Blood pressure control with glyceryl trinitrate during electroconvulsive therapy in a patient with cerebral aneurysm

EDITOR:
Electroconvulsive therapy (ECT) induces abrupt changes in systemic and cerebral haemodynamics that are problematical for patients with cardiovascular or cerebrovascular complications. Although there are no reports of ECT causing rupture of a cerebral aneurysm, excessive haemodynamic changes in such patients must be avoided. Short-acting β-adrenoceptor-blocking agents, e.g. esmolol, are suitable to attenuate hyperdynamic states after ECT. However, this type of drug is sometimes inapplicable for medical or social reasons. We describe a patient with a cerebral aneurysm who was successfully treated with glyceryl trinitrate before undergoing ECT.

A 68-yr-old female diagnosed with endogenous depression and resistant to medication needed ECT. Preoperative evaluation with computed tomography revealed a cerebral aneurysm (6 mm in diameter) with a bleb in the middle section of the anterior communicating artery. Her family refused to have surgical clipping performed before the series of ECT. They understood the risks of both the procedure and cerebral aneurysm rupture, and written informed consent was obtained.

Atropine 0.01 mg kg \(^{-1}\) intramuscularly (i.m.) was given as a vagolytic premedication. Arterial blood pressure was measured continuously at the right radial artery using a tonometric BP monitor (CBM-7000\(^{20}\), Colin Co Ltd, Komaki, Japan). The tc-Doppler probe (Sonos 5500\(^{20}\), Agilent Technology, Palo Alto, CA, USA) was adjusted to detect the middle cerebral artery flow (right temporal side) using a 2 MHz ultrasonic wave. The Doppler signals were obtained at a depth of 45–55 mm from the surface and the velocity was calculated automatically by tracing the waveforms.

General anaesthesia was induced with propofol, 1 mg kg \(^{-1}\), over 15 s through an indwelling intravenous (i.v.) cannula. After consciousness had been lost, succinylcholine chloride (1 mg kg \(^{-1}\)) was administered and the lungs inflated with 100% oxygen via a facemask. Glyceryl trinitrate (0.01–0.02 mg kg \(^{-1}\)) was administered i.v. immediately after the succinylcholine to prevent excessive hypertension during the ECT. One minute later, an electrical current was applied bilaterally for 5 s at the minimal stimulus intensity, which had been determined in a first ECT trial by increasing the electrical intensity stepwise. The electroshock was delivered by a trained psychologist using an ECT stimulator (CS-1\(^{1}\); Sakai Iryo Co Ltd, Tokyo, Japan). The efficacy of electrical stimulation was determined using a tourniquet. The lungs were then gently inflated and PETCO\(_2\); at the nostrils was maintained at 4.0–4.7 kPa and S\(_{\text{PO}}\)_2 > 98%.

The patient received ECT three times a week for a total of 20 treatments. Heart rate and blood pressure were largely unchanged after the ECT stimulus. Maximum changes of the averages (average of the 20 ECT sessions) were a 13.6% increase in heart rate (preanaesthesia 109 ± 12 beats min\(^{-1}\), pre-electrical stimulus 107 ± 10 beats min\(^{-1}\), maximum after the shock 122 ± 10 beats min\(^{-1}\), respectively). The increase in mean blood pressure was 8.4% (preanaesthesia 81 ± 12 mmHg, pre-electrical stimulus 71 ± 11 mmHg, maximum after the shock 77 ± 18 mmHg, respectively), 30 s after the electrical stimulus. The mean flow velocity in the middle cerebral artery was increased to a maximum of 12.7% at 30 s after the electrical stimulus (preanaesthesia 67 ± 8 cm s\(^{-1}\), pre-electrical stimulus 65 ± 7 cm s\(^{-1}\), maximum after the shock 73 ± 10 cm s\(^{-1}\), respectively). The patient’s mental condition improved gradually, and she was discharged after the completion of the course of treatment without any physical problems.

An intracranial aneurysm is listed as a contraindication of ECT, since the abrupt haemodynamic changes during therapy may cause aneurysmal rupture. However, there are several reports describing safe ECT management of patients with a cerebral aneurysm [1,2]. In all reports, some kind of antihypertensive drug was given to prevent excessive hypertension. Esmolol is the antihypertensive agent most used since short-acting β-adrenoceptor-blocking...
agents were suitable for controlling blood pressure during ECT [1]. Salaris and colleagues reported two patients whose blood pressures were controlled with two kinds of β-adrenoceptor-blocking agents, esmolol and labetalol [2]. Another combination of β-adrenoceptor-blocking agents, i.e. nadolol and propranolol, was reported in a patient with a carotid artery aneurysm. Recently, the use of the postsynaptic α1-adrenergic antagonist urapidil was proposed as an alternative for β-adrenoceptor antagonists in ECT.

In our patient, glyceryl trinitrate was successfully used to minimize the systemic and cerebral haemodynamic changes. We did not use esmolol because it was clinically unavailable in the country where the patient was treated. Gardner and Kellner reported the use of a nitroglycerin spray to control blood pressure during ECT [3]. Viguera and colleagues reported a patient whose blood pressure was controlled by sodium nitroprusside and atenolol, and they suggested that esmolol alone might be insufficient to attenuate haemodynamic changes during ECT [4]. Several other reports have demonstrated that pretreatment with glyceryl trinitrate prevented excessive hypertension after the electrical shock in patients without cerebral aneurysm. We have also reported that glyceryl trinitrate is effective in suppressing the increase in systemic blood pressure and cerebral blood flow velocity during ECT [5]. Recent neurophysiological observations confirmed that glyceryl trinitrate decreased cerebral blood flow velocity without changes in cerebral blood flow [6]. Since the suppression of changes in cerebral haemodynamics is considered effective in the prevention of aneurysmal rupture, use of glyceryl trinitrate or other types of nitric oxide donor may be suitable for haemodynamic management during ECT in patients with a cerebral aneurysm. When the use of β-adrenoceptor-blocking agents is considered inappropriate, e.g. in patients with bronchial asthma, glyceryl trinitrate is recommended instead. The side-effects of glyceryl trinitrate include increases in intracranial pressure or tachycardia; however, we did not observe these side-effects in our patient. There was no excessive hypotension because we injected the drug 1 min before the application of the electrical stimulus. It has been suggested that the peak heart rate after the electrical stimulus is a means of quantifying the quality of seizures and can provide feedback on the intensity of future stimuli. Since the use of an antihypertensive drug modifies the effect of the electrical stimulus on heart rate (HR), the use of antihypertensive medication during ECT might be restricted to cases with complications.

We used propofol as the anaesthetic agent, although previous reports for ECT in patients with cerebral aneurysm used methohexital [1–4]. Haemodynamic changes during ECT were less when anaesthesia was induced with propofol when compared with methohexital or thiopental. Changes in cerebral blood flow velocity are reduced and shorter when propofol is used when compared with thiopental [7]. There was no significant HR change when propofol was used. An increase in mean blood pressure was only observed at 1 min after the electrical stimulus (17 ± 13%), and an increase in the middle cerebral artery blood flow velocity was observed at 0.5 and 1 min after the stimulus (maximally 78 ± 21%). Propofol might be more suitable than methohexital or thiopental when trying to minimize systemic and cerebral haemodynamic changes during ECT. To our knowledge, there has been no study that compared the haemodynamic effects of antihypertensive medication for ECT during propofol anaesthesia. However, since the use of antihypertensive medication – such as a β-adrenoceptor antagonist or glyceryl trinitrate – stabilizes haemodynamic changes during ECT with thiopental, it is possible that appropriate use of such medication during ECT under propofol anaesthesia further stabilizes haemodynamics. In the present case, the change in haemodynamics during ECT was considered to be smaller than with thiopental with glyceryl trinitrate. Psychological observations (using the Hamilton Depression Rating Scale and the Beck Depression Inventory and electroencephalographic analysis (using the postictal suppression index and integrated amplitude) demonstrate that the therapeutic efficacy of ECT when using propofol does not differ significantly from when barbiturates or methohexital are used – despite the duration of seizures being slightly shorter with propofol. The use of short-acting opioids, e.g. remifentanil, at the induction of anaesthesia may be another option when haemodynamic stability is required during ECT management.

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References
Failure of interscalene brachial plexus blockade to produce pre-emptive analgesia after shoulder surgery

EDITOR:
It has been proposed that analgesia given before a noxious stimulus prevents central sensitization and is thus more effective than the same analgesia given after the stimulus [1–3]. Several studies using local anaesthetics, opioids, non-steroidal anti-inflammatory drugs (NSAIDs) and the N-methyl-D-aspartate (NMDA) antagonist ketamine have been conducted to test the hypothesis of pre-emptive analgesia, but with contradictory results [4–6]. Although pre-emptive analgesia has been studied in association with different kinds of surgery, there appear to be no clinical studies about any pre-emptive effects of an interscalene brachial plexus block for surgery of the shoulder.

After institutional approval from our Ethics Committee, we examined the pre-emptive analgesic effect of interscalene brachial plexus block in 60 patients (ASA I–II) undergoing elective surgery of the shoulder (diagnosis: rotator cuff syndrome) in a prospective, randomized manner. According to a pilot study with 20 patients in two groups, we wanted to detect more than a 30 min difference in the interval from the beginning of the blockade to the first dose of oxycodone, accepting a one-tailed $\alpha$-error of 5% and a $\beta$-error of 20%. This power analysis yielded a sample size of 15–16 patients.

Interscalene brachial plexus blockade with lidocaine 35 mL (10 mg mL$^{-1}$) using Winnie’s method was performed 30–60 min before the start of anaesthesia (Group 1) or just after the end of the operation, but before the end of general anaesthesia (Group 2). A third group (Group 3) did not receive any local anaesthesia, thus serving as an internal sensitivity control group. A standardized general anaesthetic was given to every patient comprising thiopental, succinylcholine, alcuronium bromide and enflurane in N$_2$O/O$_2$, 70/30%.

Postoperative pain relief was facilitated using a patient-controlled analgesia (PCA) device programmed to deliver oxycodone 0.025 mg kg$^{-1}$ body weight intravenously (i.v.), lockout time 8 min, maximal dose of oxycodone 0.1 mg kg$^{-1}$ h$^{-1}$. A loading dose of oxycodone 0.06 mg kg$^{-1}$ i.v. was administered when patients first asked for pain relief in the recovery room after operation. The consumption of oxycodone during the first 6, 12, 18 and 24 h and the interval from the start of the block to the first dose of oxycodone were registered. Patients were instructed in the use of the PCA device and the visual analogue scale (VAS) before the operation, and they assessed their postoperative pain at rest using a 10 cm VAS (0 = no pain, 10 = worst pain) starting in the recovery room when the loading dose was given and at 1, 2, 3, 6, 9, 12, 15, 18, 21 and 24 h thereafter. The recovery and ward personnel were unaware to which group the patients had been allocated.

All 60 patients enrolled completed the study. The patients’ characteristics data of the three groups did not differ statistically (one-way ANOVA). There were no significant differences between the groups at any time point in the VAS scores (median (range) 3 (0–10) in Groups 1 and 2; 5 (1–10) in Group 3; Kruskal–Wallis ANOVA), or in cumulative oxycodone consumption (at 24 h: 62.3 ± 26.7 (SD) mg in Group 1; 51.0 ± 31.5 mg in Group 2; 67.1 ± 38.9 mg in Group 3, respectively). The time intervals from the end of the operation to the loading dose of oxycodone were 49 ± 44 min in Group 1, 119 ± 65 min in Group 2 and 23 ± 17 min in Group 3 ($P < 0.02$).

There are no studies available with which to compare our results using a brachial plexus block for pre-emptive analgesia, but our negative results are in accordance with the majority of studies on pre- versus postsurgical wound infiltration and epidural block.

In all such studies, the experimental design corresponded with the recommendations for studying pre-emptive analgesia [2], which means that both the pre- and postoperative groups received an identical regional procedure.

Whether the preoperative block produces pre-emptive analgesia or not was planned to be based on the consumption of oxycodone. For this reason, the exact time - when the patient asked for pain relief for the first time and received a loading dose of oxycodone - was registered. As expected, the time interval from the end of the operation to the first request of oxycodone was shorter in patients who received no blocks at all when compared with those receiving blocks before or after surgery. The 24 h period of oxycodone consumption started approximately 70 min later after the operation in the group that received the block after the operation compared with the group which received a block preoperatively. Since 24 h is a relatively long observational period in our regional anaesthesia model employing lidocaine, this should not affect the total consumption of oxycodone.

The consumption of oxycodone and pain were registered for 24 h starting from when the loading dose for pain relief was given. The VAS scores and the consumption of oxycodone were similar in every consecutive period of 6 h in all groups, which meant that the preoperative blockade did not show any pre-emptive effect for 24 h starting from when the loading dose was given. In other words, if our study had demonstrated a pre-emptive analgesic effect in the 24 h starting from when the loading dose was given, the oxycodone consumption should have been smaller in patients treated by preoperative blockade than in those treated with the postoperative block.

We used regional anaesthesia together with a standardized general anaesthesia. No opioids or other analgesics that affect central pain processing were given before or during surgery. Regional analgesia lasted to the end of surgery in the majority of Group 1 patients. However, there may have been some patients in whom the sensory blockade may have disappeared before the end of surgery. This could have caused an impaired pre-emptive analgesia effect in them.

Another explanation for the lack of a pre-emptive effect might be that a brachial plexus block could be insufficient to block all the sensitive central input. Birbaumer and colleagues found, using neuroelectric or neuromagnetic source imaging, substantial reorganization of the primary somatosensory cortex subsequent to amputation, and a strong relationship between the degree of cortical reorganization with phantom limb pain has been demonstrated. When upper limb phantom pain was treated by brachial plexus block, a very rapid elimination of cortical reorganization in the somatosensory cortex in most, but not in all, patients was detected [7].

Shoulder surgery was used as a pain model because it usually elicits moderate to severe postoperative pain. The postoperative regional plexus blockade was effective in abolishing the requirement of oxycodone for pain relief as long as it lasted. Because of the short duration of the preoperative interscalene plexus block, inflammation and hyperalgesia at the wound may have evoked neuronal sensitization in the early postoperative period, explaining the failure to produce a pre-emptive analgesic effect. Perhaps a preincisional block covering the immediate postoperative period, i.e. employing a long-acting local anaesthetic such as bupivacaine, is required to achieve a pre-emptive analgesic effect.

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References
Laryngeal mask airway severed by biting

EDITOR:
Complications with both reinforced laryngeal mask airways and reinforced endotracheal tubes relate to compression of the lumen [1–3]. We describe what we believe is the first report of severance of a non-reinforced laryngeal mask airway during emergence from general anaesthesia.

A 27-yr-old male weighing 85 kg presented for elective knee arthroscopy. He was fit and well with no previous history of anaesthesia. He smoked 10 cigarettes per day and consumed an average of 10 units of alcohol per week. General anaesthesia was induced with fentanyl and propofol, a size 4 laryngeal mask airway was inserted easily and the patient spontaneously breathed nitrous oxide and sevoflurane in oxygen through a closed circle breathing system. Intraoperatively, he was given intravenous cyclimorph 6 mg for analgesia and ondansetron 4 mg to minimize postoperative nausea and vomiting. Surgery and anaesthesia were uneventful until emergence when the patient was positioned on his left side still breathing through the laryngeal mask airway. The patient raised his right hand to remove his laryngeal mask airway and at the same time he bit down hard and completely severed the airway (Fig. 1). He was not sufficiently lucid to open his mouth to command, which would have allowed us to retrieve the retained portion of the mask from his oropharynx, and he became hypoxic (SaO₂ 77%). Fortunately, at this moment, he opened his mouth slightly and the retained portion of the mask was removed using a Magill intubating forceps. Oxygen saturation quickly returned to 100%. His teeth were not damaged in any way. The remainder of his recovery period was uneventful and the patient was discharged home later that day.

This incident raises a number of important issues. In a previously reported case of problems associated with the reinforced laryngeal mask airway – because the sterilization record could not be found – concern was expressed that the recommended number of 40 usages had been exceeded [2]. However, the sterilization record for the reinforced laryngeal mask airway used indicated that it had only been used 16 times and was therefore well within its recommended shelf life. Furthermore, it has been recommended that bite blocks be used with all laryngeal mask airways on emergence from anaesthesia [4]. This is not standard practice in our institution because we are concerned of the potential trauma to lips and teeth in spontaneously breathing patients. However, Brimacombe and colleagues recommended that suitable bite blocks be prepared by rolling gauze swabs into a cylindrical shape and securing them tightly with adhesive tape to a thickness of 2.5 cm [4]. The Guedel airway has been shown not to be a suitable alternative to a bite block [5]. Finally, the issue of the sensible time to remove the laryngeal mask airway after the end of anaesthesia is undetermined [6,7]. Further work may find an answer to the question whether the laryngeal mask airway should be removed when patients are either deeply anaesthetized or wide awake. The solution may help avoid the occurrence of this rare but potentially serious problem.

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References
Sickle cell disease in pregnancy

EDITOR:
A 33-yr-old Nigerian female of 12 weeks’ gestation was admitted to a tertiary referral centre with an acute sickle cell crisis following a 12 h flight from Africa. She was a known sickle cell sufferer (homozygous) experiencing one to two acute crises per year, and she had a history of malaria and osteomyelitis. She had one previous uncomplicated pregnancy and delivery. She did not smoke or drink, was not taking any medication and had no known drug allergies.

Investigations included a full blood count revealing anaemia (haemoglobin, Hb, 7.9 g dL\(^{-1}\); baseline 6–8 g dL\(^{-1}\)), a blood film demonstrating polychromasia, sickle cells, target cells and Howell–Jolly bodies. She was given supplemental oxygen, intravenous fluids and intramuscular morphine to alleviate the symptoms. She was commenced on folate supplements and low-dose amoxycillin because of her hyposplenism and transferred to our hospital for antenatal care. During her pregnancy, she was frequently admitted for acute sickle cell crises. At 24 weeks’ gestation, one episode – manifested as severe upper and lower limb pain – required a patient-controlled analgesia (PCA) device. She also experienced episodes of altered levels of consciousness and focal seizure activity, assumed to be a result of the sickling process affecting the cerebral vasculature.

The patient was admitted again at 31 weeks’ gestation with a 200 mL antepartum haemorrhage and generalized musculoskeletal pains, which were managed conservatively. Causes such as placenta praevia and abruption were eliminated and the patient was discharged home with a prescription for oral morphine sulphate to control the symptoms. At 34 weeks’ gestation, one episode – manifested as severe upper abdominal pain and lower limb pain – required morphine via a patient-controlled analgesia (PCA) device. She also experienced episodes of altered levels of consciousness and focal seizure activity, assumed to be a result of the sickling process affecting the cerebral vasculature.

The patient was transferred to the high dependency unit (HDU) for postoperative monitoring. On the following day, she developed a persistent tachycardia, hypotension and reduced urinary output, which were poorly responsive to intravenous fluids. Clinical examination revealed a tense, tender abdomen and her Hb was 6.7 g dL\(^{-1}\). Owing to her acute unstable haemodynamic status, emergency exploratory surgery was scheduled under general anaesthesia using a solution containing bupivacaine 12.5 mg (hyperbaric) and fentanyl 20 µg. She remained haemodynamically stable throughout surgery, which was uneventful, and no blood transfusion was required. A baby girl was delivered weighing 1.75 kg, who was transferred to the special care baby unit for observation because of low birth weight.

The patient was transferred to the HDU for postoperative monitoring. On the following day, she developed a persistent tachycardia, hypotension and reduced urinary output, which were poorly responsive to intravenous fluids. Clinical examination revealed a tense, tender abdomen and her Hb was 6.7 g dL\(^{-1}\). Owing to her acute unstable haemodynamic status, emergency exploratory surgery was scheduled under general anaesthesia with invasive arterial and venous pressure monitoring. This revealed a large haematoma above the rectus sheath and an estimated 1 L of ‘old’ oxidized blood in the pelvis. She received 4 units of red cell concentrate intraperatively and also required fresh frozen plasma because of persistent oozing from the operative site (activated partial thromboplastin time of 77 s). Postoperatively, she was returned to the HDU and was given supplemental oxygen, intravenous fluids and antibiotics, and morphine – by the PCA – was continued. She was discharged home 21 days following delivery with the mother and infant in a satisfactory condition.

The increasingly ethnic mix of our obstetric patient population presents several challenges to our practice. We can be faced with unfamiliar medical conditions related to race and area of origin that are often problematic at presentation. Sickle cell disease is one such condition. In 2001, approximately 15% of all...
women who delivered at our institution were of ethnic origin. The prepregnancy sickling status of the woman generally predicts her antenatal course. For reasons attributable to both the gravid and sickle cell states, mothers are prone to hypoxic crises, seizures, splenic sequestration, left ventricular dysfunction, pyelonephritis, cholecystitis and pregnancy-induced hypertension. A higher incidence of placenta praevia and abruptio has also been reported [1]. Before pregnancy, this patient usually experienced one to two sickle cell crises per year. However, the physiological changes of pregnancy and labour can precipitate acute sickle cell crises, so prompt aggressive treatment of crises is advisable. It is reported that 55.8% of sickle cell sufferers will have one or more vaso-occlusive episodes during their pregnancy [2]. In general, the physiological anaemia, increased oxygen consumption and the hypercoaguable state of pregnancy can worsen the baseline disease.

Achieving adequate pain relief can be problematic as many drugs are contraindicated in pregnancy. We felt it best to use a multimodal approach, combining simple analgesics with moderate doses of opioids. Morphine is the primary opioid used to treat painful crises, as meperidine is associated with the potential for fits, the interaction with other drugs and it has a negative inotropic effect [3]. Diclofenac was used cautiously during the first two trimesters. It is classified as a Category B drug in early pregnancy. Its use in the second half of pregnancy is not recommended because of potential adverse effects to the foetus. Our patient’s symptoms were severe enough to warrant use of opioids throughout pregnancy. Neonates should therefore be carefully observed for opioid withdrawal symptoms.

Operative delivery for sickle cell sufferers is higher than for healthy parturients with reported rates of up to 36% [1]. Maintenance of a warm environment is important to reduce the risk of hypothermia and shivering. A review by Frietsch and colleagues suggested that a balanced general anaesthesia technique is preferable in sickle cell sufferers [3]. However, in pregnancy, the use of regional techniques for operative delivery avoids potential airway complications and depressant effects of anaesthetic agents on the neonate. The level of the sensory block should be closely monitored as respiratory impairment can lead to hypoventilation with hypoxia and acidosis, so precipitating a sickling crisis. If our patient had delivered vaginally, similar principles to avoid sickling would apply – with adequate hydration, maintenance of a warm environment and supplemental oxygen. Epidural analgesia would provide effective analgesia and reduce circulating catecholamine concentrations that may lessen the risk of a sickling crisis. Close attention to adequate analgesia, hydration, physiotherapy and early mobilization in the postpartum period is important to minimize further complications.

Preoperative blood transfusion in sickle cell anaemia remains controversial. A study by El-Shafei and colleagues, comparing prophylactic versus restrictive blood transfusion policies in pregnant patients, found no significant differences in maternal or perinatal outcomes between either group [4]. Another study demonstrated a similar frequency of complications with aggressive transfusion versus a conservative approach and a twofold increase in transfusion-related complications in the aggressively transfused group [5]. Symptoms of anaemia in sickle cell sufferers are generally well tolerated because HbS releases oxygen more readily than HbA (HbS $P_50 = 4.13$ kPa). Our institution has a conservative blood transfusion policy for sickle cell patients. This patient tolerated a Hb of 6–8 g dL$^{-1}$ well during her acute crises. However, it was decided to transfuse 4 units of red cell concentrate at relaparotomy owing to haemodynamic instability and the presence of a coagulopathy. Our patient remained in HDU for 6 days following delivery. We were vigilant for the ‘acute chest syndrome’ that can affect up to 40% of sickle cell sufferers presenting with pleuritic chest pain, referred abdominal pain, cough, fever and hypoxia [6]. The risk of developing this condition increases by a factor of 1.7 for every 1 g dL$^{-1}$ reduction in Hb concentration [3]. In summary, this case highlights the many issues that must be considered in the management of pregnant sickle cell sufferers that we are likely to encounter with increasing frequency in our daily practice.

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References

General anaesthesia with remifentanil and cisatracurium for a superobese patient

EDITOR:
A 28-yr-old male, ASA III, was admitted for elective gastric bypass to treat for morbid obesity. He was hypertensive (140/105 mmHg) and suffered from the obesity-hypoventilation syndrome (PaCO₂ = 7.2 kPa) [1]. His body weight was 219 kg, height 176 cm (body mass index 70.7), so he could be termed superobese [2]. According to Garrow’s scale, his ideal body weight (IBW) should be 75 kg. The night before surgery, midazolam 15 mg and ranitidine 75 mg were given orally. Intravenous access was secured and metoclopramide 10 mg and ranitidine 50 mg were given intravenously (i.v.) 1 h before operation. In the operating theatre, standard anaesthetic monitoring was instituted. The patient was positioned in the reverse Trendelenburg position (Fowler’s position) and a continuous infusion of remifentanil 0.5 mg kg⁻¹ min⁻¹ (48 mL h⁻¹) was started and midazolam 3 mg was administered i.v. Anaesthesia was induced with propofol 130 mg (1 mg kg⁻¹) using an empirical formula to estimate the dosage of propofol for the morbidly obese (corrected weight = ideal weight + (0.4 × excess weight)) [3]. After loss of the eyelid reflex, cisatracurium 16 mg (0.2 mg kg⁻¹ IBW) was administered to facilitate oral endotracheal intubation 8 min from the beginning of the remifentanil infusion and another 8 mg was administered when two responses of the train-of-four (TOF) stimuli were attained. The intubation conditions were estimated on four points according to Kriegl’s scale [4]. Midazolam 7 mg was administered i.v. and further doses of 2 mg were given hourly. A mixture of 66% nitrous oxide in oxygen maintained anaesthesia. The patients’ lungs were ventilated to normocapnia (ETCO₂ = 4.7–5.3 kPa) at a tidal volume of about 1 L at a respiratory rate of 8–10 breaths min⁻¹. When a Thompson retractor was inserted 35 min after commencement of surgery, the pressure on the diaphragm and mediastinal structures produced an increase in arterial and airway pressures similar to that seen at laparoscopy in morbidly obese patients [5]. The infusion of remifentanil was increased to 0.75 µg kg⁻¹ min⁻¹ (72 mL h⁻¹). The cardiovascular variables were then stable: systolic arterial pressure 120–155 (about 140 ± 11) mmHg; mean arterial pressure 86–114 (about 101 ± 8) mmHg; heart rate 70–89 (about 81 ± 4) beats min⁻¹, and there were no further significant responses to stimuli. Thirty-five minutes before the end of surgery, two doses of morphine 5 mg, 5 min apart, were administered. At the end of the operation, atropine 1 mg, neostigmine 2 mg and ondansetron 4 mg were given. Over the next 13 min the patient regained 80% of his initial (unblocked) response to neuromuscular stimulation. The infusion of remifentanil was then stopped and the patient’s lungs ventilated with 100% oxygen. From this moment, the time the patient took to regain spontaneous respiration and respond to verbal command was 6 min, to adequate respiration 8 min and to tracheal extubation 9 min.

The infusion of remifentanil lasted 197 min. Total usage of remifentanil was 222.5 mL (of 50 µg mL⁻¹ solution (11.12 mg)), and of cisatracurium 56 mg. Surgery, anaesthesia and recovery were all uneventful. After extubation, the patient was fully awake and in a satisfactory respiratory and circulatory status. The patient was assessed in the recovery room as 14 according to the Aldrete scoring system. Postoperative pain was estimated as 25% on an analogue scale, and on a verbal scale as mild. Postoperative analgesia comprised the combination of morphine with tramadol: tramadol 100 mg i.v. every 6 h and an infusion of morphine 2 mg h⁻¹ was started immediately after the operation as soon as was judged to be safe. The patient did not complained of nausea, vomiting, dyspnoea or muscle weakness. No complications were observed. Three days later, he was fully mobilized and started on a liquid diet. On the seventh postoperative day, he was discharged from hospital.

Obesity is associated with many other conditions, some of which have important implications for the administration of anaesthesia. There is an increase in the frequency of chronic diseases such as diabetes mellitus, systemic hypertension, hypertensive heart disease, gastro-oesophageal reflux and cardiorespiratory complications, e.g. obesity-hypoventilation syndrome, obstructive sleep apnoea syndrome, pulmonary arterial hypertension, and right and left ventricular failure. Various studies have reported that the mortality rate is significantly increased for morbidly obese patients during the perioperative period. From the anaesthetic point of view, preoperative...
evaluation and optimization, intraoperative management and postoperative care represent real challenges that determine the success of the surgical procedure, the development of complications and the final outcome. Many papers describe a number of anatomical, physiological and biochemical abnormalities associated with morbid obesity. There are studies about the pharmacology of anesthetic drugs and the methods of anesthetic management of morbidly obese patients. The use of short-acting and titratable opioids and muscle relaxants is preferable [6,7]. Remifentanil and cisatracurium are useful drugs for patients with obesity and the dosage can be based on IBW [8]. However, most studies have been reported on morbidly, but not extremely, obese patients. Approximately 5% of morbidly obese patients, including the patient described here, develop obesity hypoventilation syndrome (OHS) (Pickwickian syndrome), consisting of episodic somnolence, sleep apnoea and loss of response to carbon dioxide [1]. We are aware that the risk of prolonged muscular blockade or opioid-induced depression of respiration is highly dangerous. Thus, the use of remifentanil, which has a half-life of 3–5 min due to its metabolism by non-specific esterases in blood and tissues (independent of renal or hepatic function, so no problems about accumulation), appeared ideal. As remifentanil reduces the dose of propofol required to attenuate responses to surgical stimuli, we gave a lesser dose of propofol (1 mg kg\(^{-1}\)), which we believe may have lessened the risk of serious cardiovascular disturbances.

Cisatracurium is eliminated by tissue esterases and the Hofmann process and is a useful muscle relaxant in these morbidly obese patients who often suffer from liver dysfunction. The dose of cisatracurium, given on the basis of the IBW, was satisfactory; however, the first incremental dose (given on the basis of the response to neuromuscular stimulation) had to be given 46 min after the first dose, i.e. earlier than might have been expected in a non-obese patient (on average 74 min). In conclusion, we consider remifentanil and cisatracurium, in doses according to IBW, to be suitable for general anaesthesia for superobese patients.

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