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# Spastic Torticollis during General Anesthesia: Case Report and Review of Receptor Mechanisms

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Extrapyramidal reactions are common side effects of antipsychotic medications. We describe the first reported case of torticollis occurring during general anesthesia in a patient receiving such medication.

## CASE REPORT

A 45-yr-old, 70-kg woman with a history of schizophrenia and previous suicide attempts presented for debridement and split-thickness skin grafting of the right forearm, 3 days after a self-inflicted shotgun wound. The patient had undergone initial operative debridement under general anesthesia on the day of the injury at another hospital, without anesthetic sequelae. Other past medical history included an uncomplicated tonsillectomy as a child. She was prescribed chlorpromazine (Thorazine®) 500 mg daily, and although prior compliance could not be documented, she had received her medication at least since admission to the presenting hospital on the day of injury. She had no known drug allergies. Physical examination was remarkable for a somewhat flat affect and a 15 × 8-cm midforearm wound with exposed bone and destruction of extensor muscles.

Upon arrival into the operating room, the patient was calm, and while the usual monitors were applied, fentanyl 50 μg and midazolam 2 mg were administered without undue sedation. While the patient was breathing oxygen, anesthesia was induced with 250 mg thiopental, and tracheal intubation was facilitated with succinylcholine 70 mg. Ventilation was controlled briefly, until spontaneous ventilation returned at an end-tidal carbon dioxide tension of 35 mmHg and end-tidal isoflurane concentration of 0.55% in 70% nitrous oxide–oxygen.

Twenty minutes later (30 min after incision), the patient's head was noted to be deviated to the right. It was assumed that her head had simply rolled to the right on the pillow, but upon attempting to straighten it, the neck was noted to be stiff and could not be moved even with moderate force. Experience with cases of torticollis presenting similarly in ambulatory patients in the emergency room led to consideration of a possible dystonic reaction. Diphenhydramine 50 mg was therefore administered intravenously. The tonic neck stiffness resolved promptly, and the patient's head was centered back on the pillow. No other intravenous agents had been given since induction of anesthesia, and the end-tidal isoflurane concentration had been approximately stable for the 30 min prior to this event. Surgical stimulation at this point in the operation was minimal. Throughout this time the patient's hemodynamics, respiratory pattern, and temperature were stable, and no autonomic signs of lightness of anesthesia, such as sweating or tearing, were present.

At the end of surgery the trachea was extubated with the patient awake, without difficulty. Postoperatively she continued to receive her daily dose of chlorpromazine, and her subsequent hospital course was uneventful.

## DISCUSSION

The classic physical findings during the above-described event, together with their prompt resolution after treatment with diphenhydramine, strongly suggest that this was indeed a case of torticollis, the first such reported to have occurred during general anesthesia. Troublesome movement and posture disorders of the extrapyramidal motor system, including dystonia, motor restlessness (akathisia), drug-induced parkinsonism, and tardive dyskinesia, are common side effects of all neuroleptics, occurring in 20–50% of cases[1,2] (and up to 70–100% if the mildest symptoms are included), and are dose-related.[3] While parkinsonism and tardive dyskinesia usually are late side effects, dystonic symptoms are most common within the first 20–28 h of treatment[4] and occur more often in younger patients. Dystonia includes spasm of the neck muscles that may progress to torticollis; extensor rigidity of the back muscles, even opisthotonos; carpopedal spasm; trismus; and swallowing difficulty, oculogyric crisis, and protrusion of the tongue. Mild symptoms usually subside after the drug has been discontinued. If necessary, parenteral diphenhydramine (Benadryl®) 25–50 mg or benztropine mesylate (Cogentin®) 1–2 mg usually produces rapid reversal of symptoms. Barbiturates and diazepam also have been used to treat acute dystonic reactions.[3] In rare cases, supportive measures, such as maintaining the airway may be necessary.

Since this patient was receiving a low dose of chlorpromazine, the torticollis she developed probably was not secondary to overdose, especially since the patient reportedly took the same dose for long periods of time both before and after surgery without sequelae. Furthermore, since after years of treatment, an extrapyramidal side effect manifested as a single episode of torticollis would be very unusual, the events occurring during her anesthetic—*i.e.*, the anesthetic itself, or the surgical "stress"—would seem to be implicated in the genesis of this reaction.

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Alternatively, it is possible that this represented a true acute dystonic reaction in a patient noncompliant with her prescribed medication (noncompliant, perhaps, because of extrapyramidal side effects) who was restarted on a strict medication regimen once she was admitted to the hospital. Unfortunately, the chlorpromazine concentrations were not measured. However, acute extrapyramidal reactions are most common in the first day of neuroleptic medication,[4] whereas the reaction described occurred on at least her 4th day of certain treatment in the hospital, even if she had been noncompliant earlier. It would also have been a great coincidence for such an acute dystonic reaction to have occurred just at the time that she was receiving a general anesthetic, even more so since the literature suggests that central nervous system (CNS) depressants, among which general anesthetics can be counted, can be used to reverse acute dystonic reactions.[2,5] Indeed, it is perplexing why the thiopental administered during induction of anesthesia did not suppress the dystonic reaction in this case, although levels may not have been high enough 50 min later, when this event occurred.

*In vitro* receptor binding assays with animal and human brain tissue have demonstrated that neuroleptic drugs act at a variety of receptor sites in the CNS.[4,6] Antipsychotic activity is believed to be due to dopamine $D_2$-receptor blockade in the limbic and cortical regions in the brain. In contrast, the extrapyramidal effects are believed to be secondary to blockade of dopamine $D_2$ receptors in the nigrostriatal–basal ganglia pathway,[2] presumably upsetting the balance between dopaminergic inhibitory and cholinergic excitatory impulses in these regions.[4,7,‡] This accounts for the successful treatment of extrapyramidal side effects with anticholinergics.

Since this patient was free of extrapyramidal side effects prior to her anesthetic, it is interesting to speculate on the interactions of inhalation anesthetic agents, in this case nitrous oxide and isoflurane, with dopamine $D_2$ receptors. If the relationship between this patient's general anesthetic and her extrapyramidal symptoms truly was cause and effect, this suggests an antagonist action of the volatile agents on dopamine-receptor binding. Alternatively, volatile agents may have cellular or membrane effects in the CNS that are unrelated to dopamine receptors but that, by some other mechanism, produce end-results similar to dopamine-receptor blockade. Work by Lacey *et al.*[8] and Nicoll[9] seem to indicate just the opposite, however: they showed that general anesthetics had membrane effects equivalent to those produced by dopamine $D_2$-receptor *agonists,* that is, membrane hyperpolarization *via* increased potassium conductance.[10]

Thus, although the effect of dopamine receptor agonists on anesthetic depth (MAC) is being elucidated,[11] the converse—*i.e.*, the effect of the inhalation agents on dopamine receptor binding and the CNS implications of such—has yet to be determined. Despite the partial muscle relaxation produced by volatile agents, whatever the mechanism, it is clear that general anesthesia does not blunt all supraspinal-mediated muscle activity and may in fact facilitate neurotransmission in some of these pathways.

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