Use of epidural blockade in a patient with hip fracture and aortic stenosis

EDITOR:

Patients with hip fracture are often frail and elderly, and co-morbidities are frequently present. The following case describes perioperative use of continuous epidural blockade in a patient with hip fracture and a severe aortic stenosis. This has not been reported before. There is no evidence favouring general anaesthesia to neuroaxial blockade in patients with aortic stenosis. Several case reports have shown that the use of epidural blockade for Caesarean section, labour and delivery in patients with aortic stenosis is safe [1–3]. A recently published article discussed the possibilities for using neuroaxial blockade in patients with aortic stenosis [4].

Case report

An 80-yr-old female presented for surgery of a left-sided intracapsular hip fracture. She had a past medical history of stroke and 2 months earlier, cardiac arrest. The patient denied angina and dyspnoea. Her medications included citalopram, bendroflumethiazide with potassium chloride, oxazepam, paracetamol, simvastatin and ezetimibe. The physical examination revealed a Grade III–IV systolic murmur over the aorta with radiation to the neck. Electrocardiogram (ECG) showed sinus rhythm and a right bundle branch block. Echocardiography showed aortic stenosis and mitral insufficiency Grade I. The gradient across the aortic valve was estimated to 88–90 mmHg and the ejection fraction more than 60%. Preoperative blood tests were normal.

On arrival in the emergency room a fascia iliaca compartment block was administered using 40 mL of bupivacaine 2.5 mg mL\(^{-1}\) with epinephrine 5 µg mL\(^{-1}\) as initial pain management. A lumbar epidural catheter was placed in the L2–L3 intervertebral space after 5 h and 40 min. Correct placement of the epidural catheter was tested with 3 mL of lidocaine 20 mg mL\(^{-1}\) and epinephrine 5 µg mL\(^{-1}\). This was followed by injection of 5 mL bupivacaine 2.5 mg mL\(^{-1}\) supplemented with another 5 mL bupivacaine 2.5 mg mL\(^{-1}\) after 45 min resulting in subjective analgesia and a T10 sensory level. Subsequently, a continuous infusion with 4 mL h\(^{-1}\) of bupivacaine 0.125 mg mL\(^{-1}\) and morphine 50 µg mL\(^{-1}\) was started. Prior to the neural blockade a total of saline 0.18%/glucose 5% 1000 mL was given intravenously (i.v.), followed by saline 0.9% 500 mL. Blood pressure (BP) fell from 160/90 to 120/70 and then remained stable. Heart rate (HR) was 70–75 bpm throughout the procedure.

Before surgery the following day the patient’s BP was 120/50 with HR of 65 bpm. Supplementary oxygen (O\(_2\)) therapy was administered via nasal cannulae and routine monitoring was applied including three lead ECG, pulse oximetry and non-invasive BP. The continuous epidural infusion was paused and 25 mg ephedrine was administered intramuscularly (i.m.) and 15 mg of bupivacaine with 1 mg morphine was injected into the epidural catheter. The initial bupivacaine dose of 15 mg was repeated after 35 min resulting in a T10 sensory level and surgery commenced. During surgery saline 0.9% 1000 mL and 500 mL hetastarch 130/0.4 was administered i.v. to maintain preload. Intraoperatively a supplemental bupivacaine dose of 15 mg was repeated 55 and 95 min after the initial dose. BP and HR remained unchanged during surgery. Blood loss was estimated at 800 mL.

The recovery period was uneventful. Blood loss was replaced with 2 × 300 mL erythrocyte suspensions. As soon as the patient was able to perform normal knee flexion of the unaffected leg, the continuous epidural infusion was resumed and the patient was discharged after 2 h. Postoperative analgesia was maintained for...
Correspondence

4 days using the epidural infusion with bupivacaine 0.125 mg mL\(^{-1}\) and morphine 50 µg mL\(^{-1}\). The patient recovered without sequelae and was discharged home after 14 days of rehabilitation. Six months after discharge the patient was alive and well.

Discussion

There are no evidence-based recommendations for the best choice of anaesthesia and postoperative analgesia in the patient with aortic stenosis. At Hvidovre University Hospital perioperative continuous epidural blockade is used as a part of a multimodal intervention in a fast track regimen in patients with hip fracture. Neuraxial blockade potentially reduces mortality and other serious complications in lower extremity surgery [5]. Preoperative administration of epidural analgesia compared to opioid analgesia provides a reduction in cardiac events and pain in patients with hip fracture and cardiac co-morbidities [6]. Pain and the resulting tachycardia may have deleterious haemodynamic consequences in the patient with aortic stenosis as recently mentioned by McDonald [4]. There is ample evidence to show that epidural analgesia provides better postoperative analgesia compared to parental opioid analgesia [7]. Furthermore, it has been demonstrated that postoperative epidural analgesia, probably because of superior pain management, reduces the amount of cardiac ischaemia in patients with hip fracture [8]. In major knee surgery it has been demonstrated that earlier rehabilitation can be achieved by using epidural blockade in contrast to i.v. patient controlled morphine [9]. This may also be possible in patients with hip fracture, and therefore requires further evaluation.

When anaesthetizing a patient with aortic stenosis, the haemodynamic goals include avoiding sudden and profound decreases in systemic vascular resistance, maintaining contractility and sinus rhythm and avoiding hypovolaemia and tachycardia [4].

 Epidural blockade facilitate a gradual unet of anaesthesia and sympathetic block, and therefore a sudden and profound decrease in systemic vascular resistance is avoided. With incremental doses of local anaesthetics, an even higher degree of control is attained. Epidural anaesthesia does not affect cardiac contractility and with volume loading, preload can be maintained.

 Epidural anaesthesia provides several evidence-based advantages in the patient with hip fracture, which is why we chose it for our patient. It must be emphasized that the choice of anaesthesia must be evaluated in each case, especially in patients with aortic stenosis, based on knowledge of pathophysiology and haemodynamic goals.

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References

Brachial plexus block with ropivacaine and bupivacaine for the formation of arteriovenous fistula in patients with end-stage renal failure

EDITOR:

Patients with end-stage renal failure scheduled for surgical creation of an arteriovenous fistula for haemodialysis are at risk of developing serious cardiovascular postanaesthetic complications [1]. Brachial plexus block has been shown to improve blood flow through the vascular access and seems to be a suitable technique for this operation with minimal alteration in systemic homoeostasis [1]. Ropivacaine is a long-lasting amide local anaesthetic. Its efficacy may be compared to bupivacaine but cardiovascular and central nervous system side-effects occur less frequently [2]. The onset time, duration of action and success rate of peripheral nerve blockade are commonly used as efficacy parameters when different local anaesthetics are compared. Similarly the stability of haemodynamic and respiratory parameters during anaesthesia is widely considered to be an indicator of safety of anaesthesia. The aim of this prospective study was to compare supraclavicular brachial plexus block with 0.75% ropivacaine and 0.5% bupivacaine for the creation of vascular access in patients with end-stage renal failure.

With Ethics Committee approval we studied 50 ASA II–III patients scheduled for creation of an arteriovenous fistula for haemodialysis. Patients with coagulopathy, local skin infection and allergy to local anaesthetics were excluded. The patients were randomly allocated into two equal groups. In Group 1 (14 males and 11 females, mean age 58 ± 15 yr) a supraclavicular brachial plexus block was performed using 30 mL of plain 0.5% bupivacaine and in Group 2 (15 males and 10 females, mean age 55 ± 14 yr) the same block was performed using 30 mL of plain 0.75% ropivacaine. Nerve blockade was placed by means of a nerve stimulator (Stimuplex Dig RC, B.Braun, Germany). The onset of sensory and motor block was measured as the time from the completion of the injection of local anaesthetic to the achievement of surgical block. The duration time of sensory block was achieved in the majority of patients. There were four failed blocks in the study. Two patients in Group 1 and two patients in Group 2 required supplemental analgesia and were excluded from the statistical analysis. The onset time of sensory blockade was 22.4 ± 7.3 min in Group 1 and 19.7 ± 5.9 min in Group 2. The duration time of sensory block was 8.8 ± 3.2 h in Group 1 and 7.5 ± 2.6 h in Group 2. The time required to achieve motor blockade was 33.7 ± 10.1 min in Group 1 and 34.1 ± 12.6 min in Group 2. The only difference was found in the duration of motor block; in Group 1 it was 9.5 ± 3.2 h and in Group 2 7.6 ± 3.1 h (P < 0.05) (Fig. 1). The mean values of maximal and minimal BP, HR, oxygen saturation and respiratory rate are shown in Table 1. The minimal value of DBP in Group 2 was significantly higher than in Group 1 (Fig. 2). The quality of anaesthesia in both groups in all cases was classified by the surgeons as ‘very good’. Patients’ opinions were found to be similar. In Group 1, the quality of anaesthesia was described as ‘very good’ in 19 patients and ‘good’ in 4 patients compared to 17 and 6 patients, respectively.
respectively in Group 2 ($P > 0.05$). No side-effects were observed.

Bupivacaine remains the most commonly used long-acting local anaesthetic. Ropivacaine with its fast onset, long duration and minimal toxicity profile offers an alternative to bupivacaine for long-acting nerve block. The results of our study suggest that 0.5% bupivacaine and 0.75% ropivacaine both provide a similar onset time of sensory and motor blockade. The duration of sensory blockade was also comparable between groups but the recovery time of motor blockade was significantly quicker when ropivacaine was used. Klein and colleagues compared the activity of 0.75% ropivacaine, 0.5% ropivacaine and 0.5% bupivacaine for brachial plexus block and found no difference in their clinical effect. The authors claimed that 0.75% ropivacaine did not cause a faster onset of sensory blockade or better analgesia as they had expected [3]. McGlade and colleagues compared brachial plexus block with the use of 0.5% plain solutions of ropivacaine and bupivacaine. They noted that the quality of anaesthesia was similar, however the motor blockade lasted statistically longer when bupivacaine was used [4]. Raeder and colleagues showed that 0.75% ropivacaine used for axillary block resulted in better anaesthesia when compared with the same volume of 0.5% bupivacaine. However the onset and the duration of the blockade were similar in both groups [5]. We observed, in both groups of patients, good haemodynamic and respiratory stability. Higher values of minimal DBP in the group of patients where ropivacaine was administered may account for local vasoconstrictive activity of the anaesthetic [2]. No adverse effects requiring intervention were noted. Pere and colleagues compared the pharmacokinetic profile of ropivacaine for brachial plexus block in healthy patients with patients suffering from uraemia and showed that even high doses of ropivacaine were well tolerated by patients with renal failure [6].

We conclude that there is no clinically important difference between using 0.75% ropivacaine and 0.5% bupivacaine for supraclavicular brachial plexus block when considering such efficacy parameters as the onset and the duration time of nerve blockade and the quality of analgesia. The minimal cardiovascular toxicity of ropivacaine may offer an advantage in patients with end-stage renal failure.

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Interference of patent blue V dye with pulse oximetry and co-oximetry

EDITOR:
A 17-yr-old female was anaesthetized for removal of a cervical carcinoma using sevoflurane, sufentanil and rocuronium with monitoring consisting of electrocardiogram, pulse oximetry, end-tidal CO₂ (PETCO₂) and invasive arterial pressure (AS3® Datex, Helsinki, Finland). A blood-gas analysis (Table 1) performed at the beginning of the operation was normal (Radiometer® ABL 625, Copenhagen, Denmark). During the operation, patent blue V dye (Guerbet BP 50400, Roissy, France), 100 mg (in 4 mL), was injected into the tumour for sentinel node detection. Shortly after the injection, the pulse oximetry readings decreased from 99 to 90% and afterwards to 85%, although arterial pressure, heart rate, PETCO₂ and airway pressure were unchanged. Increasing the fraction of inspired oxygen (FiO₂) to 1.0 did not improve the pulse oximetry readings. Blood-gas analysis revealed that P O₂, pH and lactate concentration were within the normal range, but that arterial saturation was only 82% and methaemoglobin was 12% (Table 1). Methaemoglobin analysis was then repeated in the main laboratory using a multifrequency co-oximeter (LS 500®, Lange, Berlin, Germany). This analyser also detected methaemoglobinemia, albeit at the lower level of 3%. Since the patient showed no clinical signs of hypoxaemia and because lung ventilation, haemodynamic and blood-gas status, and lactate concentration were all normal, the operation was continued. Blood-gas analysis performed 120 min after the patent blue V dye injection still showed signs of interference (Table 1). Emergence from anaesthesia and the postoperative period were uneventful. Skin and urine discoloration occurred and persisted for 24–36 h.

We found that the injection of patent blue V dye, used intraoperatively for sentinel node identification, interfered with pulse oximetry and co-oximetry during anaesthesia. Of interest was the high level of methaemoglobinaemia measured with co-oximetry. Dye injection as a means of facilitating sentinel node identification has been used since the early 1990s. The method is especially useful for identification of sentinel nodes for malignant melanoma and breast carcinoma, but it is currently also used to identify

<table>
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<th>Table 1. Arterial blood-gas analysis during the operation.</th>
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<td><strong>Before dye injection</strong></td>
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<td>F O₂ (mmol L⁻¹)</td>
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<tr>
<td>pH</td>
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<td>P₆CO₂ (kPa)</td>
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F O₂: inspiratory oxygen concentration; P₆CO₂: partial pressure of arterial carbon dioxide; P O₂: partial pressure of arterial oxygen; O₂Hb: oxygenated haemoglobin as a fraction of total haemoglobin; S O₂: oxygenated haemoglobin as a fraction oxygenated and reduced haemoglobin; COHb: carboxyhaemoglobin as a fraction of total haemoglobin; MetHb: methaemoglobin as a fraction of total haemoglobin; RHB: deoxygenuated haemoglobin as a fraction of total haemoglobin.
other carcinomas. Any dye used during the procedure can potentially interfere with pulse oximetry devices which use two wavelengths (660 and 940 nm) to differentiate between oxygenated and deoxygenated haemoglobin. Dyes that absorb light near either wavelength will potentially interfere with pulse oximetry [1]. Patent blue V dye has its maximum absorbance at 640 nm and its presence in blood leads to increased absorbance of the 660 nm light emitted by the pulse oximeter. This increased absorbance of the 660 nm light leads to falsely low pulse oximetry readings [2].

In our patient, patent blue V dye also led to an increase in methaemoglobin – identified by co-oximetry, which detected methaemoglobin concentrations >12%. Only a multifrequency analysis of oxygenated blood showed this to be a falsely high amount, but even with the multifrequency analyser, the concentration of methaemoglobin was still reported as 3%.

Methaemoglobin is the result of oxidation of the iron atom in haemoglobin – an increase in methaemoglobin concentration during an anaesthetic procedure is usually a consequence of oxidation of the iron atom through drugs such as prilocaine [3]. Methaemoglobin is measured with a co-oximeter at wavelengths near 655 nm – the maximum absorbance of methaemoglobin [4]. Since the peak absorbance of patent blue dye is at 640 nm, it is not surprising the co-oximeter falsely detected increased amounts of ‘methaemoglobin’ in blood. Larsen and colleagues found that patent blue V dye could lead to an overestimation of methaemoglobin by 56% [5]. However, whether any portion of the increase in detected methaemoglobin can be attributed to a true methaemoglobin increase cannot be discerned from laboratory analysis: the proximity of peak absorbance between patent blue V dye and methaemoglobin interferes with most analysers. A recent Editorial in this journal stressed that the anaesthesiologist should be aware of this dye and its interference with pulse oximetry [6], and our case report proves that point.

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Recurrent asystole during electrocauterization: an uncommon hazard in common situations

EDITOR:
Electrosurgical units (ESU) are very safe. The typical ESU for general surgery is monopolar. The active handpiece is this single pole. The circuit is completed by a dispersive electrode placed on a well-insulated part of the patient. It is at the tip of the handpiece only that the current density is great enough to cause tissue damage. The voltages applied run from thousands to tens of thousands of volts. The power settings vary from tens to hundreds of watts [1,2]. The response to an electrical current flow through the body depends on the amplitude and frequency of the current. Small currents over large areas have less impact than the same current over a smaller area. The myocardium is most sensitive to 30–100 Hz electricity, so electrical power generation at 50 Hz or even 60 Hz is ideal for inducing fibrillation. Higher frequencies (i.e. surgical diathermy) and alternating current, which does not pass through the heart, do
not cause fibrillation but rather heat up and burn the muscle. If direct current or high-frequency alternating current passes through the body, heating effects and ultimately burns will occur [1]. It is this effect that is intentionally created when electro surgical generators are used to cut tissue and coagulate fluids. If low-frequency alternating current is applied to the body, muscular polarization and depolarization take place that can ultimately create a ‘circus movement’ in the heart muscle, resulting in fibrillation and death [3].

In the operating room, electrocution hazards are described as macroshock and microshock [2]. Macroshock refers to the application of large voltage or currents to the tissue. Microshock refers to the application of low-voltage and low-frequency current directly to the heart [2]. Microshock electrocution can cause ventricular fibrillation with voltages as low as 0.05 V.

We report a case of electrical injury during electrocautery leading to recurrent spontaneously reversible asystole.

A 60-yr-old male was scheduled for a left pneumonectomy. Preoperative physical examination and preoperative 12-lead electrocardiograph (ECG) were normal. A titanium central venous access (port-ha-cath, Celsite-ST 301, Braun, USA) was implanted 2 weeks before lung surgery to facilitate preoperative intravenous (i.v.) chemotherapy. Chest X-ray showed the reservoir under the right clavicle and the extremity of the catheter located at the junction of the superior vena cava and the right atrium.

Monitoring included an ECG (leads II, VR and V5), pulse oximeter, capnograph and invasive blood pressure (BP) monitor (AS3, Datex Cie, Helsinki, Finland). Induction of anaesthesia and tracheal intubation using a double-lumen tube were uneventful, maintenance of anaesthesia was with isoflurane and i.v. sufentanil.

The patient developed brief asystole during intercostal muscular dissection, but the cardiac rhythm returned to normal after a few seconds. However the asystole resumed a few minutes later and we realized that it was concomitant with the use of the ESU (Lamidey, intersurgical 4000, France). We asked the surgeon to use the electrocautery once more, and the sequence of the accident was as follows: the surgeon commanded the electrosurgery unit in the coagulation mode using a soft pedal, asystole occurred, then he applied the tip of the electrosurgical knife to the tissue inside the thorax and electrical artefact appeared on the ECG. The third event was registered on the monitor (Fig. 1). Trace A shows the recorded ECG (standard lead II) showing a transient period of asystole due to sinus arrest followed by the artefact due to electrocautery, Trace B the concomitant recorded radial artery pressure which was absent during the period of asystole, confirming the absence of perfusion.

We stopped surgery and examined the equipment for malfunction. The electrocauterization dispersive plate appeared normal and correctly placed. However, the plate and the ESU were changed for further technical verification and the surgeon was asked to use bipolar electrocautery. Surgery was resumed uneventfully. The patient’s electrocardiogram was monitored overnight and repeated electrocardiograms were normal. Creatinine phosphokinase and troponin Ic levels were 557 U/L and 0.42 µg L⁻¹, respectively at the end of surgery; troponin Ic levels were 0.62 µg L⁻¹ 5 h later and 0.05 µg L⁻¹ the following day (institution reference range for normal troponin level is <0.8 µg L⁻¹).

Technical investigation was conducted; electric isolation of the operating room, the electrocautery unit and electrical dispersive pad attached to the patient were tested. It was concluded that the electrical environment of the room was operating correctly and within accepted guidelines. The electrocautery unit had been tested 5 months before and was normal (i.e. alarms, level of high-frequency currents and level of leak current were tested and normal).

The link between the asystole and the use of the ESU is not in doubt because of the reproducibility of the accident. Asystole occurred three times and the reiteration of the phenomenon was exactly the same each time: command of the ESU by the soft pedal, asystole then contact of the tip of the electrosurgical knife and electrical artefact on the ECG.

The mechanism involved in the case report remains putative either low-voltage and low-frequency leakage current (i.e. microshock) or low-voltage radiofrequency leakage current (i.e. radio-frequency burn) [1,2].

The mechanism of microshock is complex, due to direct leakage currents flowing through defects in active electrode insulation or capacitive stray currents originating from the shaft of the active electrode. If electrical contact is made internally, especially on or close to the heart, very low currents, as low as 10 mA, may initiate dysrhythmia. The resistance of skin contact is eliminated and the current density at the interface between the contact and the heart is very high. Normally skin resistance is quite high and this
is a safety barrier to electrocution. In thoracic surgery, this skin barrier is lost and microshock may occur, but the risk is much more common when conductive saline is used as the fluid in a cardiac catheter, a pulmonary artery catheter or a central venous pressure (CVP) catheter [3]. Ventricular dysrhythmias due to electrical microshock have been recognized in several patients undergoing cardiac catheterization or connected to electronic equipment instruments [3]. The role of the central venous line as a vector of leakage current is supported by experimental study and clinical observations. Monies-Chass and colleagues [4] showed on dogs catheterized for the measurement of CVP, with the catheter advanced into the right ventricle, that currents less than 500 mA caused interference with heart rhythm. As ventricular fibrillation in human hearts is alleged to require a minimum of 50 mA, the accepted guidelines sets 10 mA as the maximum permissible leakage current allowed through electrodes or catheters that contact the heart. In this case we report, the injury was not ventricular fibrillation but asystole, collapse and cardio-circulatory arrest which were spontaneously reversible. Nevertheless, the link between 60 Hz intracardiac leakage current and cardiovascular collapse have been demonstrated by Swerdlow and colleagues [5]. In patients with intracardiac electrodes, stimulation by silent alternating current below the ventricular fibrillation threshold, which is neither felt nor visible on the ECG presents as hypotensive ventricular tachycardia. But as far as we know, microshock induced ventricular fibrillation and not asystole as we report.

Other hypothetical mechanisms include electrical burns caused by ESU leakage current, stray current resulting from insulation failure, direct coupling of the ESU to other surgical instruments or capacitive coupling of the ESU signal due to the nature of radio-frequency transmission [6]. The radio-frequency leakage current hypothesis is sustained by Schlapfer and colleagues [7] describe a case of asystole induced by a low-voltage radio-frequency application to the patient argues strongly for radio-frequency induced current entering the heart via the antenna that was present in the patient – the titanium central line. Other causes of transient asystole during thoracic surgery may be discussed, such as a sick sinus syndrome induced by anaesthesia medications but these were very improbable, because of the reproducibility of the event.

In conclusion, we describe the case of a patient who experienced an electric accident during thoracic surgery, which induced reversible cardio-circulatory arrest. We assume that a low-voltage leakage current was conducted via the central venous line to vagal afferent fibres located at the junction of the vena cava and the atrium. This resulted in powerful electric stimulation and to vagal overactivity, and finally to transient asystole. Patients with an indwelling catheter are susceptible to microshock electrocution and are designated as electrically vulnerable patients.

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Minimum inspired oxygen concentration alarm: do we go too low?

EDITOR:
The use of anaesthetic machine and patient monitor alarms, in addition to clinical observation, can enhance patient safety, but it is very important that these limits are accurately set.

The importance of these settings is highlighted by a critical incident which occurred recently in one of our theatres.

Case report: An 8-yr-old, American Society of Anesthesiologists (ASA) I patient was scheduled for an elective ear, nose and throat procedure. Induction was uneventful and general anaesthesia was maintained with spontaneous ventilation via a laryngeal mask. The patient’s head was draped and surgery commenced. An unobserved disconnection occurred at the junction of the circle-system breathing circuit tubing and the filter to which the gas analyser sampling line was attached (Fig. 1). The reduced movement of the reservoir bag was not initially noticed and the capnography trace continued as per normal. The inspired oxygen concentration decreased but the alarm did not sound as its setting had not been changed from the factory default of 18% for oxygen. Had it been set to above 21% it would have been activated. Fortunately the disconnection was discovered and the circuit reconnected before the patient sustained any harm.

The case prompted an audit at two hospitals in our region which demonstrated that the minimum inspired oxygen concentration alarm was changed from the factory reset by less than 10% of anaesthetists. Personal communication with a colleague from a neighbouring region revealed that two identical incidents had recently occurred in their hospital, and the same audit is at present being conducted in their hospital.

Fortunately no patient suffered any harm as a result of the disconnections but the potential for patient awareness, movement, adverse airway events and hypoxia is evident. The use of an oxygen analyser is mandatory in accordance with the checklist for anaesthetic apparatus as set out by the Association of Anaesthetists of Great Britain and Ireland and is recommended in many other countries [1]. The Association guidelines however give no advice regarding the values to which the analyser’s alarm limits should be set [2].

Oxygen concentrations are usually measured at either the fresh gas outlet or via the end-expired gas sampling line attached to, or near to, the filter. In addition to guarding against the delivery of hypoxic mixtures, the analyser (sampling end-tidal gases) can also help to detect problems with breathing circuit and ventilator integrity, and with alveolar hypoventilation [3,4].

A study by Knaack-Steinegger showed that disconnections were evident within 30 s using expired oxygen concentration alarms [5]. Disconnections are evident within a few breaths when using end-tidal gas monitoring with oxygen concentration alarms set to above 21%. Capnography alarms and the waveform are used by anaesthetists as a monitor for early
Peripheral blocks of trigeminal nerve for facial soft-tissue surgery: learning from failures

EDITOR:
Regional anaesthesia offers a suitable alternative to local infiltration anaesthesia for facial soft-tissue surgery [1,2]. Regional anaesthesia also presents several advantages over general anaesthesia including smoother recovery, less side-effects, residual analgesia into the postoperative period, earlier discharge from the recovery room and reduced costs. In addition, it avoids the risk of tracheal intubation in patients assumed to have a full stomach in emergency procedures. The ophthalmic nerve provides sensation to eyes and forehead, the maxillary nerve to mid-face and upper jaw and the mandibular nerve to tongue and lower jaw. Regional anaesthesia was carried out by blocking sensory fibres from these three major branches. One or more blocks were inserted unilaterally or bilaterally depending on the anatomical area involved in the surgical field. A supraorbital block was accomplished by palpating the supraorbital foramen, inserting a 30-mm long, 25-G needle perpendicular to the skin until bone contact without entering the canal to avoid direct nerve injury.

References

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For a supratrochlear block, a 30-mm long, 25-G needle was advanced at the eyebrow and nose root junction until bone contact. To block the nasociliary nerve or its branches, a 15-mm, 25-G needle was inserted 1 cm above the inner canthus and directed postorally/laterally keeping contact with bone. At a depth of 1.5-cm the needle should be at the anterior ethmoidal foramen. This nerve block may also block the infraorbital nerve. The infraorbital nerve block was performed by using a percutaneous approach. Depression of the infraorbital foramen was fixed with a finger while a 30-mm, 25-G needle was inserted 1 cm inferior to the foramen. The needle tip was tangentially advanced upward to the estimated location of the foramen. A percutaneous technique was used to perform a mental nerve block, a 30-mm, 25-G needle being inserted inferomedially through the skin. For each block, 3 mL of anaesthetic solution was injected after careful negative aspiration. Blocks were carried out with a mixture of equal volumes of 0.25% bupivacaine and 1% lidocaine with epinephrine 1:200 000. The nasociliary blocks were inserted without epinephrine to eliminate any risk of retinal artery spasm. Intravenous (i.v.) midazolam was used in small doses (0.03 mg kg\(^{-1}\)) just before the blocks were performed. We analysed the following variables which were systematically assessed and recorded: failure to obtain full sensory loss 15 min after injection, tolerance of the surgical procedure estimated by the surgeon on a four-point scale (excellent, good, fair or poor), patient satisfaction after surgery on a four-point scale (very satisfied, satisfied, little satisfied, dissatisfied) and side-effects.

Fifty-nine patients, 34 males and 25 females, mean age 56 yr (range 17–92 yr) had surgery under regional block during the defined period. Thirty-five patients were scheduled for elective surgery including excision of cutaneous tumour (23), cosmetic surgery (10) and surgery on the lachrymal system (2). Twenty-four patients were treated as emergencies for isolated facial lacerations. Ten patients were over 80 yr of age (17%), 11 were American Society of Anesthesiologists (ASA)-III (19%). Number and type of nerve blocks, failures and side-effects were summarized in Table 1. Depending on the location and the size of injury or lesion, the number of blocks performed on each patient was variable. Nineteen patients had 1 block, 21 patients had 2 blocks, 5 patients had 3 blocks, 6 patients had 4 blocks and 6 patients had 5–8 blocks. Onset of sensory loss occurred 2 min after injection with surgical anaesthesia ensuing within 5 min after injection for all the patients. Mean duration of surgery was 58 min (range 10–150 min). The mean duration of block before recovery of light touch sensation was not assessed. No patient received general anaesthesia but additional infiltration of local anaesthetics was necessary in 13 patients (22%) because of incomplete anaesthesia in the surgical area as judged 15 min following block completion. Only minor side-effects were noticed including diplopia or eye akinesia disappearing as the nasociliary block dissipated. Tolerance of the surgical procedure was considered as fair in 3 patients, good in 32 patients or excellent in 24 patients. Twenty-two patients were very satisfied and 29 were satisfied but 8 patients were only little satisfied because of the unpleasant feeling related to the draping over the face, pain at injection of anaesthetic or discomfort during surgery (before additional infiltration) and postural discomfort.

Our results demonstrated that many facial surgical procedures can be performed with trigeminal nerve peripheral blocks. These blocks are safe and provide good intraoperative conditions. However, the failure rate (22%) remains high for a regional technique even in regard to the total number of blocks carried out \((n = 152)\). In 4 patients, failure to obtain a full sensory loss in the surgical area with a supraorbital block may be explained by ignorance of sensory innervation. The lateral and medial aspect of the upper eyelid and brow and a triangular-shaped area bordered by the zygoma, temporal hair line and lateral orbital rim are, respectively, innervated by the lachrymal and zygomaticotemporal nerve [2]. The lachrymal nerve is a terminal branch of the ophthalmic nerve in the orbit and the zygomaticotemporal nerve is a branch of the maxillary nerve leaving the lateral orbit to emerge in the anterior temporal fossa. These nerves are not blocked by a supraorbital approach [3] and a surgical procedure in their territory requires additional local anaesthetic

<table>
<thead>
<tr>
<th>Type of blocks</th>
<th>Supraorbital</th>
<th>Supratrochlear</th>
<th>Nasociliary</th>
<th>Infraorbital</th>
<th>Mental</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blocks performed ((n))</td>
<td>31 (8 bilateral)</td>
<td>30 (10 bilateral)</td>
<td>20 (5 bilateral)</td>
<td>46 (11 bilateral)</td>
<td>25 (10 bilateral)</td>
</tr>
<tr>
<td>Failures ((n))</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Side-effects ((n))</td>
<td>Oedema: 4</td>
<td>Oedema: 3</td>
<td>Oedema: 2</td>
<td>Oedema: 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Haematoma: 2</td>
<td>Haematoma: 2</td>
<td>Haematoma: 2</td>
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<td></td>
<td>Diplopia or eye akinesia: 2</td>
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</table>

infiltration and/or a block of the zygomaticotemporal nerve at the level of the lateral canthus [2]. Similarly, two failures concerning the infraorbital nerve block were related to the lack of nasociliary nerve block for surgery involving the ala and the apex of the nose; this area being innervated by its nasal branches [4]. Finally six partial failures (46%) can be explained by ignorance of facial innervation and the direct failure rate was 12%.

The infraorbital block was associated with the highest rate of failure in our study. Nevertheless, this block is considered as technically easy to perform in other studies where the intraoral route was used [5,6]. We suggest that the higher frequency of insufficient blocks measured with the percutaneous technique was due to inappropriate placement of the needle secondary to our tangential percutaneous approach having regard to nerve distribution. When the infraorbital nerve emerges through the infraorbital foramen, it divides into four groups of branches supplying four different cutaneous areas including the skin of the lateral nose, the lower eyelid, the cheek and the upper lip. These areas matched perfectly with those where we failed to obtain a full sensory loss in our study. This observation suggests a partial blockade of the nerve. The intraoral route is considered to be more reliable than the external percutaneous approach [5,6]. However, a comparative study where bilateral infraorbital nerve blocks were performed using the intraoral technique on one side and the percutaneous technique on the other, failed to find any difference between the two approaches [5]. 12 of the 13 failed blocks in our study were performed by residents. Their reported experience was <5 with the different blocks, whereas trained anaesthetists have carried out at least 20 of each block. The exact number of each procedure required before attaining acceptable failure rates remains unknown and may have a wide inter-individual variability. However, it must be emphasized that the failure rate included only partial failure allowing surgery to be proceeded with single local anaesthetic infiltration.

Peripheral blocks of the trigeminal nerve can achieve safe and effective regional anaesthesia for facial soft-tissue surgery. Although easy to perform, the success of these techniques depends on the experience of anaesthetist and requires a detailed knowledge of facial innervation, especially with overlapping sensory areas of nose apex and orbital structures and surroundings. Regarding rate of failure and side-effects, we recommend particularly the use of supratrochlear, nasociliary and mental nerve blocks.

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References

General and subarachnoid anaesthesia in a patient with acquired C1 esterase inhibitor deficiency

EDITOR:

C1 esterase inhibitor deficiency may be either hereditary (hereditary angioneurotic oedema) or acquired in nature. It is a dominant autosomal disorder characterized by either decreased esterase inhibitor concentrations (Type I) or a faulty protein with normal levels (Type II). Acquired angioedema results from either increased C1 esterase inhibitor consumption associated with lymphoproliferative conditions or connective tissue involvement (Type I), or altered autoimmune pathways (Type II) [1,2]. Both hereditary and acquired angioedema are a major concern for the anaesthetist because patients with these
pre-existing conditions may develop relapsing massive oedema involving the airway, notably the oral cavity, pharynx and larynx [1,3]. We report the perioperative management of a patient who presented with acquired angioneurotic oedema.

A 46-yr-old female (65 kg in weight and 160 cm in height) was scheduled for intraoperative biopsy plus elective mastectomy for a nodule located at the external upper quadrant of the right breast. History revealed contact eczema caused by nickel, chromium and cobalt, and infantile poliomyelitis that had given rise to a residual paralysis of the upper right limb. In the few previous months, the patient had had several episodes of face, lip and eyelid swelling, and had been diagnosed as having acquired angioneurotic oedema (decreased C1 INH, C1q, C2 and C4 levels). She had been prescribed danazol as a prophylactic treatment.

Full blood count, coagulation, blood biochemistry and chest X-ray were normal. Electrocardiogram (ECG) showed sinus rhythm with left anterior hemi-block. Premedication consisted of 1 mg oral lorazepam the night before surgery. On the morning of surgery, two units of fresh frozen plasma were prepared and the patient was administered 200 mg of danazol orally. Preparation was made in case of a difficult intubation. ECG, non-invasive blood pressure monitor, pulse oximeter and capnograph were connected. Induction of anaesthesia was performed with propofol and the trachea intubated. Anaesthesia was maintained using oxygen (O2)/nitrous oxide (N2O), isoflurane, fentanyl and vecuronium. The patient remained haemodynamically stable throughout the procedure and there was no significant bleeding. On completion of surgery, she was awoken and extubated. However, a few minutes later, the patient developed respiratory distress, stridor, increased mucus secretion, facial and upper limb swelling and O2 saturation fell to 72%. An otorhinolaryngologist was called and 100% O2 administered by mask. Two units of fresh frozen plasma were given. Within 35 min this worrying clinical picture had subsided, the patient was responding to verbal commands and O2 saturation had returned to 99%, facial and upper limb swellings persisted. The patient was transferred to the postanaesthesia recovery unit. Four hours later, she was symptom-free and facial and upper limb swellings had subsided.

Two years after the first operation, an osteolytic lesion involving the proximal end of the right femur was found in a routine surveillance visit at the Department of Oncology of our hospital, thereby the patient was scheduled for biopsy and prophylactic screw-plate osteosynthesis. Subarachnoid anaesthesia was administered uneventfully.

Hereditary angioneurotic oedema is a rare condition (incidence 1 : 50 000–1 : 500 000). It is most often seen during childhood and adolescence and other members of the family are affected. Acquired angioneurotic oedema is probably less frequent than the hereditary type, appearing later in life and without a family history. The incidence of acquired angioneurotic oedema is unknown and it may be associated with lymphoproliferative diseases or lupus erythematosus [4].

C4 concentration screening can readily provide the differential diagnosis for forms of angioedema. Normal levels during an acute episode of angioedema rules out the diagnosis of C1 inhibitor deficiency, whereas decreased levels arouse suspicion of this enzyme deficiency. Patients with the acquired type have decreased C4, C2, C1 inhibitor and C1q levels, as well as C3 deficiency in Type II. Patients with the hereditary type may have either decreased or normal C1 inhibitor concentrations along with decreased C2 and C4 levels, and C1q levels within the normal range [1,4].

Treatment for C1 inhibitor deficiencies include androgens, antifibrinolytic drugs, fresh frozen plasma and C1 inhibitor concentrate. Prophylactic treatment is aimed at preventing airway obstruction in those patients who suffer from episodes of angioedema more frequently than once or twice a month. Favoured drugs for long-term treatment are danazol (600 mg day−1) and stanozolol (2 mg day−1) orally [1,2]. e-aminocaproic acid, tranexamic acid and aprotinin are antifibrinolytic drugs which inhibit plasminogen and plasmin activation; therefore, they are candidate drugs for prophylactic treatment of angioedema episodes. Fresh frozen plasma contains C1 inhibitor but also kinins and substrates that play a role in complement activation and may result in hepatitis virus and human immunodeficiency virus transmission. While fresh frozen plasma is at times used preoperatively as well as for treatment of acute angioedema, it is advisable to reserve it for those cases in which purified C1 inhibitor is not available, as was the case in our patient [1]. C1 inhibitor concentrate is the treatment of choice for acute angioedema episodes, resulting in partial remission of symptoms within 16–60 min and full remission within 24 h of administration. Furthermore, its protective action may last as long as 2 days. While C1 inhibitor has been used as a prophylactic treatment in patients with poor response to other therapeutic modalities, its routine usage is not because of the associated hazard of virus transmission. The common initial dose is 1000–2000 U; however, it should be borne in mind that patients with acquired angioneurotic oedema usually need higher doses because of increased C1 inhibitor consumption [2,4,5]. Preoperative diagnosis of hereditary or acquired angioedema is crucial for adequate perioperative management. Alonso and Fas have recently published a list of guidelines for perioperative management of these patients [2].

In these patients, the anaesthesiologist should be ready to manage the airway electively, when the
Airway is not compromised, urgently when mild to moderate airway oedema is present or in an emergency when airway swelling is life-threatening to the patient. Generally, oedema develops slowly, but at times it may develop within minutes with catastrophic consequences. The clinical presentation is similar in all types and includes a fullness in the mouth, dysphagia, facial tightness, hoarseness, dysphonia, stridor and laryngeal oedema or laryngospasm. Laryngoscopy for intubation may worsen laryngeal oedema, in which case emergency tracheotomy is needed [1]. The role of the laryngeal mask is not established, although it seems likely that its use may worsen airway oedema [1,2].

Premedication with anxiolytic drugs is of paramount importance in these patients because preoperative stress may trigger an acute episode of oedema. Both general and subarachnoid anaesthesia have been used [2,6,7]. The presence of angioedema should not be a consideration in choosing induction agent or muscle relaxant. Succinylcholine appears to be safe [8]. There are no articles in the literature addressing the use of volatile anaesthetic agents. Some authors advocate considering local and regional anaesthesia in patients with oedema in order to avoid orotracheal intubation [1,8].

For postoperative care, transfer to a high-dependency unit after surgery is advisable and airway oedema should be carefully monitored all the time. The decision to transfer the patient to the intensive care unit should be governed by the patient’s general health, co-morbidities, complexity of the surgical procedure performed and complications, including those resulting from airway oedema [2]. The postoperative episode of respiratory distress in our patients was likely to be due to airway oedema resulting from airway manipulation during extubation. There was a good response to fresh frozen plasma replacement therapy.

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