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Unilateral Blindness after Prone Lumbar Spine Surgery

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VISUAL loss after nonocular surgery is a rare but devastating perioperative complication.[1] It has been documented in a wide variety of procedures, the most common of which are cardiopulmonary bypass, head and neck operations, and prone spine operations.[2] Ischemic optic neuropathy is the most common diagnosis in these cases. Factors believed to be associated with its occurrence are large intraoperative blood loss, long duration in the prone position, and intraoperative hypotension and anemia.[3,4] We present a case of unilateral posterior ischemic optic neuropathy occurring after an uneventful prone lumbar spine operation in a relatively healthy man who did not have decreased blood pressure and hematocrit perioperatively.

Case Report

A 58-yr-old, 80-kg man presented for an L2–L3 posterior spinal instrumentation and fusion. The patient had a 22-pack-year history of tobacco, but had stopped smoking at the age of 36 yr. He had undergone radiation therapy for Graves disease 10 yr previously but was not noted to be proptotic at the time of presentation. Preoperative hematocrit was 50% (high end of normal at our institution), and baseline blood pressure was 134/92 mmHg (mean arterial pressure [MAP], 106 mmHg). A 12-lead electrocardiogram showed normal sinus rhythm with left ventricular hypertrophy and left atrial enlargement. Fentanyl (3 μg/kg), propofol (2.5 mg/kg), and lidocaine (0.5 mg/kg) were used for induction of anesthesia. Rocuronium (1 mg/kg) was administered for muscle relaxation before tracheal intubation. Isoflurane (0.8–0.9% end-tidal) in a 50:50 air:oxygen mixture and a sufentanil infusion (0.2–0.3 $\mu g \cdot kg^{-1} \cdot h^{-1}$) were used for anesthetic maintenance. The patient was turned prone onto a Wilson frame with his head supported solely by a soft foam cushion with a cutout for the eyes, nose, and mouth. The head was slightly dependent with an approximate 15° tilt from the longitudinal axis of the body. After turning prone, a low-dose phenylephrine infusion (0.01%) was administered for the majority of the 6.5-h case and was titrated at a rate of 1.67 $\mu g \cdot kg^{-1} \cdot min^{-1}$ or less to maintain an MAP of predominately 80–90 mmHg (range, 70–95 mmHg), despite adequate volume replacement. At the time of initiation of the phenylephrine infusion, 2,000 ml crystalloid had been administered to the patient (calculated fluid deficit, 1,440 ml), and urine output was 600 ml. The requirement for phenylephrine was thought to result from loss of vascular tone during general anesthesia. The eyes were checked approximately every 30 min throughout the procedure. The patient was turned supine after approximately 320 min in the prone position and was noted to have an extremely edematous face. He underwent extubation uneventfully after confirmation of a leak around his endotracheal tube with the cuff deflated and was transported to the recovery room. Estimated blood loss for the case was 800 ml, and urine output was 700 ml. Eight thousand milliliters crystalloid was administered intraoperatively. No blood products were administered. The hematocrit at the end of the procedure was 40% in the operating room and 39% in the recovery room.

That evening, the patient was noted to be comfortable, with intact lower extremity motor and sensory function. Early the next morning, the patient reported decreased vision in his left lateral field to the orthopedic service for the first time. He had noted blurry vision in the recovery room the day before, but did not verbalize any complaints because he thought it was caused by residual anesthesia. An ophthalmology consult was obtained. There were no signs of facial bruising or trauma. Fundoscopic examination yielded normal results bilaterally. An afferent pupillary defect was present in the left eye, and visual acuity was decreased to 20/800 compared with 20/20 on the right. Intraocular pressures were 18 mmHg bilaterally. Laboratory workup revealed a normal erythrocyte sedimentation rate and negative syphilis serologies. A magnetic resonance image of the head, performed approximately 24 h after surgery, was read by the radiologist as a probable lesion in the left posterior optic nerve on the T2-weighted images, but it could not be confirmed because of lack of enhancement. Fine sections through the optic nerve were not obtained because magnetic resonance imaging was performed on a "stroke protocol." Re-review of the image with a neuroradiologist did not reveal any definitive lesions. Visual evoked potentials and electroretinography were not performed. The final diagnosis was posterior ischemic optic neuropathy. No treatment was recommended by the ophthalmologic consultants.

At the time of discharge, the visual acuity in his left eye had improved to 20/400. At 3 weeks' follow-up, the patient's ophthalmologic examination revealed a pale optic disc and a persistent relative afferent pupillary defect in the left eye. Color vision was significantly decreased in the left eye, and the visual field was still restricted with an inferotemporal quadrant defect, but central visual acuity had improved to 20/60. Six months after the injury, the left eye had improved central visual acuity to 20/25 and improved color vision but remained with a persistent temporal field deficit.

This article is accompanied by an Editorial View. Please see: Roth S, Barach P: Postoperative visual loss: Still no answers—yet. ANESTHESIOLOGY 2001; 95:575–7.

Discussion

Symptomatic visual defects have been reported to occur as infrequently as 1 in 60,965 anesthetic procedures for nonocular surgery,[1] but the incidence in selected operations, such as cardiac surgery, may be as high as 4.5%.[5] Asymptomatic retinal microemboli have been documented in up to 100% of patients undergoing cardiopulmonary bypass procedures.[6] Other procedures that account for a large proportion of the cases of perioperative visual deficits are head and neck surgery (*e.g.*,

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sinus surgery and radical neck dissections) and spine surgery in the prone position.[2] The etiology of the injury to the eye is unknown in most cases but is probably multifactorial, and a number of potential risk factors have been implicated.[2,4] Although prolonged direct compression of the globe can cause blindness, it is not the usual cause. Blindness has occurred after operations in the supine position and after prone positioning with the head in Mayfield pins.[7] Visual injury due to direct compression of the globe is caused by retinal vascular occlusion,[2] which can also occur as a result of emboli and possibly increased intraocular pressure.[2,4] The most common diagnosis in patients in whom perioperative visual deficits develop after nonocular surgery is ischemic optic neuropathy.[2] Ischemic optic neuropathy is categorized as either anterior or posterior, depending on the location of the lesion in the optic nerve. Retinal vascular occlusion and cortical blindness are less common diagnoses. Procedure-related factors that have been implicated in this complication are emboli, extreme hypotension of prolonged duration, direct pressure on the globe, large blood loss, massive transfusion of blood and fluid, and anemia.[2–5] However, some cases of perioperative visual injury, such as the current case, do not have any of these speculated associated factors, except perhaps for a large amount of intravenous fluids.

A number of case reports and case series of perioperative visual complications occurring after spine surgery in the prone position have been published.[2,3,7,8] Myers *et al.*[3] compared a group of 28 patients in whom visual deficits developed after prone spine surgery to a group of 28 controls matched for age, type of surgery, approach, number of spinal levels being fixed, instrumentation, and primary *versus* revision surgery. Factors that were significantly different between groups in the study of Myers *et al.*[3] were operative time (mean 430 min in the blindness group *vs.* mean 250 min in the control group) and estimated blood loss (mean 3,600 ml in the blindness group *vs.* mean 880 ml in the control group). Despite these significant differences in mean operative time and estimated blood loss between groups, most patients who undergo spine surgery in the prone position for a prolonged duration with a large estimated blood loss do not wake up with visual deficits. Therefore, long operative time and large estimated blood loss may place patients at higher risk for development of visual deficits, but they are not absolute determinants for this complication. Clearly, other factors, intrinsic either to the patient (*e.g.*, ocular vascular anatomy, coexisting illnesses) or to the operation (*e.g.*, positioning on frames, type and amount of fluid and blood replacement), may have important contributory roles in the development of postoperative visual deficits.

The operation described in this case report had a prone duration intermediate between the times for the blindness group and the control group in the study of Meyers *et al.*,[3] and the estimated blood loss in this case report was approximately the same as in the control group of Myers *et al.*[3] The blood pressure was maintained within 25% of baseline, and the eyes were checked frequently to ensure there was no direct pressure on the globes. This patient may have had mild untreated hypertension based on his preoperative diastolic blood pressure of 92 mmHg and his electrocardiogram, which showed left ventricular hypertrophy and left atrial enlargement. Graves disease is known to cause proptosis, which theoretically may place a patient at higher risk for visual loss, but his thyroid had been irradiated 10 yr before this operation, and he was not proptotic. Although his preoperative hematocrit of 50% was in the high normal range, he was hemodiluted with crystalloid to a hematocrit of 40% by the end of the procedure. This makes the possibility of high viscosity causing decreased blood flow an unlikely explanation.

To what extent the phenylephrine infusion, the 8,000 ml crystalloid, or the positioning on the Wilson frame may have contributed to this complication is not clear. The effects of phenylephrine on the vascular supply to the eye have not been studied, and the available literature is contradictory as to whether vasoconstrictors are beneficial or harmful.[9,10] It has been speculated that posterior ischemic optic neuropathy may be caused by increased central venous pressures in the prone position and venous congestion in the orbit of patients who are in the prone position for a long duration while receiving large amounts of fluid.[2,8] Many of the orthopedic frames (*e.g.*, Relton-Hall and Wilson) are designed so that the head is in a dependent position to the body, which decreases venous outflow from the head. The prone position is also known to cause an increase in intraocular pressures (IOP),[11] which may decrease the ocular perfusion pressure (PP_{op}) by the equation $PP_{op} = MAP - IOP$.

The current case is unusual because it lacked excessive intraoperative blood loss, hypotension, or anemia. Although the patient may have had mild hypertension, his degree of vasoocclusive disease does not fit the usual description of ischemic optic neuropathy that occurs spontaneously in the community.[12] If a similar patient were to present to the hospital today for the same procedure, he or she would not be considered at high risk for developing this complication. This suggests that, despite the apparent increase in the incidence of perioperative visual deficits over the past 5–10 yr, we do not know the precise etiologic factors, nor do we know how to prevent it. Other factors that may never be discernible preoperatively, such as unique vascular anatomy or oc-

ular hemodynamics, may be the most important risk factors for developing perioperative visual deficits.

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Off-pump Coronary Artery Bypass Surgery in a Patient with C1 Esterase Inhibitor Deficiency

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ANGIOEDEMA is a rare disease caused by a deficiency of C1 esterase inhibitor. Deficiency, either absolute or functional, of this moderator of the complement cascade can result in unregulated complement activation during periods of infection, trauma, or stress. The clinical presentation can include edema of the head and neck, mucous membranes, and gastrointestinal tract. The most alarming cases include the potential for severe airway edema.[1]

Cardiopulmonary bypass (CPB) is a particularly stressful perioperative event and is known to significantly increase complement activation, further increasing the risk of angioedema in C1 esterase inhibitor–deficient patients.[2,3] Few reports exist of the management of this rare but significant disease during cardiac surgery. We present a successful case of coronary artery bypass grafting (CABG) in a C1 esterase inhibitor–deficient patient using an off-pump technique.

Case Report

A 45-yr-old man was admitted to the hospital for monitoring and investigation of unstable angina. He had a history significant for coronary artery disease, including an inferior myocardial infarction 2 yr previously. Also significant was a history of C1 esterase deficiency (with several previous episodes of angioedema) being treated with stanozolol (6 mg daily by mouth). He was also receiving an oral β blocker and calcium channel but not an angiotensin converting enzyme inhibitor. Coronary angiography revealed a 100% stenosis of the left anterior descending artery and a 75% stenosis of the right coronary artery, with a 50% left ventricular ejection fraction. Given these results, it was elected to perform CABG surgery.

Considering the risk associated with the activation of complement caused by the extracorporeal circulation, it was opted to perform off-pump CABG (OPCAB). The patient was brought to the operating room, and during placement of invasive monitors, 2 units fresh frozen plasma, aprotinin (1×10^6 kIU intravenously), and hydrocortisone (100 mg intravenously) were administered. Anesthesia was induced with thiopental, fentanyl, and succinylcholine and maintained with isoflurane, pancuronium, and intermittent boluses of fentanyl. Heparin (20,000 U) was administered before vascular grafting, with two additional boluses (each 5,000 U) administered to maintain an activated clotting time greater than 300 s (a level consistent with our institutional OPCAB standards). Protamine (150 mg intravenously) was administered after completion of all three bypass grafts. The patient remained hemodynamically stable throughout the 232-min procedure and was subsequently transferred to the intensive care unit. In the absence of angioedema, he was extubated 3 h after arrival, and after an unremarkable hospital stay, he was discharged to his home 3 days later.

Blood samples for C1 esterase inhibitor concentration determinants were drawn from the arterial line and immediately spun with the supernatant frozen at −80°C until analysis. The analysis was performed using radioimmunodiffusion with the results reported as a percent of normal C1 esterase inhibitor function. The levels were 17, 63, 69, and 50% of normal in the preinduction, postheparin, final anastomosis, and postprotamine periods, respectively.

Discussion

Angioedema, also known as angioneurotic edema caused by the psychologic stress that may precipitate attacks, is a rare condition (most commonly hereditary, but occasionally acquired) with potentially life-threaten-

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ing complications. C1 esterase inhibitor, deficient in angioedema patients, regulates the classic complement pathway, and its absence leads to unregulated complement activation. This can cause increased endothelial and bronchial permeability with resultant angioedema and bronchial edema.

There are four principle reasons why cardiac surgery is particularly problematic in patients with angioedema. Cardiopulmonary bypass leads to an increase in activated complement factors.[2] Complement activation is also observed secondary to the surgical trauma itself.[4,5] Dilution associated with the additional fluid in the CPB prime may further decrease C1 esterase inhibitor concentrations. Finally, it has been demonstrated that heparin–protamine complexes activate the classic complement pathway.[3,6]

This case shows the successful use of an alternative surgical technique for performing CABG in a patient with C1 esterase inhibitor deficiency. The advantages of this technique are several-fold. Firstly, it minimizes direct complement activation by avoiding the foreign surfaces of the CPB apparatus.[5] In a study of 62 patients, Gu *et al.*[7] demonstrated lower complement levels in patients undergoing CABG without CPB compared with those undergoing conventional CABG. Secondly, OPCAB avoids the obligatory dilution of C1 esterase inhibitor resulting from the hemodilution caused by the pump prime. Finally, OPCAB can generally be performed with lower doses of heparin, with the potential for less complement activation from fewer circulating heparin–protamine complexes after heparin reversal. The potential disadvantage of using an OPCAB technique may be an increased propensity toward hemodynamic instability because of the manipulation of the beating heart. Whether this increases the stress response, and with it the potential for angioedema, is not known.

There are several other potential modalities available to treat angioedema patients requiring CABG. In addition to modifying the surgical technique, fresh frozen plasma was preoperatively administered to this patient. Fresh frozen plasma contains a range of complement proteins, including C1 esterase inhibitor. The effect of the fresh frozen plasma can be seen in the increase in C1 esterase inhibitor functional activity from 17% to 63% after administration. The patient was already receiving stanozolol, which may increase C1 esterase inhibitor concentrations through increased hepatic production. However, it may take from 5 to 12 days to be effective. Aprotinin was administered for its generalized antiinflammatory effect, which may decrease complement activation. The rationale for administering steroids was based on its complement production–modifying effects (not on any specific effect in C1 inhibitor deficient patients); however, it may also attenuate the activation of complement caused by heparin–protamine complexes.[8]

There have been several reports about the use of CPB in patients with angioedema. In particular, a patient with angioedema who underwent conventional CABG died after the development of progressively increasing airway pressures, coagulopathy, and pulmonary edema.[9] Successful CPB in patients with C1 esterase inhibitor deficiency has also been reported in cases in which C1 inhibitor concentrate was infused preoperatively, thereby preventing an acute attack of angioedema.[10] Haering and Comunale[11] reported successful use of chronic stanozolol therapy for angioedema with stanozolol increasing C1 esterase inhibitor concentrations to 68% of normal. In comparison, the current patient had activity that was only 17% of normal, despite androgen use.

The patient with C1 esterase inhibitor deficiency requiring CABG presents the anesthesiologist with unique challenges. There are several management options to consider, and we report the use of successful OPCAB.

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Successful Use of Combined High-frequency Oscillatory Ventilation, Inhaled Nitric Oxide, and Prone Positioning in the Acute Respiratory Distress Syndrome

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MANAGEMENT of patients with acute respiratory distress syndrome (ARDS) is a significant challenge to clinicians. Recently, concern has been expressed that conventional strategies using high airway pressures may contribute to lung injury and perhaps to multisystem organ failure in patients with ARDS.[1] Consequently, other strategies of respiratory support have been used in the hope of improving gas exchange while avoiding ventilator-induced lung injury, including prone positioning, high-frequency ventilation, inhaled nitric oxide (INO), and partial liquid ventilation. Because of the complexity of this illness, although individual interventions may not result in improved outcome, combined modalities as part of a comprehensive treatment strategy may become an important feature of future investigations. The strategies mentioned have been used in combination in animals, but rarely in humans.[2–4] We present a case report of the successful use of high-frequency oscillatory ventilation (HFOV), prone positioning, and INO in a patient with severe ARDS.

Case Report

A 56-yr-old man was brought to the emergency department of a community hospital with respiratory failure caused by a drug overdose and aspiration of gastric contents. His medical history was significant for bipolar affective disorder and type 2 diabetes mellitus. He had overdosed on a number of medications, including a benzodiazepine, an antipsychotic, and an antidepressant. He underwent intubation in the emergency department and was transferred to the intensive care unit. His sputum and blood cultures were subsequently positive for *Staphylococcus aureus*, and bronchoalveolar lavage showed herpes simplex virus. He was treated with appropriate antibiotics and supportive care. His chest radiograph initially showed a focal infiltrate, but this progressed to diffuse bilateral infiltrates. One week after admission, he was transferred to our intensive care unit in a tertiary care university-affiliated hospital for further management of severe ARDS. On day 1 in our intensive care unit, conventional mechanical ventilation was continued. A pressure-control mode was used with a peak airway pressure of 30 cm H_2O and a positive end-expiratory pressure of 15 cm H_2O. Delivered tidal volume was 450 ml (5.5 ml/kg). While breathing a fraction of inspired oxygen (F_{IO_2}) of 0.5, his blood gas showed a pH of 7.33, an arterial carbon dioxide tension (P_{aCO_2}) of 61 mmHg, an arterial oxygen tension (P_{aO_2}) of 64 mmHg, a bicarbonate concentration of 30 mEq/l, and an arterial oxygen saturation (S_{aO_2}) of 93%. His oxygenation status worsened, and he required an F_{IO_2} of 1.0 to maintain an S_{aO_2} of 90% or more. The patient was already deeply sedated, and a neuromuscular blocking agent was administered. INO was initiated, and within a short period, his F_{IO_2} was reduced to 0.55 (see table 1 for a summary of ventilator settings, oxygenation, and ventilation at the time of initiation of INO, HFOV, and prone positioning). His respiratory status, however, continued to worsen. Despite a peak inspiratory pressure of 40 cm H_2O, tidal volumes decreased to 300 ml, and worsening hypercapnia developed. Because of concern about high peak pressures as well as the increasing P_{aCO_2} and increasing F_{IO_2} requirements, the patient was placed on a high-frequency oscillatory ventilator (3100 B; Sensormedics, Yorba Linda, CA). The mean airway pressure was initially set at 32 cm H_2O, 3 cm H_2O above the mean airway pressure applied during conventional mechanical ventilation. A few hours later, blood gas with an F_{IO_2} of 0.7 showed an improved P_{aCO_2}. The following morning, because of worsening hypoxemia, the mean airway pressure was increased to 36 cm H_2O, but with further deterioration, the patient was placed in the prone position. Shortly thereafter, the patient's oxygenation improved, and the F_{IO_2} and the mean airway pressure were reduced to 0.5 and 32 cm H_2O, respectively. The oscillatory frequency throughout his course was 4–6 Hz. Because of this excellent response, the patient was placed in the prone position every 6–8 h and left prone for 6–8 h at a time. After 4 days of combined prone positioning and HFOV, he was returned to conventional mechanical ventilation and kept in the supine position. He was gradually weaned from INO, and INO was discontinued after a total of 9 days. During the next month, he was gradually weaned to supplemental oxygen *via* tracheostomy and was then transferred to the ward. No evidence of multisystem organ failure nor any complications related to mechanical ventilation developed. He was subsequently discharged from hospital.

Discussion

Despite profound gas exchange abnormalities, the most common cause of death in patients with ARDS is sepsis and multisystem organ failure.[5] Patients with ARDS have significantly reduced respiratory system compliance; hence, it is often difficult to support oxygenation and ventilation adequately without subjecting them to potentially harmful transpulmonary pressures and tidal volumes. There is a concern that ventilator strategies used in the treatment of these patients not only result in local damage to the lung, but may promote more widespread inflammation, which contributes to

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Table 1. Summary of Ventilatory Modes and Arterial Blood Gases

ICU Day	Time	INO (ppm)	Patient Position	Vent Mode	P_{aw} (cm H_2O)	F_{IO_2}	pH	$Paco_2$ (mmHg)	Pao_2 (mmHg)	Spo_2 (%)	Pao_2/F_{IO_2} (mmHg)	OI
3	17:00	0	Supine	PCV	20	1.0	7.35	67	68	94	68	29
4	04:30	20	Supine	PCV	27	0.55	7.34	70	59	90	107	25
5	10:20	10	Supine	PCV	29	1.0	7.27	82	59	89	59	49
5	12:20	20S	Supine	HFOV	32	0.7	7.37	61	48	86	73	47
6	04:30	20	Supine	HFOV	35	1.0	7.25	90	47	86	47	74
6	08:10	10	Prone	HFOV	32	0.6	7.24	98	73	91	122	26
6	17:15	12	Supine	HFOV	32	0.95	7.32	76	56	89	59	54
7	13:15	10	Prone	HFOV	32	0.5	7.45	59	61	93	122	26

ICU = intensive care unit; INO = inhaled nitric oxide; ppm = parts per million, P_{aw} = mean airway pressure; F_{IO_2} = fraction of inspired oxygen; $Paco_2$ = arterial carbon dioxide tension; Pao_2 = arterial oxygen tension; Spo_2 = oxygen saturation measured by pulse oximetry; OI = oxygenation index; PCV = pressure control ventilation; HFOV = high frequency oscillatory ventilation.

multiorgan failure.[1] A number of strategies have been developed that can support ventilation in patients with ARDS while potentially reducing exposure to these harmful effects. Two such strategies are HFOV and prone ventilation.

High-frequency oscillatory ventilation is one of a number of high-frequency ventilatory modes that has been investigated in the setting of ARDS. The mean airway pressure is generally set 3–5 cm H_2O higher than the mean airway pressure applied during conventional mechanical ventilation, and the alveolar pressure is well above the pressure at which derecruitment of lung units is thought to occur. The variations, or oscillations, around this mean airway pressure are believed to be dissipated and not transmitted to the alveolar epithelium. This theoretically allows the recruitment of lung units without overdistention.

Studies in neonates and children have shown improvements in oxygenation and reduction in chronic lung disease using HFOV; however, no mortality benefit has been demonstrated.[6–8] In adults, studies are limited to case series in which HFOV has been used as a rescue strategy.[9,10] HFOV has been used safely and seems to improve oxygenation; however, randomized control trials assessing secondary outcome measures are lacking.

The prone position has been investigated for a number of years as a therapeutic intervention in ARDS. There have been several uncontrolled trials showing that oxygenation can safely be improved in patients who are turned prone,[11–13] and two randomized controlled trials are currently underway. Patients in the supine position have a pleural pressure gradient that increases dorsally because of the weight of the lung and mediastinal structures. In ARDS, this gradient is exaggerated because of inflammation and edema present in the lung. Prone positioning reduces the pleural pressure gradient as the mediastinal and abdominal organs move ventrally. This allows for recruitment of dorsal alveolar units at any given alveolar pressure. In addition, blood flow is redistributed away from shunt regions, thus increasing areas with a normal ventilation/perfusion ratio.[14]

Another concern in ARDS is severe hypoxemia. Although the major cause of death in patients with ARDS is multiple organ failure, a proportion of patients may die as a result of hypoxemia. In addition, high oxygen levels can theoretically result in promotion of diffuse alveolar damage and possibly impact on long-term lung function. Both HFOV and prone positioning may improve oxygenation by means of alveolar recruitment. Nitric oxide, on the other hand, is a strategy that may improve oxygenation by selectively improving perfusion to well-ventilated areas of the lung, thus improving ventilation/perfusion matching. Recently, a large randomized trial showed acute improvements in oxygenation without mortality benefit.[15]

Given the mechanisms of action and potential benefits of each of these interventions alone, it is possible that together they may have an additive or even synergistic effect. There have been a few small studies that have evaluated such combined modalities, but to our knowledge, none have shown successful use of INO, HFOV, and prone ventilation in patients with ARDS. In the case described, despite high driving pressures, $Paco_2$ continued to increase, and oxygenation deteriorated despite high levels of positive end-expiratory pressure. The use of INO was followed by an improvement in oxygenation. The use of HFOV allowed improved ventilation and adequate oxygenation while potentially exposing alveoli to lower pressure excursions than those during conventional mechanical ventilation. Turning the patient prone led to further oxygenation improvement, allowing further reductions in mean airway pressure. Although no definite conclusions about outcome can be drawn from a single case report, we have described one patient in whom multiple modalities were used safely and effectively in combination. Patients with ARDS clearly have many complex and dynamic physiologic derangements. During their course in the intensive care unit, these patients often receive hundreds of interventions. Therefore, it is not surprising that trials examining a single intervention have failed to show an improvement in clinical outcome. Future studies should give consider-

ation to combining modalities with complementary physiologic endpoints and perhaps should test a comprehensive treatment strategy compared with usual care.

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Anesthetic Management for Patients with Postpolio Syndrome Receiving Electroconvulsive Therapy

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ALTHOUGH acute poliomyelitis has been virtually eliminated in the United States because of a successful vaccination program, it is estimated that there are between 250,000 (Driscoll *et al.*[1]) and 300,000 (Dalakas *et al.*[2]) survivors of acute poliomyelitis in this country. Many of these survivors are susceptible to the development of postpolio syndrome 25–30 yr after the initial infectious episode.[3] Patients with postpolio syndrome may have severe respiratory sequelae and neuromuscular dysfunction. Thus, they are a significant challenge to anesthesiologists when they undergo operative intervention secondary to coexisting disease. We present a patient with a major depressive disorder and postpolio syndrome who was scheduled to undergo electroconvulsive therapy (ECT).

Case Report

The patient is a 62-yr-old man admitted for ECT to treat major depression refractory to medical management. He had acute poliomyelitis in 1946 and recovered, but postpolio syndrome was diagnosed in 1996, with progressive weakness of both lower extremities. He was wheelchair bound. Other than symptoms related to his postpolio syndrome, his medical history was positive for chronic sinusitis, occasional heartburn, a spinal fusion in 1960 for scoliosis, and shoulder operations in 1995 and 1997, which were performed during general anesthesia without complications. His weight was 70 kg, his blood pressure was 133/73 mmHg, and his pulse was 88 beats/min. Physical examination results were negative except for muscle atrophy and weakness in both lower extremities and decreased reflexes.

We anesthetized the patient on four separate occasions within a period of 8 days to facilitate ECT (table 1). Monitoring consisted of electroencephalography, electrocardiography, blood pressure, pulse oximetry, and neuromuscular response to electrical stimulation. Each time, the patient was preoxygenated using a Mapleson D system and face mask. Esmolol was administered to modify the anticipated sympathetic response to ECT. Anesthesia was induced with 60 mg methohexital (0.85 mg/kg), and as soon as the patient lost consciousness, mivacurium was administered to attenuate the muscular response to ECT. When application of electric current was completed, the patient underwent ventilation with bag and mask until adequate spontaneous recovery of respiratory function. The ECT-induced seizures on these four occasions ranged in duration from 20–57 s. Blood pressure and pulse rate did not vary significantly during any of the four treatments. For the first two treatments, neostigmine and glycopyrrolate were not administered until the patient showed some respiratory effort. For the third and fourth treatments, neostigmine and glycopyrrolate were

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Table 1. Dose of Medication, Seizure Duration, and Anesthetic Time

	ECT			
	1st	2nd	3rd	4th
Methohexital (mg)	60 + 10, 10, 10	60 + 10, 10, 10, 10, 10	60 + 10, 10	60 + 10, 10
Mivacurium (mg)	7 + 3	10	8	8
Neostigmine (mg)	5.0	2.5	5.0	2.5
Glycopyrrolate (mg)	0.5	0.5	0.5	0.5
Esmolol (mg)	50	50	50	50 + 30
Seizure duration (s)	35	57	25	20
Anesthetic time* (min)	30	35	22	24

* Time from beginning of anesthetic induction to when patient was awake, alert, and with adequate spontaneous ventilation in the postanesthesia care unit.
ECT = electroconvulsive therapy.

administered immediately after the electroencephalograph no longer showed convulsive activity.

The anesthesia time, which we defined as the time from the induction of anesthesia until the patient clinically recovered use of his upper intercostal muscles of respiration and was left in the care of nurses in the postanesthesia care unit awake, responsive, and with adequate spontaneous ventilation, was shortened by 6–13 min when neostigmine and glycopyrrolate were administered immediately after his electrically-induced seizure ceased (table 1). In all cases, the patient was closely observed for 1.5 h to ensure that he maintained adequate muscular tone and normal respiratory function.

Because it was anticipated that the muscle relaxant would outlast the methohexital, additional 10-mg increments of methohexital were administered at approximately 4- to 5-min intervals until the patient regained adequate spontaneous ventilation. For the first treatment, 0.1 mg/kg mivacurium was administered, but it was necessary to administer an additional 0.04 mg/kg to produce the desired degree of muscle relaxation. For the second treatment, the entire 0.14 mg/kg was administered in one bolus. For the third and fourth treatments, we decreased the amount of mivacurium administered to a single dose of 0.11 mg/kg with equal success when the ECT stimulus was delivered 5 min after the initial methohexital was administered. The patient's neuromuscular response to train-of-four stimulation did not always correlate with his clinical response to ECT application nor to return of adequate spontaneous respiration as judged by clinical observation and maintenance of excellent saturation of hemoglobin with oxygen. The patient's clinical course improved with each successive treatment, and he spontaneously expressed his satisfaction with his anesthetic management and outcome.

Discussion

Postpolio syndrome is typified by its development 25–30 yr after an acute attack of paralytic poliomyelitis.[3] It is characterized by the new onset of progressive muscle weakness and fatigue in skeletal or bulbar muscles that is unrelated to any other known cause. Dalakas *et al.*[2] reported a long-term follow-up study of patients ranging from 4.5 to 20 yr after being diagnosed with poliomyelitis and studied at the National Institutes of Health. They found that all patients had decreased muscle strength averaging approximately 1% per year. The pathophysiologic basis for this weakness was thought not to be caused by a loss of whole motor neurons but to dysfunction of the surviving neurons that causes a slow disintegration of the terminals of the individual nerve axons.[2] Cashman *et al.*[4] concluded that the extensive reinnervation of denervated muscle that occurs in paralytic poliomyelitis may be followed by late denervation of the previously reinnervated muscle fibers. Clinical symptoms may include fatigue, myalgia, fasciculation, and weakness of skeletal and bulbar muscles with development of new respiratory difficulties and sleep apnea. Physical signs showed muscle atrophy, decreased reflexes, abnormal swallowing function, and, in some cases, vocal cord paralysis.[2]

Postpolio respiratory impairment entails considerable risk of morbidity and mortality, particularly related to anesthesia. Bach[5] reported that in approximately 42% of these patients, new breathing problems develop that require interventional respiratory management. These patients often have laryngeal dysfunction, ranging from laryngeal muscle weakness to unilateral or bilateral vocal cord paralysis. This is especially true for patients with a history of bulbar polio.[1] The patients are at high risk for postanesthetic apnea, aspiration, and vocal cord paralysis.[6,7] Severe hyperkalemia and circulatory collapse have been reported after administration of succinylcholine in patients with neuromuscular disease.[8] Also, these patients frequently report intense muscle pain after receiving succinylcholine. Gyermek[9] observed that patients might have an increased sensitivity to the nondepolarizing muscle relaxants after poliomyelitis. He recommended that the dose of such relaxants should be decreased by half and that neuromuscular function should be carefully monitored.

The anesthetic management of patients with postpolio syndrome for ECT deserves special mention. In many of these patients, major depression that is refractory to medical management may develop, and ECT may be the treatment of choice. The anesthetic management for the majority of our other patients who receive ECT is simple and routine. A short-acting induction agent, such as methohexital or diprivan, is used to induce unconsciousness. Succinylcholine is administered to minimize muscular response to the ECT, thereby minimizing the risk for skeletal fractures, and a β-adrenergic blocking agent is administered to blunt the sympathetic response to stimulation resulting from the electroconvulsive shock.

Full recovery from the anesthetic is anticipated in approximately 10 min.

Because of the potential hazards of succinylcholine in patients with postpolio syndrome, we believe it is prudent to use a very short-acting, nondepolarizing muscle relaxant in these patients. We found that mivacurium, in a dose of 0.11 mg/kg, is adequate to prevent the massive muscle contractions that result from ECT. Administering drugs to reverse muscle paralysis as soon as the convulsion is completed resulted in timely restoration of normal spontaneous ventilation. We believe that it is important to administer small additional doses of a barbiturate during the period when the patient still has the effects of the muscle relaxant because if the patient awakens before adequate muscle strength returns, the possibility exists that he or she will panic from not being able to breathe normally and will fear that something has happened to cause permanent damage. Although we used a neuromuscular blockade monitor during this procedure, it was our clinical observation that adequate respiratory function returned before complete return of the train-of-four.

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Prolonged Horner Syndrome Due to Neck Hematoma after Continuous Interscalene Block

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HORNER syndrome is common immediately after performance of an interscalene block, and its clinical manifestations are generally transient.[1,2] To our knowledge, there is only one report of long-lasting Horner syndrome after single-shot interscalene plexus anesthesia in the literature.[3] We describe prolonged Horner syndrome in two patients in which the formation of a lateral neck hematoma after interscalene block is believed to be the cause of this complication.

Case Reports

Case 1

A 48-yr-old woman, 162 cm tall, 85 kg in weight, was admitted for treatment of a persistent complex regional pain syndrome type I of the right hand. Except for obesity, routine physical and laboratory examinations yielded normal results. Blood coagulation tests were within the normal range. The patient was not using nonsteroidal antiinflammatory drugs or aspirin treatment. We decided to start treatment by means of continuous interscalene analgesia. After identification of the interscalene groove by palpation, a 21-gauge, short-bevel stimulating needle (Stimuplex-A; B. Braun, Melsungen, Germany) connected to a nerve stimulator (Stimuplex-DIG; B. Braun) was introduced through the skin and advanced in the direction of the interscalene groove. With the first attempt, muscle twitches of the deltoid and triceps muscle could be elicited by a current of 1.4 mA with a pulse duration of 0.1 ms. The placement of the needle was adjusted so that the muscle twitches were still present with a threshold stimulation of 0.35 mA. Then a 20-gauge, 50-mm cannula was advanced over the stimulating needle into the interscalene space, and a 22-gauge catheter with a stylet (Polyplex N30-T; Polymedic, Bondy, France) was introduced through the cannula and pushed forward 3 cm into the interscalene space (cannula-over-needle technique). After tunneling the catheter subcutaneously with the aid of an 18-gauge intravenous cannula and fixing it with dressing tapes,[4] interscalene block was performed by injecting 40 ml ropivacaine, 0.6%, through the indwelling catheter. Within 15 min, the block was complete, and the patient experienced no more pain in her hand. Six hours after the initial bolus, a continuous interscalene infusion of 0.2% ropivacaine at a rate of 8–10 ml/h was started through the interscalene catheter, with excellent pain relief. On the third day after interscalene catheter placement, the patient reported blurred vision and painful swelling on the right lateral aspect of her neck. Inspection revealed a swelling around the site of the catheter insertion and concomitant Horner syndrome on the ipsilateral side, including myosis, ptosis, enophthalmia, anhydrosis on the whole ipsilateral side of the face, and conjunctival hyperemia, which was not present at the time of the initial block. The interscalene catheter was removed immediately. Subsequent investigation of the neck by means of ultrasound showed a hematoma (4 × 5 cm) expanding from behind the anterior scalene muscle toward the prevertebral longus colli muscle. No compression of the interscalene brachial plexus

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within the interscalene groove could be shown. Eight hours after removing the interscalene catheter, sensory block of the right arm resolved, but the Horner syndrome remained. Neurologic investigation confirmed the Horner syndrome, but electroneuromyography and the sympathetic skin response did not show neurologic damage of the right upper extremity, including the sympathetic fibers.

The pain associated with the complex regional pain syndrome type I returned and was further treated conservatively. After 3 months, the Horner syndrome was still present. Six months after its first appearance, the symptomatology of the Horner syndrome started to improve. A light residual ptosis was the only remaining symptom. After 1 yr, the Horner syndrome resolved completely.

Case 2

A 20-yr-old woman, 170 cm tall, 65 kg in weight, with anterior instability of the right shoulder was admitted for Bankart repair. At the time of admission to the hospital, physical examination was unremarkable, and the results of routine laboratory investigations were normal. The patient was not to take any medication.

The surgical procedure was performed during general anesthesia in the beach chair position using propofol and fentanyl. After an uneventful surgery that lasted 2 h, the patient underwent extubation and brought to the recovery room. The presence of severe postoperative pain led to the placement of an interscalene catheter to provide continuous analgesia. The same technique described in case 1 for catheter placement was used, but two attempts were necessary. An initial bolus of 30 ml ropivacaine, 0.2%, was administered through the catheter, and 20 min later, the patient was pain-free. Analgesia was maintained with a continuous infusion of 0.2% ropivacaine at a rate of 9 ml/h. The patient left the recovery room 3 h after the initial bolus and was pain-free and comfortable until the next day. Then, the patient reported visual disturbances and a swelling on the neck. The clinical examination showed edema at the site of the catheter insertion and Horner syndrome similar to that of the first case (myosis, ptosis, enophthalmia, anhydrosis, and conjunctival hyperemia) on the same side. The catheter was withdrawn immediately, and ultrasound investigation of the neck was performed. A hematoma (3 x 4 cm) was found, expanding between the prevertebral and scalene muscles, without evident compression of the interscalene brachial plexus. Three hours after removal of the catheter, shoulder pain returned and was further treated with 0.1 mg/kg subcutaneous morphine and 2 g intravenous propacetamol. Further investigations showed no neurologic deficit in her right upper extremity. The only abnormality was the persistence of Horner syndrome.

Two days later, the ptosis improved slightly, and the patient was discharged to her home. Two weeks later, the patient showed no further amelioration. At this time, neurologic examination of the right upper extremity with the aid of electromyography, magnetic resonance imaging of the neck, and duplex sonography of the major cervical vessels were performed. The results of these investigations were all normal. After 3 months, improvement of the ptosis and myosis was evidenced, and 6 months later, only slight ptosis was present, without visual disturbances. The symptoms resolved completely after 1 yr.

Discussion

Transient Horner syndrome is a well-known side effect of stellate ganglion block, interscalene block of the brachial plexus, and occasionally epidural analgesia.[5,6] However, prolonged Horner syndrome occurring after interscalene block is rare and is a matter of significant concern because it may represent traumatic interruption of the sympathetic chain of the neck. Sukhani *et al.*[3] described the only prolonged Horner syndrome after single-shot interscalene block. They did not find a direct cause of this complication, and the presence of a hematoma was not mentioned.

In the current cases, a hematoma expanding between the prevertebral longus colli muscle and the anterior scalene muscle was present and may explain the persistence of Horner syndrome. Indeed, such a hematoma may have compressed and damaged the thin preganglionic cervical sympathetic fibers, which are located in this area.

It is known that the sympathetic preganglionic fibers have the potential to regenerate after denervation. Langley[7] was the first to show the regenerating potential of the cervical sympathetic trunk. In this study, the cervical sympathetic trunk of six anesthetized cats was cut below the superior cervical ganglion, and the author noted that the time needed for full regeneration of the sympathetic nerves was between 1 and 15 months. In another study, McLachlan[8] resected the cervical sympathetic trunk of guinea pigs below the superior cervical ganglion and observed that 3 months were necessary after section to obtain a nearly complete reinnervation. Purves[9] also demonstrated in guinea pigs the ability of sympathetic preganglionic nerve fibers to regenerate. Using the same electrophysiologic method as McLachlan,[8] he showed that 6 months after cervical sympathetic trunk section, the degree of innervation was again almost normal.

According to these animal findings, we believe that a prevertebral hematoma, as described in the current cases, may have compressed and damaged preganglionic cervical sympathetic fibers, resulting in prolonged Horner syndrome. The presence of anhydrosis on the whole ipsilateral side of the face—not only limited to the forehead—is a characteristic sign of preganglionic Horner syndrome, which strongly suggests preganglionic sympathetic fiber damage. The fact that Horner syndrome resolved completely in both of our cases reflects the potential of human sympathetic fibers to regenerate spontaneously. The time needed for complete regeneration and resolution of the clinical symptomatology was approximately 1 yr, which is in accordance with the results obtained from experimental studies. The absence of disturbances of the sympathetic supply of the upper limb in both cases, as shown by electroneuromyographic investigation and sympathetic skin response, suggest the hematoma was located above the stellate ganglion and damaged the sympathetic nerves at this level, sparing those going through the stellate ganglion.

In summary, we described two cases of prolonged Horner syndrome occurring during continuous interscalene analgesia. A complete remission of the symptoms occurred spontaneously after 1 yr. In both cases, a prevertebral hematoma was diagnosed and is likely to be the cause of the preganglionic sympathetic trunk damage. In case of persistent Horner syndrome after interscalene block, examination of the neck by means of

ultrasound to exclude the presence of a hematoma is recommended. It is still not known whether the placement of the catheter itself potentially increases the risk of hematoma, and prospective studies of complications associated with the interscalene catheter are welcomed.

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Comparison between Sevoflurane and Propofol Neuromuscular Effects in a Patient with Myasthenia Gravis: Effective Doses of Vecuronium

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PATIENTS with *myasthenia gravis* (MG), an autoimmune disorder characterized by reduction in functional acetylcholine receptors at the neuromuscular junction,[1] show increased sensitivity to nondepolarizing muscle relaxants.[2–5] During surgery necessitating muscle relaxation, paralytic techniques must be chosen carefully based on neuromuscular function. Many reports have described neuromuscular responses to nondepolarizing muscle relaxants during general anesthesia in MG patients, but little information is available regarding responses during sevoflurane or propofol anesthesia. We describe an MG patient anesthetized with each of these agents given singly in procedures separated by 7 months.

Case Report

A 42-yr-old man was scheduled for fenestration of a mediastinal abscess. The patient, 174 cm tall and weighing 71 kg, had an 8-yr history of MG. When thymectomy was performed shortly after onset of MG, his symptoms decreased but did not resolve. Long-term therapy with prednisolone was required, which may have contributed to abscess formation in the mediastinum. The patient showed a positive response to intravenous injection of edrophonium (10 mg) and also showed a decremental response preoperatively after electromyography with 3-Hz stimulation. Antibody to acetylcholine receptor was highly increased in serum (24.6 nM; normal < 0.37 nM). He was classified as having type IIb MG by the Osserman classification.[6] Results of preoperative examination were unremarkable except for the limb weakness. Prednisolone administration (7.5 mg/day) was continued until the morning of surgery. Hydroxyzine (50 mg) and atropine (0.5 mg) were administered intramuscularly as premedication 1 h before induction of anesthesia. Anesthesia was induced with 4 mg/kg thiopental and 1.5 μg/kg fentanyl followed by sevoflurane (2%) and nitrous oxide (60%) in oxygen. The trachea was intubated after topical anesthesia, without administration of muscle relaxants. The lungs were ventilated so that end-tidal carbon dioxide pressure was maintained at approximately 40 mmHg. Skin temperature over the forehead and the thenar region was maintained between 32 and 33°C.

Neuromuscular function was assessed intraoperatively by acceleration measurements in the orbicularis oculi and adductor pollicis muscles. Electrodes were placed 2 cm anterior to the earlobe and at the wrist. Piezoelectric transducers (TOF Guard; Biometer International, Odense, Denmark) were affixed to the upper eyelid using adhesive tape, as well as to the distal phalanx of the thumb. The temporal branch of the facial nerve and the ulnar nerve were stimulated with train-of-four supramaximal square pulses of 0.2 ms duration (60 mA). The nerves were stimulated every 15 s. At the eyelid, the transducer measured acceleration generated by circumferential contraction of the orbicularis oculi muscle.[7] After 10 min to allow the response to stabilize, 10 μg/kg vecuronium was administered intravenously, followed by increments of 5 μg/kg until blockade in the adductor pollicis muscle exceeded 90%. Responses of the orbicularis oculi and adductor pollicis muscles were monitored continuously. After blockade exceeding 90% was obtained, inspired concentration of sevoflurane was decreased to 1.0%. Neuromuscular data for the orbicularis oculi and adductor pollicis muscles were recorded on a memory card of the TOF Guard. All graphic and numerical neuromuscular data were obtained using TOF Guard Reader software (Biometer International).

Blockades induced in the orbicularis oculi and adductor pollicis muscles with 10 μg/kg vecuronium were 91% and 4%, respectively. Effective doses for 50% and 90% blockade (ED_{50} and ED_{90}) were measured in the orbicularis oculi and adductor pollicis muscles using linear regression for a dose–response curve plotting the probit transformation of the block (%) *versus* the logarithm of the dose (μg/kg). We could not obtain these data for the orbicularis oculi because the first incremental dose of vecuronium produced 100% block. In the adductor pollicis muscle, ED_{50} and ED_{90} were 16.6 and 25.6 μg/kg, respectively. The patient was extubated the next day, with no residual neuromuscular effects of the drug.

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Seven months later, the patient was scheduled to undergo left lung resection to treat the abscess. His myasthenic condition had worsened; the dose of prednisolone had been increased to 25 mg/day, and azathioprine (50 mg/day) had been added. Antibodies to acetylcholine receptors in serum were 77.2 nM. The Osserman class remained IIb. Premedication was the same as before. Anesthesia was induced with 1.5 mg/kg propofol and 1.5 μg/kg fentanyl, followed by continuous intravenous infusion of propofol at 8 $mg \cdot kg^{-1} \cdot h^{-1}$ during administration of oxygen. Neuromuscular function was monitored as before. After baseline measurement, 10 μg/kg vecuronium was administered, followed by additional increments of the same size. Blockades in the orbicularis oculi and the adductor pollicis muscles with 10 μg/kg vecuronium were 81% and 0%, respectively. ED_{50} and ED_{90} were calculated as previously. Ventilation with a bag and mask was continued as greater than 90% blockade of the adductor pollicis muscle was induced for double-lumen tracheal tube intubation. ED_{50} and ED_{90} were 3.0 and 17.7 μg/kg in the orbicularis oculi muscle, and 27.7 and 42.5 μg/kg in the adductor pollicis muscle, respectively. Anesthesia was maintained with propofol and fentanyl, and recovery was uneventful.

Discussion

As sensitivity of nondepolarizing muscle relaxants is increased in patients with MG,[1–5] monitoring of their neuromuscular function is of particular importance. Neuromuscular function is influenced not only by severity of MG, but by anesthetics. In healthy patients, inhaled anesthetics accelerate neuromuscular blockade by nondepolarizing muscle relaxants, but intravenous anesthetics like propofol have only a minor effect.[8–10] In healthy preparations, propofol reduces acetylcholine release from nerve terminals in brain and vascular smooth muscle,[11–13] whereas in MG patients, little information is available about the effects of propofol. Moreover, the backgrounds of MG patients differ greatly in titers of antibodies to acetylcholine receptors, MG symptoms, muscles affected, and treatments. Therefore, estimation of the actual influence of anesthetics on neuromuscular function in patients with MG is considerably difficult. We describe one patient with MG who underwent anesthesia twice. MG was worse at the time of the second operation than at the first. Anesthetics used were sevoflurane for the first operation and propofol for the second.

Anesthetic potency between sevoflurane (first procedure) and propofol (second procedure) is difficult to compare: first, the former is inhalation, whereas the latter is intravenous anesthetics; second, corticosteroid inhibits the synthesis of GABAergic steroids[14] and may lead to antagonistic interference with propofol. Therefore, comparison of their neuromuscular effects requires a deliberate interpretation.

We used the orbicularis oculi as a measure of neuromuscular blockade of forehead muscles. The orbicularis oculi and corrugator supercilii muscles are representative of forehead muscles and can be monitored by piezoelectric transducers,[15] but the orbicularis oculi has been proposed as an informative index in patients with myasthenia gravis.[5]

We tried to calculate ED_{50} and ED_{90} in both muscles, but the orbicularis oculi muscle was so sensitive at the time of the first operation that the first incremental administration of vecuronium caused 100% blockade, so a dose–response curve for the orbicularis muscle during sevoflurane anesthesia could not be constructed. However, in the orbicularis muscle, administration of 10 μg/kg vecuronium caused 91% blockade during sevoflurane anesthesia and 81% during propofol anesthesia. Sensitivity to vecuronium was greater during sevoflurane anesthesia than during propofol anesthesia in both muscles, even though MG was worse at the time of surgery during propofol anesthesia.

Antagonistic interactions between aminosteroid relaxant (vecuronium) and corticosteroid were shown by a previous study.[16] The dose of prednisolone was increased from 7.5 (first procedure) to 25 mg/day (second procedure). Therefore, in the second procedure, neuromuscular effect had been expected to be greater if the myasthenic condition was the same. These results indicated that the difference in effective doses between sevoflurane and propofol anesthesia may be more marked.

As in a previous report of MG patients, sensitivity to vecuronium was greater in the orbicularis oculi than in the adductor pollicis muscle,[5] unlike the situation in healthy patients.[7,17] The pattern of increased sensitivity to vecuronium in both muscles in this patient was consistent with MG. The differential response in the orbicularis oculi and the adductor pollicis muscle can be attributed to a greater decrease in margin of safety for neuromuscular transmission in the ocular muscles than in the adductor pollicis, reflecting the preferential involvement of ocular muscles in MG.[1]

In the current MG patient, sevoflurane had a more marked effect than did propofol on neuromuscular function, as is true for healthy patients. Residual neuromuscular blockade would be more frequent during sevoflurane anesthesia than during propofol anesthesia. However, the effective dose of nondepolarizing muscle relaxants varied considerably in the MG patients described previously. Inhaled anesthetics, such as sevoflurane, remain important when muscle relaxation is necessary; blockade need not be kept light in MG patients, but neuromuscular function must be closely monitored.

In conclusion, we described two separate instances of anesthetic management with different agents in the same patient with MG. As is true in general, propofol had less neuromuscular effect than sevoflurane in the current MG patient.

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