Lysis of Interarytenoid Synechia (Type I Posterior Glottic Stenosis): Vocal Fold Mobility and Airway Results

Tanya K. Meyer, MD; Jeffrey Wolf, MD

**Background:** The Type I Posterior Glottic Stenosis (PGS-I) is a well-described but uncommon clinical entity. Despite this, there is little known about the outcome of surgical treatment.

**Methods:** Retrospective case series.

**Results:** Thirteen cases met inclusion criteria. All but one patient had a tracheostomy at the time of initial evaluation. At the postoperative visit, seven patients (54%) had completely normal vocal fold motion. Of the verbal patients, 6 (50%) had normal vocal function as reported by both the patient/caregiver and the physician, and 10 (83%) patients were successfully decannulated.

**Conclusions:** Patients with an isolated interarytenoid synechia have an excellent prognosis with regard to decannulation. Although many patients regain normal vocal fold motion and a return to their preintubation vocal function, a significant proportion can have persistent deficits in vocal fold mobility and some level of dysphonia. This report represents the largest known series of PGS-I cases.

**Key Words:** Posterior glottic stenosis, glottic scar, cricoarytenoid joint ankylosis, cricoarytenoid joint immobility, tracheostomy, voice, decannulation, type I posterior glottic stenosis.

**Level of Evidence:** 4.

**INTRODUCTION**

Posterior glottic stenosis (PGS) can be described as total or partial fixation of the vocal folds in an adducted position as a result of one or more of the following: 1) fibrous interarytenoid adhesion, 2) scarring and contracture of the posterior glottic structures, and/or 3) cricoarytenoid joint fixation. These conditions cause limitation of vocal fold motion with subsequent airway restriction. Depending on the structures involved, PGS is most commonly classified into four types as per Bogdassarian and Olson. The type I posterior glottic stenosis (PGS-I) consists of an interarytenoid fibrous adhesion or synechia with a patent aperture posteriorly. Types II through IV develop progressive involvement of additional structures of the posterior glottis (Fig. 1).

Studies to date regarding surgical management of PGS have reported mixed series (all types) with a 70% to 90% decannulation rate. The treatment and prognosis of the PGS-I is anecdotally felt to be dramatically different from the more advanced types. In the most favorable case, the interarytenoid synechia of the PGS-I can be divided with full return of vocal fold mobility and laryngeal function. In general, it is felt that more complex reconstructive procedures are required for types II–IV. There have been no dedicated reports detailing the clinical characteristics and outcomes of surgical management of the PGS-I with resection of the interarytenoid synechia. This report summarizes our experience with the airway and voice outcomes in this selected group of patients.

**METHODS**

**Patients**

Institutional review board (IRB) approval for this project was obtained from University of Maryland Medical Center in Baltimore, Maryland. This was a retrospective review of all cases of surgically treated PGS-I at a single tertiary care center between the dates of July 1, 2005 to July 30, 2009. The patients were identified using ICD-9 codes 519.0, 478.74, and 478.3, and the CPT code 31541. Medical records were evaluated to determine a diagnosis of PGS-I confirmed by the description of an interarytenoid fibrous adhesion or synechia with a patent aperture posteriorly. Between the dates of July 1, 2005 to July 30, 2009. The patients were identified using ICD-9 codes 519.0, 478.74, and 478.3, and the CPT code 31541. Medical records were evaluated to determine a diagnosis of PGS-I confirmed by the description of an interarytenoid fibrous adhesion or synechia with a patent aperture posteriorly. The patients were identified using ICD-9 codes 519.0, 478.74, and 478.3, and the CPT code 31541. Medical records were evaluated to determine a diagnosis of PGS-I confirmed by the description of an interarytenoid fibrous adhesion or synechia with a patent aperture posteriorly.

**Surgical Technique**

The following describes the general techniques used for most patients in this series. Patients were given 10 mg of dexamethasone and a single dose of intravenous antibiotics (3 g
ampicillin/sulbactam or 900 mg clindamycin) preoperatively. General anesthesia via a tracheostomy site and a laser safe endotracheal tube was used for this procedure except in two cases: one patient did not have a tracheostomy and was managed with jet ventilation; another patient could not undergo direct laryngoscopy due to cervical spine precautions and she underwent transnasal flexible endoscopic CO₂ laser fiber lysis (OmniGuide®, Cambridge, MA) of her synechia under sedation with spontaneous ventilation. For all other cases, the largest laryngoscope that would allow direct visualization of the posterior glottis was used, and the patient was placed into suspension. The tissues of the glottis and trachea were inspected with 0°/C14 and 30°/C14 rigid telescopes. All patients underwent CO₂ laser excision of the interarytenoid synechia as described below except for one patient who had sharp resection of his scar band due to inability to decrease oxygen in the ventilatory circuit to below 60%.

We used the an AcuBlade™ micromanipulator (Lumenis, Santa Clara, CA) attached to an operating microscope (Zeiss, Germany) to deliver laser energy in an Ultrapulse™ mode at 1.5 to 2 watts in a linear pattern. An attempt was made to resect a “bar” of tissue that compromised the synechia rather than just divide the tissue (Fig. 2). For this reason, the band was divided at each side of the bar to an estimated 80%. Then each end was sequentially divided to complete the excision of the “bar.” In this manner we were able to maintain tension on the tissue during the division process.

After division of the synechia, the mobility of the cricoarytenoid joints was assessed by palpation with a blunt spatula. If there was any motion restriction, the glottis was dilated with Jackson dilators to 36 in females or 40 in males, or a pneumatic balloon dilator to 18 mm (either gender). Finally 0.1 cc of triamcinolone acetonide 40 mg/mL was injected into the medial face of the arytenoid tower at the estimated level of the cricoarytenoid joint bilaterally and into the interarytenoid mucosa (Fig. 3).

Patients were monitored overnight in the hospital and given two more doses of 10 mg dexamethasone at 8-hour intervals. They were discharged on a proton pump inhibitor and on 1% prednisolone acetate drops through the tracheostomy site to cough up around the tracheostomy tube and coat the glottic tissues. The patient without a tracheostomy was sent home on a fluticasone propionate 220 mg inhaler, two puffs twice a day. Patients were seen at approximately 2 weeks and started on capping trials, and decannulated at 4 to 6 weeks either at their skilled care facility or after an overnight monitored capped...
hospital stay. Patients were seen for at least one follow-up after
decannulation.

RESULTS

Nineteen cases were identified but six were lost to
follow-up immediately after surgery, and decannulation
status was unknown. Thus, 13 cases were included in
this study; seven women and six men, with an average
age of 43 years (Table I). All patients had a previous his-
tory of prolonged intubation, and all but one patient had
a tracheostomy at the time of initial evaluation. The pri-
mary diagnosis for their hospitalization requiring the
prolonged intubation/tracheotomy is detailed in Table I.

At the initial office visit, all patients had bilateral vocal
fold immobility. By office endoscopy, six patients were
diagnosed with a PGS-I—usually through retroflexed endo-
sopic examination of the subglottis through the
tracheostomy site (Fig. 4). The other seven patients had sus-
ppected posterior glottic stenosis (of unknown type) and were
diagnosed with a PGS-I at the time of operative endoscopy.

Preoperatively, three patients were aphonic due to
inability to pass air through their glottis and four
patients had significantly strained speech from the same
etiology. Postoperatively, 10 patients had moderate to
significant improvement in voicing as self-reported or as
reported by a caregiver or family member. Six patients
rated their voice as near normal. No patients had a dec-
rement in their vocal quality as a result of the surgical
procedure. Patient 1 was nonverbal due to cerebral dys-
function from her closed head injury, and although she
regained vocal fold movement and glottic aperture after
her procedure she remained nonverbal. Patient 8 was
aphonic preoperatively but able to use a speaking valve
postoperatively.

Intraoperatively, nine patients demonstrated cri-
coarytenoid joint fixation or immobility by passive
palpation with a blunt spatula after lysis of their syne-
chia. Four of these patients fully recovered normal vocal
fold motion, including one patient with complete fixation.
Overall, 12 patients (92%) had improved vocal fold mobility
and improved glottal aperture at the postoperative visit. Of
these patients, seven had normal vocal fold motion, five had
some movement limitation, and one patient remained func-
tionally immobile. Ten of 12 patients with tracheostomy
(83%) were successfully decannulated.

This surgical procedure did not improve or worsen
feeding status in any of the patients. Four patients were
PEG dependent preop and remained so in the immediate postoperative time period.

DISCUSSION

Before the advent of prolonged intubation, most cases of laryngeal stenosis resulted from infectious diseases such as syphilis, laryngeal diphtheria, tuberculosis, or typhoid fever. After World War II with the advent of modern antibiotics, this etiology waned and was replaced by blunt trauma to the neck through motor vehicle accidents. In the 1950s, during and after the poliomyelitis epidemic with the dramatic increase in the use of endotracheal tube intubation and mechanical ventilation, prolonged intubation has now become the predominant cause of posterior glottic stenosis. Other etiologies are rarely encountered and include iatrogenic injury, autoimmune and inflammatory conditions, postradiotherapy, and idiopathic causes.5

The development of mucosal disruption of the posterior glottic structures is a common event after even short periods of intubation. Elegant animal studies by Weymuller have clearly described a reproducible sequence of events.6 Glottic injury occurs because the curved body of the endotracheal tube lies against and puts pressure upon the structures of the posterior glottis that includes the medial surface and vocal process of the arytenoid with overlying mucosa, the cricoarytenoid joint, and the interarytenoid muscle with overlying pliable and distensible mucosa. The pressure exerted against these delicate structures causes tissue ischemia and reactive edema within hours. This tissue can subsequently develop superficial mucosal ulceration with an inflammatory infiltrate. With longer periods of intubation, the damage extends to deeper tissue layers. Full-thickness mucosal ulceration develops with erosion of the perichondrium and formation of microabscesses within cartilaginous structures. Reactive granulation develops. Intense inflammatory infiltrates can extend to the cricoarytenoid joint space with synovial hypertrophy and fibrovascular proliferation. Eventually, during the healing process, the distensible and pliable interarytenoid mucosa may be replaced with rigid scar tissue that can extend to one or both cricoarytenoid joint complexes.

Although the majority of patients will evidence some sign of acute laryngeal mucosal disruption with prolonged intubation, most individuals will never suffer any chronic sequelae. Kastanos et al.7 in a prospective study of 19 patients intubated between 2 and 14 days reported that 63% of patients evidenced acute glottic injury including granulation, ulceration, edema, and motion abnormalities. Langmore et al.8 had described some evidence of arytenoid erythema and edema in 95% of patients intubated for over 48 hours and motion abnormalities in 41%. Fortunately, most patients experience spontaneous organ healing. Whited9 reports an incidence of posterior glottic stenosis of 5.5% in a prospective series of 200 patients intubated 5 to 24 days, with a threefold higher likelihood of PGS in the group intubated greater than 11 days. This and similar data have led to the recommendation of tracheotomy for
patients with an anticipated need for mechanical ventilation longer than 7 to 10 days, although not all modern studies support early tracheotomy. The risk factors associated with persistence of intubation injury to the glottis remain unclear, and we cannot yet predict which individuals will go on to develop complications.

It is important to make two critical observations that relate to complications of intubation although not specifically to posterior glottic stenosis. Primarily that an additional laryngeal complication of intubation is vocal fold paresis or paralysis that is felt to be a consequence of pressure against the recurrent laryngeal nerve as it enters the larynx from the cuff of the endotracheal tube. Although this is also considered a pressure phenomenon, it often occurs without the mucosal disruption described above. It may be that there are different patient-related structural factors that determine patterns of injury. As a second point, the tip and cuff of the endotracheal tube can exert pressure on the subglottic and tracheal structures with associated cricotracheal injury and stenosis. Fortunately, these two additional complications probably also share a high rate of spontaneous resolution as does intubation related acute posterior glottic injury.

A review of the literature yields several studies of posterior glottic stenosis that include PGS-I and also descriptive case reports (Table II). Of these 10 cases, 7 had a tracheostomy and all patients developed an improved airway postoperatively, with most reporting normal voice. Only one patient was unable to achieve decannulation, and that was due to tracheostomy site malacia necessitating a t-tube, although he eventually achieved normal glottic function. Wolf additionally performed laryngeal electromyography on his three patients, and all had normal electrical signals.

This is the first reported clinical series describing the surgical results of endoscopic management of PGS-I. In this study, patients with an isolated interarytenoid synechia had a decannulation rate of 83%, improved vocal fold movement in 92%, with seven (54%) patients regaining normal vocal fold excursion. All patients but one experienced vocal improvement with the procedure, although many patients still experienced some degree of persistent hoarseness. No patient had any perturbation of their swallowing function with the procedure.

In this case series the etiology of each case of PGS-I was prolonged intubation, and every patient but one had a tracheotomy. This is consistent with other cases reported in the literature. From this we can postulate on the development of the interarytenoid synechia. Some patients develop quite exuberant granulation tissue surrounding the endotracheal tube (Fig. 5). These patients may fail extubation due to airway obstruction from the fronds of granulation tissue. When a tracheotomy is performed, continued sedation and ventilation discourages laryngeal activity and maintains a permissive laryngeal environment for the granulation tissue to adhere and thereby form a synechia that may remain separated.
from the interarytenoid mucosa posteriorly. If the patient has not suffered additional injury to other posterior glottic structures, lysis of the interarytenoid synechia should permit return of vocal fold movement and laryngeal function.

Many studies have indicated that patients with tracheotomy can have a higher incidence of glottic and tracheal complications—but it is unclear if tracheotomy contributes to injury or is just a marker of a group of patients at high risk of postintubation laryngotracheal compromise.9,11,17 For this reason many authors have advocated a laryngeal examination in patients that undergo tracheotomy with treatment of granulation by careful debridement and intralesional steroid injection.18 Furthermore, this author advocates vigilance in the posttracheotomy care of these patients to expediently progress to cuff deflation, downsizing the tracheostomy tube, and institution of speaking valve trials to normalize laryngeal function.

**TABLE II. Summary of Reports of Type I Posterior Glottic Stenosis.**

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Patients with PGS-I</th>
<th>Total Patients in study</th>
<th>History of Intubation (PGS-I)</th>
<th>Tracheotomy (PGS-I)</th>
<th>Outcome (PGS-I)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Semmler (2010)1</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>Full mobility, good voice, good airway</td>
</tr>
<tr>
<td>Liu (2010)2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Full mobility, good voice, required T-tube for tracheal malacia</td>
</tr>
<tr>
<td>Rovo (2008)3</td>
<td>1</td>
<td>32</td>
<td>NR</td>
<td>NR</td>
<td>“breathing immediately and considerably improved”</td>
</tr>
<tr>
<td>Davis-Malesevich (2007)4</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Decannulated “with excellent results”</td>
</tr>
<tr>
<td>Mau (2007)5</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Full mobility, good voice, good airway</td>
</tr>
<tr>
<td>Wolf (2007)6</td>
<td>3</td>
<td>10</td>
<td>NR</td>
<td>3</td>
<td>All decannulated, no mention of voice outcome</td>
</tr>
<tr>
<td>Whited (1983)7</td>
<td>1</td>
<td>12</td>
<td>1</td>
<td>1</td>
<td>Decannulation, no mention of voice outcome</td>
</tr>
<tr>
<td>Bogdasarian (1980)8</td>
<td>1</td>
<td>10</td>
<td>1</td>
<td>0</td>
<td>Normal voice and airway</td>
</tr>
</tbody>
</table>

NR = not reported.

For this reason many authors have advocated a laryngeal examination in patients that undergo tracheotomy with treatment of granulation by careful debridement and intralesional steroid injection.18 Furthermore, this author advocates vigilance in the posttracheotomy care of these patients to expediently progress to cuff deflation, downsizing the tracheostomy tube, and institution of speaking valve trials to normalize laryngeal function.

**Fig. 5. Proposed sequence of events leading to the development of a type I posterior glottic stenosis.** Please note that image B and C are from the same patient, and images A, D, and E are from three different patients. (A) This operative image shows a normal larynx with an endotracheal tube. (B, C) These images were taken just before tracheotomy of a patient after intubation for 10 days. Panel B shows the granulation tissue that has developed around the endotracheal tube. Panel C shows the endotracheal tube removed and how these fronds will prolapse into and occlude the airway. (D) This image is from a patient with a tracheotomy who developed more “mature” fronds of granulation tissue. (E) An image of a patient with a Type I posterior glottic stenosis.
Although the reported cases in the literature describe uniform return of normal laryngeal motion, this series demonstrated variability in laryngeal function when outcomes are examined more closely. These patients had, to varying degrees, additional damage either to one or both cricoarytenoid joints, to the mucosa of the posterior glottis, and/or to the innervation of the larynx. Although the PGS classification system is conceptually useful, patients may have combined patterns of injury. This classification also does not address possible deficits due to neurologic damage.

There are several flaws in this evaluation, the most significant of which is the retrospective nature that limits the uniformity of the reported results. Standardized measurements of vocal quality were not recorded, laryngoscopy to determine vocal fold motion was not performed in a systematic fashion, nor were validated instruments completed to grade patient perceived vocal improvement. Although the dominant clinical goal of decannulation was achieved at a very high rate, full motion was not achieved in all cases. It is likely that these patients had some residual joint ankylosis—although laryngeal electromyography was not performed to determine if there was any additional neurologic dysfunction that would contribute to lack of motion. Because these patients were often referred from other healthcare facilities, details of the hospitalization at which they received their tracheotomy were not available, nor were associated comorbidities at the time of that hospitalization. Finally, the follow-up of these patients was neither uniform nor sufficient for academic considerations. Patients who lived at a distance or in a skilled facility were asked to follow up on an as-needed bases if problems developed to conserve healthcare resources. Ideally, at least a 1-year follow-up if not more should be reported, as it is known that airway stenosis can be a slowly progressive process and delayed cicatrix can occur.

As a final comment, we have described our perioperative routine but make no attestation that this medical regimen had any influence on the final outcome. Perioperative antibiotics and steroids are routinely given for airway cases, and postoperative topical steroids are given to prevent granulation formation and influence wound healing. Additionally, use of a laser for this procedure is convenient but not required, and any method to conserve healthcare resources. Ideally, at least a 1-year follow-up if not more should be reported, as it is known that airway stenosis can be a slowly progressive process and delayed cicatrix can occur.

As a final comment, we have described our perioperative routine but make no attestation that this medical regimen had any influence on the final outcome. Perioperative antibiotics and steroids are routinely given for airway cases, and postoperative topical steroids are given to prevent granulation formation and influence wound healing. Additionally, use of a laser for this procedure is convenient but not required, and any method to conserve healthcare resources. Ideally, at least a 1-year follow-up if not more should be reported, as it is known that airway stenosis can be a slowly progressive process and delayed cicatrix can occur.

CONCLUSIONS

- Surgical lysis of PGS-I offers an excellent prognosis with regard to decannulation (85% in this study).
- After surgical lysis of PGS-I, most patients will report improved voicing (85%), but may have some persistent deficits in mobility (46%) and persistent dysphonia (50%).
- Surgical lysis of PGS-I generally does not affect swallowing function (none in this study).
- It can be difficult to determine the exact classification of PGS during clinic endoscopy, and these patients should be taken for operative evaluation under anesthesia, especially considering the excellent prognosis if a PGS-I is discovered.

Acknowledgments

I would like to thank Dr. Scott Strome at the University of Maryland for his mentorship, Fleesie Hubbard and Margaret Anderson for their tireless support in maintaining the appropriate regulatory documentation, Allison Comer and Tasha Evans for assistance with figures and artwork, and Dr. Albert Merati and Dr. Allen Hillel for critical review of this manuscript.

BIBLIOGRAPHY

1. Bogdasarian RS, Olson NR. Posterior glottic laryngeal stenosis. Otolar- 
3. Wolf M, Primov-Fever A, Talini YP, Kronenberg J. Posterior glottic steno-
4. Terra RM, Minamoto H, Carneiro F, Pego-Fernandes PM, Jatene FB. La-
6. Weymuller EA Jr. Laryngeal injury from prolonged endotracheal intuba-
8. Colton House J, Noordzij JP, Margia B, Langmore S. Laryngeal injury from prolonged intubation: A prospective analysis of contributing fac-
tors. Laryngoscope 121:596–600.
11. Koshtkareva Y, Gaughan JP, Soliman AM. Risk factors for adult laryngo-
12. Rovó L, Vinciel K, Torkos A, Majovos V, Sotani B, Jori J. Endoscopic ary-
13. Davis-Malesevich M, Merati A. Views of a type I posterior glottic steno-
14. Mao T, Fletcher SD, Cavanagh PW, Coursey MS, Wang SJ. Minicricothyro-
16. Semmler M, Keck T, Reiter G, Gruen PM. Endolaryngeal posterior mu-
17. Stauffer JL, Olson DE, Petty TL. Complications and consequences of endo-