Abstract: The role of regional anesthetic techniques such as cervical epidural and inter-scalene brachial plexus blockade (ISB) in rehabilitated stroke patients remains uncertain. We present a case of rehabilitated stroke patient with reactive airway disease, which was managed using cervical epidural anesthesia for an above the elbow amputation. We further discuss the implications of the performance of cervical epidural anesthesia in post stroke patients.

CASE REPORT

A 64 year old male [body mass index (BMI) : 30.5 ; height : 172 cm ; weight : 90 kg] presented to the emergency for an above elbow amputation of left upper arm. The reason for the amputation was a distal wet gangrene secondary to a peripheral vascular disease. Medical history included type 2 diabetes mellitus and hypertension. The patient was on insulin for glycemic control and metoprolol 50 mg twice daily for blood pressure control. He also had asthma and was treated with inhaled salmeterol, budesonide and ipratropium bromide. One year ago, he had a cerebrovascular accident, the consequences of which were a residual weakness on the left side and a right facial nerve palsy. A non-contrast CT scan of the brain at the time of the cerebrovascular event did not reveal any infarct or hemorrhage. The search for an etiological source revealed atherosclerosis of the right internal carotid artery, which was 50% occluded. There were no other sources of emboli. A clinical diagnosis of lacunar stroke causing pure motor hemiparesis due to a posterior limb of the internal capsule infarct was made. Putative causes were a thrombosis of the feeding vessel or emboli from the carotid source. The patient was not receiving any secondary prophylaxis for stroke, although clopidogrel was advised after the initial event. The respiratory examination was normal except for the presence of mild polyphonic wheezes in lung fields bilaterally. Neurological examination revealed a residual weakness on the right facial nerve territory, resulting in a slurring of speech and dysarthria. There was no other cranial nerve involvement. The patient had a left sided motor weakness (4/5 power in upper limb and 5/5 power in lower limb), with no sensory deficit. The deep tendon reflexes were exaggerated in the hemiplegic side. The trunk muscle function (abdominal and intercostal) was found to be normal. The preoperative spirometry showed a forced vital capacity (FVC) of 3.8 L (predicted : 4.12 L), a forced expiratory volume in 1 sec (FEV1) of 2.8 L (predicted 3.31 L), a peak expiratory flow (PEF) of 265 L/min (predicted 355 L/min) and a forced expiratory flow 25-75% (FEF25-75) of 112 L/min (predicted 152 L/min). This was compatible with a mild obstructive pattern. The response to bronchodilators was positive. The improvement was 15% in FEV1 and 260 ml in FVC. Other laboratory investigations were normal and resting echocardiography revealed a normal systolic function (visual ejection fraction of approximately 55%) and a grade 1 diastolic dysfunction.

After obtaining written informed consent and shifting into the operating room, monitoring of blood pressure, pulse oximetry and ECG was installed. The 3 interscalene brachial plexus block attempts using a nerve stimulator technique (Stimuplex® – DIG, B Braun, Melsungen, GMBH, Germany) failed and further attempts at blockade were abandoned. As a consequence, a cervical epidural anesthesia (CEA) was proposed to the patient, after explaining that the technique would also block his contralateral limb. A catheter was inserted into the radial artery for invasive arterial
blood pressure monitoring and close watch at the hemodynamic response to CEA. Repeated blood gas analyses were also planned. The cervical epidural space was identified at C7-T1 by the loss of resistance to saline technique. It was found at 4 cm from the skin. The epidural catheter was inserted and a length of 4 cm was kept inside the epidural space. After ruling out intrathecal or intravascular placement, a total of 8 ml of 0.5% bupivacaine was administered in a graded fashion, until obtaining a C5 to T2 segment blockade. Surgery lasted for 80 minutes and was uneventful except for a fall in mean arterial blood pressure from a baseline value of 108 mmHg to a lowest value of 84 mmHg. Low blood pressure was managed using intermittent boluses of IV phenylephrine. The respiratory rate increased from a baseline value of 22-24 breaths/min to 28-32 breaths/min. Baseline blood gas analysis was normal. Mild hypercarbia (47 mm Hg, 8 mm Hg above baseline value) occurred during surgery.

**DISCUSSION**

Regional anesthetic techniques are a known risk reduction strategy to limit the incidence of respiratory system decompensation in poorly controlled reactive airway disease (1). The beneficial effect is mainly due to the attenuation of bronchial reactivity by local anesthetic agents (2). However, reports of CEA in post stroke patients and their implications are sparse. An obstructive airway disease places an extra burden on the diaphragm. It increases breathing work, more so if a residual paralysis of trunk muscles is present. Before opting for an upper limb regional anesthesia technique in post stroke patients, it is imperative to know the residual power of trunk muscles (3). Indeed, both brachial plexus blockade and CEA are known to cause phrenic nerve palsy (4). In that case, the 10 to 30 % associated decrease in FVC needs to be compensated by the trunk muscle activity, which involves abdominal and intercostal muscles (5).

The respiratory effects of CEA depend on the block extent and, hence, on the administered amount of local anesthetic agents. Usually, CEA induces a mild restrictive pattern, with a slight reduction of lung volumes due to partial paralysis of the diaphragm and intercostal muscles (6). Extension of the block has an inherent risk of respiratory failure due to the exacerbation of these effects. The superimposition of a restrictive pattern in a patient with an already present obstructive syndrome may further increase breathing work. The slight increase in respiratory rate in our case could denote the increase in trunk muscle activity to compensate for a decreased elastance and increased resistance. Such a response can be severe when inadvertent spread involves the intercostal muscles, although not noticed in our case. Our patient had good recovery from his stroke with adequate muscle function on the affected side. His obstructive lung disease was minimal in severity. However, the application of CEA in such a patient is not without risks. Signs and symptoms of compromised respiratory functions should be looked for during the preoperative evaluation to decide whether CEA can be applied. A history of primary etiology of stroke, extent of rehabilitation, residual muscle function, presence and severity of the underlying lung disease and adequacy of its control should be considered prior.

The potential cardiovascular complications of CEA are bradycardia and hypotension. The occurrence of such events depends on the extent of blockade. A close watch to the progression of blockade and a graded administration of local anesthetic agents may help preventing them. In contrast to the earlier hypothesis that a cervical epidural blockade results in a higher parasympathetic tone, it has been shown that CEA depresses the vagal modulation of heart rate and depresses the baroreceptor reflex (8). It can be safely used without any increased risk of vagal hypertonia. Hypotension only occurs when upper thoracic segments are blocked, as seen in our case.

Stroke during the perioperative period is more common than previously thought. Predisposing factors are a previous history of stroke, vascular and metabolic diseases and atrial fibrillation. The anesthesiologist performing CEA should closely monitor patients during the perioperative period, and be aware of potential complications that may mimic stroke, such as epidural hematoma, total spinal anesthesia or excessive extension of the epidural block (7).

In our patient, other managing options could have been contemplated. They include general anesthesia and brachial plexus blockade. High BMI is known to be associated with an increased risk of brachial plexus block failure, as seen in our case. Hence, further block attempts were avoided, apart from the concerns of potential complications such as pneumothorax and nerve injury (9, 10). The risks associated with general anesthesia in our patient included an exacerbation of the underlying reactive airway disease during airway instrumentation. As a
consequence, regional anesthetic techniques were proposed. Given the apparent risk of respiratory failure secondary to diaphragmatic and intercostal palsy, administration of CEA could be riskier in post stroke patients than general anesthesia. The risk is highest if residual motor weakness does not allow compensation for the changes in lung volumes. A complete neurological examination with emphasis on the adequacy of motor power can aid in decision making. Nerve conduction studies were not performed preoperatively in our patient, given the emergent nature of surgery. However, such studies are important to evaluate the quality of rehabilitation, and identify patients at risk of developing neuropathies (11). In our patient, the graded anesthetic agent administration ensured an adequate spread of block. In addition, the reactive airway disease was mild, and the patient’s rehabilitation from stroke was good. All these factors probably explain the absence of respiratory complications during surgery. However, systematic management of post stroke patients using CEA should certainly not be recommended. Safer strategies such as echo-guided brachial plexus blockade or general anesthesia without airway instrumentation should always be considered.

We did not measure spirometry during the surgical procedure because of evident practical limitations. Ultrasound was not available to guide plexus blockade, and the use of a paresthesia technique was not possible because of the patient’s dysarthria. The side of surgery is also an important consideration. In our case, hemiplegia and surgery were on the same side, possibly limiting the consequences of an additional phrenic nerve blockade on an already weak hemi-diaphragm. Contrarily, blocking a healthy hemi-diaphragm in the presence of a contralateral weak one could be catastrophic.

In conclusion, caution should be exercised while considering the performance of CEA in post stroke patients. Knowledge of the contralateral diaphragmatic function and of the residual power of trunk muscles is vital to appreciate the patient’s ability to compensate for eventual respiratory effects of CEA. CEA cannot be used as a rescue technique in patients at risk of respiratory compromise and with conditions such as chronic obstructive pulmonary disease or post stroke muscle weakness.

References