows).

(isotonic saline instillation before dura closure, keeping drainage site at the lowest point of head) being taken, air may have still collected in the left frontal region. The head was thereafter turned to the left for the burr hole evacuation of the haematoma on the right side. The table was tilted thrice upwards and downwards to facilitate the haematoma evacuation. During the head turning or table tilting, the air may have transgressed from the supratentorial compartment to the infratentorial compartment. When the patient was reversed, a transient rise in the intracranial pressure would have occurred leading to conversion of pneumocephalus to tension pneumocephalus. This tension pneumocephalus could have caused compression on the brain stem leading to irregularity in respiration. The other possibility could have been an inadequate reversal of the neuromuscular blockade. As neuromuscular monitoring was continuously performed until reversal, this possibility of residual neuromuscular paralysis is unlikely.

Aspiration of air is the treatment of choice for emergency decompression of tension pneumocephalus. Young *et al.* [6] have described four cases of tension pneumocephalus after posterior fossa surgery. In all cases but one, surgical aspiration improved the neurological status. Aspiration was not considered in our case, as the location of the air was near the vital structure in the brain stem. Instead, we initiated mechanical ventilation to tackle the respiratory distress with irritable behaviour (probably due to high p_{aCO_2}) so as to allow the tension pneumocephalus to resolve spontaneously. The posterior fossa is a narrow space with small volume. Therefore, even a small

quantity of collected air, as in our case, could have led to a significant increase in the intracranial pressure. Hence, in the event of respiratory distress following craniotomy, the possibility of tension pneumocephalus in posterior fossa should be considered.

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One-lung ventilation in a patient with tracheobronchomegaly: a case report and literature review

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Editor,

A 67-year-old man presented for an elective Ivor–Lewis oesophagectomy as treatment for carcinoma of the oesophagus. He had been diagnosed with late-onset asthma 15 years previously and was managed with a combination of inhaled bronchodilators and steroids. His pulmonary function tests showed severe, partially reversible airways obstruction; however, he denied any limitation in exercise tolerance.

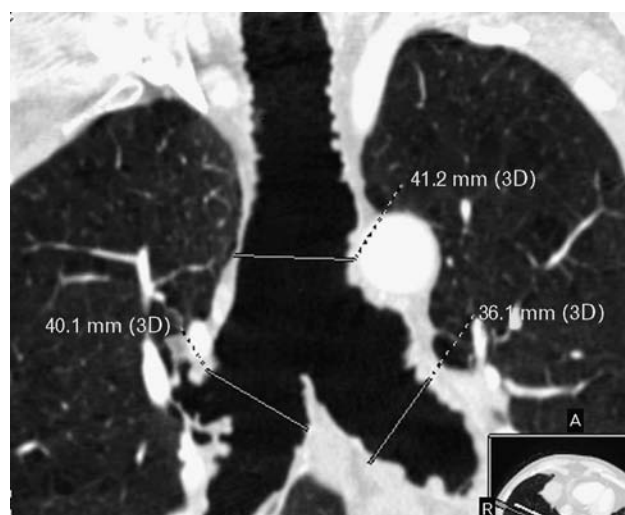
After induction of anaesthesia, the trachea was intubated with a size 39 F Bronchocath left-sided double-lumen tube (Mallinckrodt Medical, Inc., St. Louis, Missouri, USA). However, it quickly became apparent that an adequate seal was not being achieved around the tracheal cuff in spite of inflation with increasing amounts of air. A fiberoptic bronchoscope was used in an attempt to confirm correct placement of the tube, but the views obtained were very poor secondary to a collapsing airway lumen and copious amounts of purulent sputum. It was

impossible to visualize the carina through the tracheal lumen or the left subcarina through the bronchial lumen. Further attempts were made at positioning the tube after suctioning but were eventually abandoned as it was felt that tube positioning could not be achieved safely. The patient was therefore woken and transferred to the recovery room.

On further respiratory and radiological review of the original staging computed tomography (CT) scans, tracheobronchomegaly was noticed. The trachea measured 41 mm at its maximum diameter, and the left and right main bronchi measured 36 and 40 mm, respectively. A corrugated outline and sacculations were also present (Fig. 1). These CT appearances were not recognized on the original radiological report. On the chest radiograph, however, the main airways appeared normal. Consequently, a diagnosis of tracheobronchomegaly, or Mouniere–Kuhn syndrome, was made. Following discussion at a multidisciplinary meeting, and after counselling the patient in relation to his condition, including the increased risk of respiratory-related morbidity and mortality, he was rescheduled for his oesophagectomy.

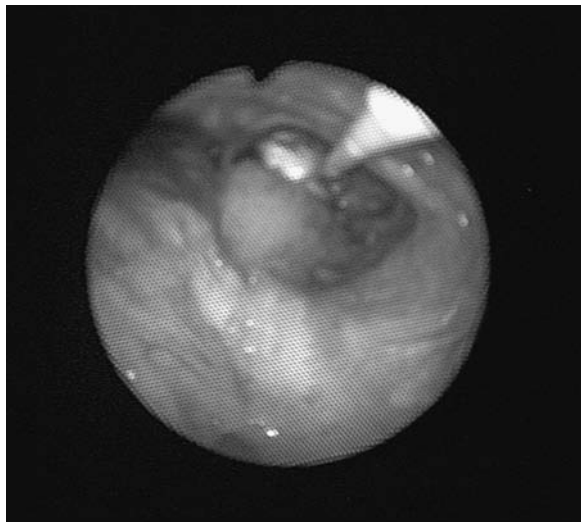
Anaesthesia was induced in a manner identical to that previously mentioned, and his trachea was intubated with a size 9.5 mm endotracheal tube (Mallinckrodt Medical, Inc.). Twenty millilitres of air was required to inflate the cuff in order to achieve an adequate seal within the trachea. Fiberoptic bronchoscopy revealed a floppy collapsing trachea and main bronchi, with visualization possible only during the inspiratory phase of positive pressure ventilation when the airways were stented

Fig. 1



An oblique coronal multiplanar computed tomography reconstruction of the chest showing markedly dilated trachea and main bronchi with sacculations.

Fig. 2



Bronchoscopy image at the time of placement of right endobronchial blocker. Copious purulent secretions can be seen.

open by pressure. Copious secretions were also apparent, causing further deterioration of views (Fig. 2). A size 9 elliptical Arndt endobronchial blocker (Cook, Bjæverskov, Denmark) was then advanced over the bronchoscope into the right main bronchus, and the balloon was inflated in an attempt to seal it off. This required a volume of 35 ml of air, which was far in excess of the 12 ml recommended by the manufacturer. In spite of attempting to reposition the bronchial blocker, the volume of inflated air remained very high.

The abdominal stage of the operation proceeded uneventfully; however, following the onset of one-lung ventilation (OLV) and the subsequent right-sided thoracotomy, it was found that the lung was only partially deflated. Further repositioning of the blocker was attempted in case movement had occurred while the patient was turned from the supine to the left lateral position. This failed to improve the lung deflation, and it was assumed that a leak still existed around the inflated cuff due to the enormously dilated bronchus. No further air was introduced into the cuff because of fears that any additional overdistension may lead to rupture of the balloon within the bronchus. The surgeon proceeded to pack the partially inflated lung away from the surgical field, but this proved to be quite difficult; hence, the remainder of the operation was made somewhat awkward by the poor surgical access. As expected, a degree of hypoxia was encountered during OLV, which improved with the usual manoeuvres.

On completion of the operation, the patient was transferred to the ICU where he remained intubated for 3 days.

He was finally discharged from the hospital 3 weeks after his operation and has been making a steady recovery since.

Tracheobronchomegaly, or Mouniere–Kuhn syndrome, is a rare condition affecting the lower respiratory tract. It is characterized by marked dilatation of the trachea and main bronchi and is associated with chronic pulmonary parenchymal disease [1]. Although it is considered to be extremely rare, with only 82 cases reported worldwide in a review of the literature in 1988, the actual number is likely to be much higher as patients can be asymptomatic or the diagnosis often overlooked [2].

The pathophysiology of tracheobronchomegaly is characterized by marked weakness of the cartilaginous and membranous portions of the trachea and main bronchi [3]. This is due to severe atrophy or absence of longitudinal elastic fibres and thinning of the muscularis mucosa, which consequently allows tracheal and bronchial dilatation. Dynamic studies of the airways demonstrate marked flaccidity of the trachea and main bronchi [4]. On deep inspiration, the airways can distend to vast proportions and then, on expiration, can markedly collapse. During forced expiration, or on coughing, the trachea and main bronchial lumens can completely occlude. This luminal obliteration renders the cough mechanism ineffective and, together with retained secretions, predisposes to chronic, recurrent pulmonary infection.

The diagnosis is made by identification of a dilated trachea and main bronchi, either on chest radiograph or more commonly on CT scanning [4]. The mean transverse diameter of the trachea is 18.2 mm in men and 15.2 mm in women, with the upper limits of normal being three SDs greater than the mean, giving values of 21.8 and 19.4 mm in men and women, respectively [5]. The tracheal diameter in the patient presented in this case was 41 mm, one of the largest reported.

To our knowledge, this is the first case reporting OLV in a patient with tracheobronchomegaly and only the second reported case of general anaesthesia [6]. In the management of our patient, various factors were taken into consideration. First, the method employed to isolate the lung for thoracotomy. It had already been established that the cuffs on a conventional 39 F double-lumen tube could not be inflated sufficiently to achieve an adequate seal. Although it had not been established at the time of anaesthesia that our patient had grossly dilated airways, we could have considered an attempt with a 41 F tube. This would have been unlikely to be successful, however, and direct trauma from repeated attempts at positioning would have probably damaged the thin and atrophied tracheal walls.

Secondly, the large cuff volumes that were used at the second operation, both in the endotracheal tube and in

the bronchial blocker, were very concerning. Inability to produce an effective seal could lead to pulmonary aspiration, and gross overinflation could lead to cuff rupture and associated damage to the weakened tracheobronchial walls. If cuff rupture were to have occurred intraoperatively, the requirement to reinsert one or both tubes while in the lateral position would be extremely difficult and also hazardous.

Another possible method of isolating the lung could have been by inserting the endotracheal tube directly into the left main bronchus at the time when OLV was required and preferentially ventilating it. The volume of air required for cuff inflation would again have been very high, and it is not certain whether an adequate seal could have been achieved. A case study in *Anaesthesia* in 1989 recommended using an uncuffed tube for intubation together with throat packing to reduce or prevent gas leak and possible pulmonary aspiration [6]. Although a useful suggestion during two-lung ventilation, this would not have been beneficial in this case. As we failed to achieve adequate OLV in this patient both with a double-lumen tube and with a bronchial blocker, the only remaining option for us may have been to consider using high-frequency jet ventilation. Double-lung jet ventilation has been proven to be a safe technique of ventilation for thoracotomy and, indeed, is considered comparable to OLV [7]. This could have been achieved by using a conventional endotracheal tube with a special adapter.

Tracheobronchomegaly is a rare, but probably often overlooked, condition that has implications for the provision of adequate, safe anaesthesia. This case report will hopefully alert colleagues to the potential problems of undertaking anaesthesia, and in particular OLV, in patients with chronic suppurative lung disease.

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Increase of propofol requirement after repeated administration for experimental anaesthesia

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Editor,

We report on the apparent development of tolerance to the Electroencephalography (EEG) effects of propofol in three female participants during a study on the influence of experimental anaesthesia on nociception and pain with repeated propofol administration.

After approval by the Ethics Committee of the Medical Faculty of the University of Erlangen-Nuremberg and written informed consent, 22 healthy volunteers (71 ± 13 kg, 32 ± 8 years, 12 men, 10 women) were enrolled. Three sessions, each beginning at 9 a.m., with quantitative sensory testing before and after anaesthesia were performed with an interval of 6 months between each session. Propofol was administered with either remifentanyl or S-ketamine or isotonic saline in a double-blind, randomized, crossover design. Each session consisted of three periods. During the first period lasting 45–60 min, only propofol was given. In the second period that lasted 60 min, the study drug (remifentanyl, S-ketamine or saline) was added, and in the final 30 min period, propofol was again given by itself. In this report, we focus on the propofol dosage that was necessary to obtain the defined endpoints during the first period.

Propofol (Diprivan 2%, Astra Zeneca, Germany) was administered as a target-controlled infusion (TCI) using the pharmacokinetic model of Marsh *et al.* [1]. An occipital EEG (O1-Cz) was recorded (Neurofile XP, IT-med, Usingen, Germany) and the power spectrum in the range of 0.5–47 Hz was extracted in 8 s EEG epochs. The following parameters were determined: median frequency (MEF), spectral edge frequency (95% quantile), burst suppression ratio and approximate entropy [2]. Anaesthesia was induced with an initial propofol target concentration of $6 \mu\text{g ml}^{-1}$, and a laryngeal mask airway was inserted when the volunteer no longer responded to verbal commands. The propofol target concentration was

* Dr Harald Ihmsen and Dr Christian Jeleazcov contributed equally to the writing of this article.

Table 1 Electroencephalography parameters and propofol requirement in the three sessions

	Group	Session 1	Session 2	Session 3
MEF baseline (Hz)	N	9.4 ± 0.7	9.3 ± 0.8	9.2 ± 0.8
	T	8.8 ± 0.4	8.6 ± 0.1	8.6 ± 0.9
MEF endpoint (Hz)	N	1.4 ± 0.5	1.3 ± 0.3	1.4 ± 0.5
	T	1.3 ± 0.2	1.3 ± 0.5	1.2 ± 0.2
Propofol target ($\mu\text{g ml}^{-1}$)	N	4.6 ± 0.7	4.6 ± 0.4	4.8 ± 0.5
	T	4.5 ± 0.1	5.1 ± 0.4	7.0 ± 0.5*.*
Propofol infusion rate ($\text{mg kg}^{-1} \text{h}^{-1}$)	N	10.1 ± 1.7	10.0 ± 1.2	10.9 ± 1.2
	T	9.8 ± 0.4	11.6 ± 1.1	16.2 ± 1.7*.*

Data are mean ± SD. Group N, volunteers without development of tolerance ($n=19$); group T, volunteers with development of tolerance ($n=3$); MEF, EEG median frequency. * $P<0.005$ session 3 vs. session 1. # $P<0.01$ session 3 vs. session 2.

then adjusted to provide deep anaesthesia with delta waves and an MEF of 1–2 Hz, but without burst suppression. This was maintained for at least 20 min before the second drug was started and kept constant for the rest of the session. Remifentanyl and S-ketamine were administered by TCI with target concentrations of 7 ng ml^{-1} and $0.1 \mu\text{g ml}^{-1}$, respectively. Baseline values of the EEG parameters were obtained by averaging the data of the last 10 min before the start. The endpoints of the EEG parameters and the corresponding propofol requirement were assessed by averaging the data of the last 10 min of the first anaesthesia period. The data of the three sessions were analysed by repeated measurement analysis of variance and the Scheffé test (STATISTICA 6.0, StatSoft, Inc., Tulsa, Oklahoma, USA). A P value lower than 0.05 was considered statistically significant.

All 22 volunteers completed the study. There were no differences between the three sessions with regard to the investigated EEG parameters (Table 1). The burst suppression ratio at the endpoint was below 3% in all sessions. Although there were no differences in propofol requirement between the sessions in 19 participants (group N), there were three obvious outliers in whom the required propofol target concentrations and infusion rates were significantly higher in the third anaesthesia session (group T, all women, 28–31 years, 52–58 kg, Table 1). The calculated propofol effect site concentrations at placement of the laryngeal mask also increased in these participants (5.4 ± 0.6 , 5.2 ± 0.2 and $6.3 \pm 1.4 \mu\text{g ml}^{-1}$ in sessions 1, 2 and 3, respectively), but not in group N (5.2 ± 0.3 , 5.2 ± 0.4 and $5.1 \pm 0.3 \mu\text{g ml}^{-1}$ in sessions 1, 2 and 3, respectively). In group N, the propofol target concentration could be reduced after the laryngeal mask airway had been inserted, on average from 6.0 to $4.5 \mu\text{g ml}^{-1}$. In group T, the target concentration had to be increased in the last session; in two participants from 6.0 to 6.5 and $7.0 \mu\text{g ml}^{-1}$, respectively, and in one volunteer, the target concentration was only slightly reduced from $8.0 \mu\text{g ml}^{-1}$ at placement of the laryngeal mask to $7.5 \mu\text{g ml}^{-1}$ in the further course.

The sequences of the study drugs in group T were ketamine–saline–remifentanyl in two participants and

ketamine–remifentanyl–saline in one. Increases in the propofol concentrations required to obtain the same EEG effect can indicate tolerance development. As none of the investigated EEG parameters differed between the sessions, we conclude that the achieved hypnotic effects did not differ either. Although the targeted blood concentrations were rather high, a ceiling effect seems unlikely, as complete EEG suppression was not induced. As the propofol target at the endpoint was kept constant during the preceding 20 min, one may also presume a steady state between effect site and plasma concentration at the endpoint.

An important limitation of the present finding is the lack of measured propofol concentrations, so that we could not differentiate altered pharmacokinetics (metabolic tolerance) from altered pharmacodynamics (receptor-site tolerance). There are only a few studies [3–5] of propofol tolerance in humans, mainly after long-term infusion in intensive care patients. For repeated propofol administration at 5-day intervals for electroconvulsive therapy, Cohen *et al.* [6] found that propofol requirements increased by more than 100% in 13 of their 30 patients. Although both alterations of propofol metabolism as well as adaptation at the level of the receptor is conceivable during long-term infusion, the present finding of an apparent development of tolerance after 6-month intervals is surprising.

One could argue that ketamine might have caused long-lasting pharmacodynamic changes. However, even though all three participants with increasing propofol requirements had received ketamine in their first session, there was no increase in the second session. Circadian effects can be ruled out, as all sessions began at the same time. A psychological effect in the sense that the participants behaved differently in the third session because it was the last one cannot be ruled out. The fact that only female participants experienced the increase in propofol requirements raises the question of a possible sex effect. Unfortunately, we did not note the menstrual cycle at the time of each session.

In conclusion, we observed an increase in propofol requirement to achieve the same EEG effect after repeated administration. Further research would be needed to investigate whether this was only incidental or whether true development of tolerance to propofol can occur after repeated administration and what are the underlying mechanisms.

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Intraocular pressure measurement in children under general anaesthesia with sevoflurane

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Editor,

Diagnosis and postoperative management of paediatric glaucoma is based on intraocular pressure (IOP) measurement in children. Few studies calculated normal IOP in children and the clinical conditions and devices used differ between each other. Data collected under general anaesthesia are variable because of the effect of the particular drugs, tracheal intubation and depth of anaesthesia [1,2]. Determinations made on alert noncooperative children overestimate IOP due to rises in central venous pressure, extraocular muscle contractions and changes in choroidal blood volume [3]. A general anaesthetic standard that allows us repeated determinations of IOP on a quiet child could be useful. That protocol should be safe and reliable, using drugs with no or little effect on IOP or, in some cases, with a known effect on IOP. Domínguez *et al.* [4] measured IOP in children under general anaesthesia, concluding that halothane used in an O₂/N₂O mixture (40/60) provides reliable values of IOP in healthy children. Nowadays halothane is no longer used and it has been replaced by sevoflurane, which is a potent and low-irritant volatile agent that can be used for anaesthetic induction and maintenance in children. It produces a decrease in IOP values that oscillate between 20 and 30%, estimated in adults [5,6]. The mechanisms of these changes remain unclear, but the most accepted are the central depressive effect of sevoflurane, and its effects on extraocular muscle relaxation and aqueous humour outflow.

The aim of this study was to develop a sevoflurane-based anaesthetic method to allow reliable IOP measurement

in children and to determine the dependence of bispectral index (BIS) and sevoflurane end-tidal values on IOP.

We undertook a prospective, descriptive observational study in 30 ASA I children scheduled for botulinum toxin injection for strabismus under general anaesthesia. Those with ocular or systemic disorders were excluded from the study. The local ethics committee assessed the study and informed consent was obtained from parents.

Induction was by facemask with 8% inspired sevoflurane in 100% oxygen until the BIS was at or below 50 and maintenance was with sevoflurane in an N₂O/O₂ mixture (40/60) titrated to keep the BIS at 50 or less. Patients did not receive any opioids or premedication. Tracheal intubation and the use of a laryngeal mask were avoided to prevent IOP artefacts, so airway management was performed by face mask and spontaneous/assisted ventilation, keeping the end-tidal CO₂ at 30–35 mmHg. Monitoring included heart rate, noninvasive blood pressure, oxygen saturation (Datex-Ohmeda, cardiocap/5), end-tidal CO₂ (EtCO₂), end-tidal sevoflurane (EtSevo) (Ohmeda, 5250 REM) and BIS (BIS XP, Aspect Medical). Haemodynamic and respiratory variables were recorded every minute while taking IOP measurements.

After anaesthetic induction, IOP was measured once and botulinum toxin was injected. Afterwards, sevoflurane was stopped and the IOP was measured at 1 min intervals until arousal. Local anaesthesia (lidocaine 4%) and sodium fluorescein (0.25%) dye were instilled. A handheld Perkins applanation tonometer (Clement-Clarke) was used and the same ophthalmologist took all readings.

The results of this study were analysed using Microsoft Excel 8 and Windows EPI.INFO 2002. Statistical tests included Student's *t*-test and χ^2 for comparative analysis and linear regression for correlation analysis.

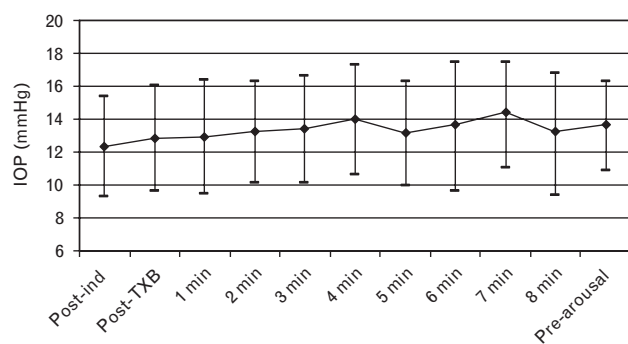
The IOP was measured in 30 children (60 eyes) aged between 3 and 10 years (5.5 ± 2.6 years). There were no significant changes in haemodynamic and respiratory parameters during sevoflurane anaesthesia (Table 1) and no complications were associated with it. Mean duration of anaesthesia was 16.1 ± 4.2 min. Induction, defined as the time to achieve BIS values at or below 50, was 3.7 ± 1.6 min. At the time of the first measurement of the IOP (postinduction data), BIS mean values were 418 ± 6 . Arousal was defined as impossibility of measuring IOP. The mean time from sevoflurane discontinuation until arousal was 7.2 ± 2 min.

The IOP was determined at a median of eight points (range 4–11) in each eye (Fig. 1). All of them were in the normal range. No significant differences were observed between each point, but there was a significant increase

Table 1 Haemodynamic and respiratory parameters at each time point of intraocular pressure measurement

	MAP (mmHg)	HR (beats/min)	SpO ₂ (%)	EtCO ₂ (mmHg)
Postinduction (n=30)	74 ± 10	101 ± 14	98 ± 2	30 ± 2
Post-TXB (n=30)	73 ± 9	95 ± 16	97 ± 2	29 ± 3
1 min (n=30)	71 ± 7	92 ± 17	98 ± 2	30 ± 7
2 min (n=28)	72 ± 8	93 ± 17	97 ± 3	31 ± 3
3 min (n=26)	74 ± 8	97 ± 18	98 ± 1	31 ± 3
4 min (n=25)	72 ± 7	97 ± 14	98 ± 1	31 ± 3
5 min (n=18)	70 ± 7	94 ± 16	98 ± 1	31 ± 2
6 min (n=11)	69 ± 4	90 ± 18	97 ± 3	29 ± 3
7 min (n=10)	72 ± 3	91 ± 18	98 ± 2	34 ± 4
8 min (n=8)	79 ± 13	97 ± 13	97 ± 2	35 ± 6
Preadrousal (n=30)	78 ± 11	98 ± 20	97 ± 3	37 ± 7

Data are means ± SD. EtCO₂, end-tidal concentration of CO₂; HR, heart rate; MAP, mean arterial pressure; SpO₂, oximetry; TXB, botulinum toxin; prearousal, last measure of intraocular pressure before arousal, of varying time point.

Fig. 1

Means ± SD intraocular pressure in children receiving sevoflurane general anaesthesia for botulinum toxin (TXB) injection. * $P \leq 0.05$.

in IOP just prior to arousal compared with postinduction values.

At the beginning of the measurement of the IOP, the mean EtSevo was 2.4% (range 1.7–3.4%) and concen-

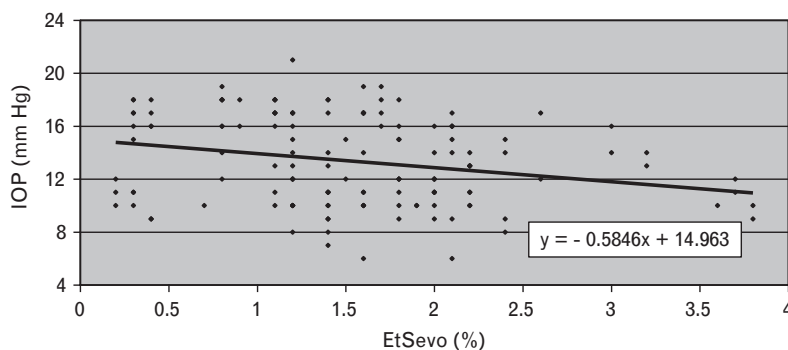
Fig. 2

Diagram of intraocular pressure – end-tidal sevoflurane data pairs. The best line of fit for the set of data pairs was a linear equation. Correlation coefficient is 0.4 ($P < 0.001$).

trations recorded during IOP measurement were between 0.3 and 3.4%. There appeared to be a steady and consistent increase in IOP with time as EtSevo decreased. A linear regression coefficient of 0.40 ($P < 0.001$) was obtained, supporting the observation that IOP increases as etSevo decreases (Fig. 2). As sevoflurane was stopped, the BIS increased progressively until arousal. A linear regression analysis revealed a statistically significant correlation between IOP and BIS (Pearson's coefficient = 0.51; $P < 0.001$).

The mean IOP values found in our study were 13.3 ± 3.1 mmHg, similar to the results reported in previous articles [1–3], taking into account differences in time between reports. Ramanjit *et al.* [1] studied 405 children aged between 0 and 12 years and observed a mean IOP of 12 ± 3.7 mmHg in cooperative nonsedated children. Although IOP determination in nonsedated cooperative children is the best way to obtain no distorted values of IOP, this is not always possible. They had a rate of success of reliable IOP values of about 50–65% in the 1–3-year age group, with greater success in infants and older children. Postoperative management of paediatric glaucoma includes frequent measurements of IOP, and an ophthalmologist needs all the IOP values to be consistent.

In our study, the time for induction was longer than the data in the literature. Kataria *et al.* [7] reported an induction time for 2% sevoflurane of 2.1 min in a multicentre study of 428 children. This difference can be explained by the different meaning of induction time. Classically, induction time has been defined as meaning time to loss of eyelash reflex, whereas we have defined it as the time to reach BIS values at or below 50. That is why we have recorded an induction time (3.7 ± 1.6 min) higher than that reported previously in the literature.

The IOP measured in our study correlated negatively and significantly with sevoflurane concentration (EtSevo),

with a correlation coefficient of 0.4. As the child eliminates sevoflurane after it has been discontinued, the IOP increases slightly and continuously, reaching the maximum value just prior to arousal, when the child is still quiet, but the EtSevo values are close to zero. Although this relation is statistically significant, variations of IOP with end-tidal concentrations between 0.5 and 3.5% are really modest, with a 10% decrease in IOP with 2% sevoflurane, calculated throughout the linear regression equation.

The depth of anaesthesia has been anticipated as an important factor in the reduction of IOP during general anaesthesia [5,6], but no previous study has correlated the IOP with any hypnosis monitor. We found the BIS index to have a statistically significant relation with IOP values (correlation coefficient of 0.51), with a stronger association with IOP than with sevoflurane concentration.

We conclude that sevoflurane inspired via a face mask in a N₂O/O₂ mixture is a well tolerated anaesthetic regimen for IOP measurement in children. Sevoflurane affects IOP values but leads to a small decrease in IOP. The best time point to measure IOP in children receiving sevoflurane is just before arousal, when the child is still quiet, sevoflurane is almost fully eliminated and the BIS is reading light anaesthesia.

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Kennedy's disease and postoperative complications

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Editor,

A case report of a 32-year-old man known to have Kennedy's disease is presented. He was admitted for an elective urological procedure and developed left-sided hemiparesis and paraesthesia postoperatively.

This man was diagnosed with Kennedy's disease when he was 28 years old after a muscle biopsy and DNA testing after a period of being symptomatic with muscle weakness, which affected mainly walking and lifting. He was also troubled with lower back pain and cramps in his limbs after strenuous activity. He became dyspnoeic when he had climbed a flight of stairs or after walking half a mile on level ground.

This man was admitted for buccal mucosal urethroplasty as a day case. He had a caudal block preoperatively and was given propofol and fentanyl for induction. He also had 25 mg of atracurium for muscle relaxation. The operation lasted 2 h, after which he was taken to the recovery room. He developed left-sided paraesthesia and weakness in the recovery room and was also complaining of headache associated with photophobia and earache. On examination, he had a focal neurological deficit (sensory and motor) on the left side of the body. On detailed questioning, he admitted to these episodes previously when a general anaesthetic was given, but by far this was the worst episode. He had a computed tomography (CT) scan of the head the next day, which did not reveal any abnormality. After a couple of days, his clinical condition improved and he was discharged home.

Discussion

Kennedy's disease is an X-linked recessive genetic progressive neuromuscular disease that affects men. The age of onset is usually adolescence. It presents as a progressive weakness and atrophy of facial, bulbar and proximal muscles of the peripheries [1]. Paresis is usually seen in most of the patients with Kennedy's disease and could be asymmetric [2]. Sensory impairment in Kennedy's disease, previously thought to be minimal or nonexistent, is present in nearly half of the patients presenting with this disease and could be asymmetrical in distribution as well [2].

The problem areas associated with anaesthetic management of patients with Kennedy's disease are not fully clarified but broadly fall into two main groups [3]. The use of general anaesthesia can lead to loss of swallowing and airway reflexes, which could worsen the scenario for patients with bulbar involvement [3]. Another area of complications could be consequent postoperative muscle weakness and a compromised baseline pulmonary function [4]. This can exacerbate the patient's neurological symptoms asymmetrically as a result of the stress from the operation, as probably happened in our case.

There is currently no evidence of how to best administer anaesthesia in a patient with Kennedy's disease. The popular opinion is that muscle relaxants should be avoided if possible and patients with bulbar involvement who are already at risk of aspiration should have a cuffed endotracheal tube *in situ* while having a general anaesthetic. Use of neuraxial block or regional anaesthesia is advocated in case reports but, nonetheless, the number of reported cases is too small to exclude the possibility of long-term neurological complications after regional anaesthesia [5]. Also, one has to be alert to the possibility of asymmetrical focal neurological deficit. It would also be better if patients were told beforehand to avoid severe anxiety.

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