provide much additional information. Traditional thermodilution cardiac output measurements are inaccurate in the setting of an intracardiac shunt, and because of the patient's anatomy, placement of such a catheter was not an option.

An oximetric central venous catheter was therefore chosen to give at least indirect information about systemic oxygen delivery and myocardial function. Following shunt fractions and potentially manipulating the balance between pulmonary and systemic blood flow was another concern. Increasing the Pa_{CO_2} and lowering the Pa_{O_2} before incision aimed to divert blood away from the pulmonary bed by causing pulmonary vasoconstriction. The rising Sv_{O_2} suggested an increase in systemic oxygen delivery. The estimated shunt fractions remained fairly constant throughout the procedure and no interventions were made on this basis alone.

In our opinion, the use of central venous Sv_{O_2} monitoring substantially facilitated the care of our patient. We also believe that the continuous availability of Sv_{O_2} would have proven extremely helpful in the event of any untoward cardiovascular events. We suggest that anesthesiologists consider such monitoring carefully when confronted with patients such as ours.

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Acute Dystonia during Sevoflurane Induction

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DRUG-INDUCED extrapyramidal syndromes occur most frequently with use of neuroleptic agents, but can also be induced by many drugs acting on the central nervous system.[1] Among these syndromes, dystonic reactions are characterized by involuntary contractions in opposing flexor and extensor muscles that produce sustained and fixed abnormal postures, such as oculogyric crises, tongue protrusion, trismus, laryngeal–pharyngeal constriction, torticollis, or bizarre positions of the limbs and the trunk. In the majority of cases, acute dystonia occurs within 1–3 days of initiating treatment or increasing neuroleptic dosages. Surprisingly, there are few cases of dystonic reactions induced by anesthesia in the literature (*e.g.,* narcotics, propofol, diazepam, enflurane, barbiturates)[2–5] and only one in which the relation between acute dystonia and volatile anesthetics was probable.[6] We describe a patient being treated by phenothiazine drug, who experienced severe dystonia during inhalation of sevoflurane.

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Key words: Antipsychotic medication; dopaminergic pathways; halogenated drug; torticollis.

Case Report

A 19-yr-old man with a history of schizophrenia was scheduled for removal of multiple impacted teeth during general anesthesia. He had been treated by cyamemazine, a phenothiazine antipsychotic drug, in a daily dose of 75 mg for 2 yr, along with 180 mg dihydroergotamine

to avoid neuroleptic-induced hypotension. Physical examination showed an anxious but docile, young man with normal vital signs, except for weight excess and gynecomasty. Findings from neurologic examination were normal. The patient had no history of involuntary movements or illicit substance consumption. No recent change was made in neuroleptic dosage, and medications were administrated up to the eve of surgery. Because he was afraid of venipuncture, a single-breath inhaled sevoflurane induction was planned after premedication by oral 5 mg midazolam. The patient was asked to take a deep breath of room air and forcibly exhale to residual volume, at which time a face mask was applied. He took 4 or 5 maximum breaths in a circle system filled with 8% sevoflurane in a 50% N_2O-O_2 mixture and lost consciousness. Approximately 1 min later, a torticollic posturing began to develop, the stiffness rapidly extending to the left trapezius and scalenus muscles. Then, a severe rotation of the head developed, a trismus and an opisthotonus. An intravenous cannula was inserted and muscle spasm resolved with injection of 30 mg atracurium. The trachea was intubated and lungs were mechanically ventilated. During the sustained contraction of the neck and trunk muscles, the lowest oxygen saturation (Sp_{O_2}) value was 87%. No significant changes in arterial pressure and heart rate were noted. Subsequent anesthesia (300 μg fentanyl, 0.8% end-tidal isoflurane in 50% N_20-O_2) was uneventful. No dystonic reaction was observed during recovery from anesthesia. Findings from postoperative neurologic examination were normal.

Discussion

Neuroleptic treatment is a frequent cause of dystonic movements, although dystonia can be provoked by any lesion in the basal ganglia, brainstem, and thalamus, or can be hereditary. Dystonic movements are characterized by an abnormal pattern of activity seen on the electromyogram, with cocontraction of antagonist muscles and overflow into extraneous muscles.[7] This could be caused by deficient reciprocal inhibition represented at multiple levels of the central nervous system.[8] Such cortical inhibition depends on thalamocortical messages released by the basal ganglia, for which the major role in movement control is to maintain an adequate balance between excitation and inhibition. Dopamine levels in the caudate nucleus and the putamen are clearly involved in the pathophysiology of dystonias. Curiously, the dystonia occurs during hyperdopaminergic and hypodopaminergic states, depending on dopamine relative influence on the direct or indirect striatothalamocortical pathways.[7,9] Moreover, other neurotransmitter mechanisms in the extrapyramidal system may be involved, and the ratios of dopamine:acetylcholine and of dopamine:serotonin blockade are important as well.[10] D_2-dopamine receptor blockade in the basal ganglia is widely believed to be the underlying pathophysiologic mechanism of neuroleptic-induced extrapyramidal syndromes. Halogenated anesthetics do not have any known action on such receptors, but sevoflurane may have interacted with cyamemazine in the current patient by altering a dynamic relation between dopamine receptor blockade and other neurotransmitters in the brain.

There are few reports of acute dystonia induced by anesthesia in the literature. Zabani and Vaghadia[4] describe a patient who exhibited a torticollic dystonia during propofol anesthesia, but the cause of this disorder was a focal cerebral trauma. Another case seems to be attributable to enflurane, but it was impossible to exclude pharmacologic interactions between many central drugs in this patient.[2] Stemp and Taswell[6] describe a woman in whom a spastic torticollis developed during isoflurane anesthesia. As our patient, she took antipsychotic drug (chlorpromazine) for a long time. However, dystonia occurred after 50 min of inhaled isoflurane at a 0.55% concentration in 70% N_2O-O_2, and the patient previously received 50 μg fentanyl, 2 mg midazolam, and 250 mg thiopental; a possible interaction between any of these additional drug could not be excluded. In the current patient, acute dystonia occurred in the first minute after inhalation of sevoflurane, and responsibility of these drugs is very likely. However, the role of N_2O cannot be precluded.

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