

## Postextubation Laryngospasm in a Patient with Spasmodic Dysphonia

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THE anesthesia literature is full of information and recommendations for safely administering general anesthesia to patients with various myotonic, neuromuscular, and vocal cord disorders<sup>1-3</sup>; however, little is published on speech disorders and how these often vocal cord-related afflictions can influence anesthetic management.<sup>4-6</sup> This report discusses an incident of laryngospasm in a patient with spasmodic dysphonia (SD) and demonstrates how even a relatively benign presentation deserves heightened vigilance.

### Case Report

A 59-yr-old, 100-kg man presented for elective bilateral blepharoplasty secondary to age-related ptosis. His medical history was significant for mild hypertension, obesity, and newly diagnosed "spastic dysphonia," for which he was undergoing a workup. He had several previous surgeries during general anesthesia without complication. The surgeon requested that the procedure be performed during general anesthesia secondary to patient anxiety. During the preoperative interview, the patient was noted to have intermittent episodes of difficulty with speech in which he displayed sudden, brief episodes of arrested phonation accompanied by occasional abnormal alterations of intonation. When questioned about this condition, the patient stated that it had been present for many years but that he had never had problems with breathing or general anesthesia. The results of his physical examination were otherwise unremarkable.

The patient was premedicated with midazolam and fentanyl, preoxygenated, induced with propofol, and mask ventilated with ease. Rocuronium was administered, and he was intubated with an 8.0 endotracheal tube without incident. Anesthesia was maintained using nitrous oxide, oxygen, isoflurane, and fentanyl, and the case proceeded without complication. At the end of the 90-min case, neuromuscular blockade was reversed with neostigmine and glycopyrrolate. When the patient was following commands and met extubation criteria (four train-of-four twitches and 5-s sustained tetany of the adductor pollicis muscle, 5-s sustained head-lift, oxygen saturation of 100% with 16 breaths/min, vital capacity > 15 ml/kg), the oropharynx was suctioned, and extubation was uneventful. The monitors were removed, and the patient was in the process of moving himself to the gurney when he began to display signs of respiratory stridor. Laryngospasm was suspected, and positive pressure with 100% oxygen and jaw thrust

were administered by mask. Positive pressure did not break the laryngospasm, and the patient became progressively more agitated as his oxygen saturation fell into the low 70s. Succinylcholine, 80 mg, was administered intravenously. Laryngospasm persisted despite continued positive pressure by mask. Direct laryngoscopy was performed, and the vocal cords were noted to be closed. A second dose of 80 mg succinylcholine was administered, and laryngoscopy was repeated. The vocal cords were open, and the patient was intubated. The patient's oxygen saturation returned to 100%, and volatile anesthetic was administered until neuromuscular function returned. Approximately 45 min after the laryngospasm and succinylcholine administration, the patient again met all extubation criteria. However, this time, the patient was placed in the sitting position to improve respiratory mechanics. He was successfully extubated without signs of postextubation respiratory distress.

The patient was transferred to the postanesthesia care unit in stable condition and observed overnight. He did not experience further episodes of laryngospasm or respiratory distress and sustained no sequela from his brief period of hypoxia.

### Discussion

*Spasmodic dysphonia* is a poorly understood voice disorder resulting from involuntary contraction of the laryngeal muscles.<sup>7</sup> It is a focal dystonia similar to blepharospasm, torticollis, or writer's cramp.<sup>8</sup> The vocal cord spasms of SD typically occur during speech, affecting the voice without impeding respiration.<sup>6</sup> Two clinical types of SD have been identified: the more common adductor type, which affects the thyroarytenoid muscles, and the less common abductor type, which affects the posterior cricoarytenoid muscles.<sup>7-9</sup> In adductor SD, voice breaks are characterized by a strained or strangled quality caused by hyperadduction of the vocal folds.<sup>10</sup> In abductor SD, the vocal fold hypoadduction is thought to be caused by spasmodic bursts in one or both of the posterior cricoarytenoid muscles, the primary abductor muscles of the larynx.<sup>11</sup> The etiology of SD is unknown, and there are no published estimates of its incidence in the general population. It was once thought to be a psychogenic disorder; however, recent evidence suggests an underlying neuropathology.<sup>12-15</sup> It is thought that SD is caused by defective motor control of speech involving systems of motor neurons in the pallidum-subthalamic-supplementary motor area of the midbrain.<sup>16</sup> Attempts to treat SD have included pharmacotherapy, psychotherapy, voice therapy, surgical intervention, and botulinum toxin (botoxin) injections.<sup>17</sup>

In the anesthesia literature, the only discussion of SD is published in the fourth edition of *Anesthesia and Co-Existing Disease*.<sup>5</sup> It references a single case report from

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2000 in which a patient with SD had recurrent postoperative stridor requiring tracheostomy.<sup>6</sup> In that report, the patient had undergone left recurrent laryngeal nerve sectioning, Teflon (DuPont, Wilmington, DE) injections of the left vocal cord, botoxin injections every 3–4 months for many years, and tracheal intubation for postoperative stridor after a diagnostic laryngoscopy. After a urologic procedure during general anesthesia, she experienced postoperative respiratory stridor that required reintubation and tracheostomy due to multiple anatomical problems preventing her from maintaining a patent airway.<sup>6</sup> In contrast, our patient had received several general anesthetics without complication, and although some patients with SD may have other associated neurologic symptoms<sup>5,16,18</sup> or even rarely respiratory symptoms,<sup>5</sup> both of these were denied. Our patient had not undergone any treatment that contributed to the complicated postoperative course in the first case, but he still experienced severe life-threatening postoperative laryngospasm. This may have been due to the increased level of anxiety associated with surgery and the physical stress of mechanically manipulating the airway, each known to exacerbate the symptoms of SD.<sup>5,8</sup> Other possible contributing factors from this and the previous case include evidence that 59% of dysphonic patients have narrowing of the laryngeal vestibule.<sup>19</sup> Both cases of SD-related laryngospasm were intubated, suggesting that laryngeal edema<sup>20</sup> coupled with preexisting laryngeal narrowing may limit airflow through a narrowed orifice. Finally, recent studies performed in patients with multiple system atrophy<sup>21</sup> and animals<sup>22</sup> suggest that negative airway pressure generated during inspiration through a narrow glottis reflexively activates the vocal cord adductor. Negative airway pressure is increased by anxiety, restoration of respiratory drive after general anesthesia, and upper airway obstruction. Safer airway strategies in patients with SD may include preoperative laryngoscopy to assess the degree of laryngeal narrowing, avoidance of endotracheal tube placement or use of smaller-diameter endotracheal tubes, use of nasal continuous positive

airway pressure after extubation,<sup>21</sup> and management of pain and anxiety.

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