Laryngomalacia
when to treat the squeak

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Children’s Hospital of Pittsburgh of UPMC
Overview

- Evaluation of the Noisy Infant
- Historical Considerations
- Clinical presentation
- Diagnosis
- Theories of Etiology
- Medical Management
- Surgical Management
Overview

- Abbreviations:
  - Laryngomalacia = LM
  - Supraglottoplasty = SGP
  - Polysomnogram = PSG
  - Laryngeal Adductor Reflex = LAR
  - Laryngopharyngeal Sensory Testing = LPST
  - Functional Endoscopic Evaluation of Swallowing = FEES
  - Flexible Fiberoptic Laryngoscopy = FFL
  - Microlaryngoscopy and Bronchoscopy = MLB
Evaluation of the Noisy Infant

- Character
  - Stridor
  - Stertor
  - Wheezing

- Phonation
  - Aphonia
  - Hoarseness

- Degree of distress
  - Retractions
  - Apnea
  - Cyanosis
Evaluation of the Noisy Infant

- Supraglottic
  - Collapses with inspiration
  - Inspiratory stridor, relatively normal phonation

- Glottic/Subglottic (fixed)
  - Biphasic stridor
  - Muffled/hoarse or aphonic voice

- Tracheal
  - Collapses with expiration
  - Expiratory stridor, relatively normal phonation
Differential Diagnosis

- **Congenital**
  - Laryngomalacia
  - Vocal paralysis
  - Congenital subglottic stenosis
  - Saccular cysts
  - Laryngoceles
  - Subglottic Hemangioma
  - Laryngeal Webs/Atresia

- **Acquired**
  - Subglottic stenosis
  - Tracheal stenosis
  - Vocal cord granuloma
  - Subglottic cysts
  - Supraglottic stenosis
  - Papilloma
  - Viral croup
  - Membranous croup
  - Epiglottitis
History

- First described in 1853
  - Barthez and Billiet
  - One of original Pediatrics Texts
- Otherwise normal newborn with inspiratory stridor
  - Worse with agitation
  - Resolved at 10 months
  - External compression?
History

- First laryngoscopic description 1883 (Lees)
  - Presentation to London Pathological Society
  - “...the epiglottis being folded on itself so that the posterior surfaces of its lateral halves were almost in contact and the aryepiglottic folds close together and almost overlapping”
History

- Laryngomalacia “soft larynx”
- ...the commonest congenital laryngeal anomaly of the newborn characterized by flaccid laryngeal tissue and inward collapse of the supraglottic structures leading to upper airway obstruction*

Mechanism of Stridor

Intrinsic laryngeal muscles
Extrinsics (hyoglossus, digastric, palatopharyngeus, and palatoglossus)
Clinical Presentation

- Most common congenital laryngeal anomaly
- Most common cause of stridor in infants and children
  - Intermittent, inspiratory, high pitched
  - Worsens with increased respiratory rate
    - with agitation, crying, feeding
  - Positional
    - Worse: supine, neck flexion
    - Better: prone, neck extension
Clinical Presentation

• Natural History
  • Typically a disease of term infants
  • Age at onset:
    • Birth to 14 days (Thompson 2007, Giannoni 1998)
  • Worsens, peaks 6-12 months
  • Typically benign course
    • 10-20% pts present with severe symptoms warranting surgical diagnostic and therapeutic intervention (Giannoni 1998, Richter 2008)
Clinical Presentation

- Feeding Difficulty
  - 2nd most common sx of LM
    - (Thompson 2007, Giannoni 1998)
  - Dysphagia, aspiration, oral incontinence, slow feeding, regurgitation, GERD
  - Can lead to weight loss, FTT

- Exact mechanism unknown
  - Incoordinated suck-swallow
  - Often improves after surgical management or resolution of stridor
Dysphagia is common in patients with LM and assessment is recommended.
Co-morbidities: GERD

- Most common comorbidity (Thompson 2007, Giannoni 1998)
  - 70-100%
- Sx:
  - Emesis
  - Dysphagia
  - Belching
  - Choking
  - Gagging
  - Failure to thrive
Respiration against a fixed obstruction

Negative intrathoracic pressure

Reflux into esophagus and LPR

Increased obstruction

Increased prolapse

Laryngeal edema

Mutual Pathogenesis?
Co-morbidities: other

- Other comorbidities
  - Neurologic disease (up to 50%)
  - Secondary airway lesions (20%)
  - CHD (7%)
  - Pulmonary disease
  - Craniofacial dysmorphisms

- Trisomy 21
  - LM is most common airway anomaly seen on endoscopy
# Grading Clinical Severity

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
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<tbody>
<tr>
<td>Stridor</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Apnea/cyanosis</td>
<td>No</td>
<td>Brief</td>
<td>Life threatening</td>
</tr>
<tr>
<td>Dyspnea/retractions</td>
<td>No</td>
<td>Yes, not requiring medical tx</td>
<td>Yes, requiring medical attn</td>
</tr>
<tr>
<td>Feeding difficulties</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Weight loss</td>
<td>No</td>
<td>+ or -</td>
<td>Yes</td>
</tr>
<tr>
<td>Failure to thrive</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Other</td>
<td>No</td>
<td>No</td>
<td>Cor pulmonale, pulmonary hypertension, hypoxia</td>
</tr>
</tbody>
</table>

N = 201 (80, 83, 38)

Adapted from Thompson (2007)
### Predictors of Clinical Severity

<table>
<thead>
<tr>
<th>Sx</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
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</thead>
<tbody>
<tr>
<td>Cough/choke with feeds</td>
<td>55%</td>
<td>100%</td>
<td>90%</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>48%</td>
<td>98%</td>
<td>75%</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>4%</td>
<td>33%</td>
<td>82%</td>
</tr>
<tr>
<td>Feeding difficulties</td>
<td>40%</td>
<td>94%</td>
<td>92%</td>
</tr>
<tr>
<td>Weight loss</td>
<td>3%</td>
<td>22%</td>
<td>92%</td>
</tr>
<tr>
<td>Apnea</td>
<td>4%</td>
<td>28%</td>
<td>82%</td>
</tr>
<tr>
<td>Pulm Hypertension</td>
<td>0%</td>
<td>6%</td>
<td>24%</td>
</tr>
<tr>
<td>Cor Pulmonale</td>
<td>0%</td>
<td>1%</td>
<td>10%</td>
</tr>
</tbody>
</table>

N = 201 (80, 83, 38)

Adapted from Thompson (2007)
## Predictors of Clinical Severity

<table>
<thead>
<tr>
<th>Comorbidities</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
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<tbody>
<tr>
<td>GERD</td>
<td>29%</td>
<td>93%</td>
<td>84%</td>
</tr>
<tr>
<td>Neurologic Dx</td>
<td>8%</td>
<td>11%</td>
<td>34%</td>
</tr>
<tr>
<td>CHD</td>
<td>1%</td>
<td>8%</td>
<td>34%</td>
</tr>
<tr>
<td>Genetic Syndrome</td>
<td>3%</td>
<td>12%</td>
<td>40%</td>
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</tbody>
</table>

N = 201 (80, 83, 38) 
Adapted from Thompson (2007)
Laryngomalacia Subtypes

- Six abnormalities (Holinger 1960s)
  - 1. long tubular, omega shaped epiglottis
  - 2. prolapse of cuneiforms
  - 3. prolapse of arytenoids
  - 4. epiglottis falls forward
  - 5. overtly acute epiglottic angle
  - 6. shortened AE folds
Laryngomalacia Subtypes

- Three predominant types
  - Posterior-lateral
    - Arytenoid prolapse
    - Infolding of AE folds
    - Associated with Omega epiglottis
  - Posterior
    - Arytenoid/cuneiform cartilage or mucosal prolapse
  - Anterior
    - Posterior epiglottic displacement

Shah & Wetmore (1998)
Diagnosis

- Radiographic imaging relatively poor sensitivity and specificity.
- Awake flexible fiberoptic transnasal laryngoscopy (FFL)
  - No sedation
  - Upright or semi-reclined
- FEES
  - Penetration and aspiration may suggest need to “upstage” infants (Richter 2008)
Direct Laryngoscopy

- Anterior component
- Epiglottic prolapse
Direct Laryngoscopy

- Posterolateral component
- Tubular epiglottis
- Short AE folds
- Indrawing of arytenoids
Direct Laryngoscopy

- Posterior component
- Prolapse of cuneiforms
Direct Laryngoscopy

- Posterior component
- Prolapse of redundant arytenoid mucosa
Direct Laryngoscopy

- Asymmetrical
- Epiglottic prolapse
- Left arytenoid prolapse
Direct Laryngoscopy

- Anterior and Posterolateral component
- Epiglottic prolapse
- Very tight omega shaped epiglottis
- Indrawing of arytenoids and tight AE folds
Secondary Airway Lesions

- Gonzalez et al (1987)
  - 103 infants DLB for UAO or stridor
  - 57% had LM
  - Synchronous lesions in 17.5%
- Rates 12-64% in subsequent series
  - Tracheomalacia, TVC paresis, SGS
- Need to assess for synchronous lesions below glottis
- Able to assess for posterior laryngeal cleft

Bailey & Johnson 4th Edition, Chapter 78
Secondary Airway Lesions

  - 90 infants with FFL confirmed LM underwent DLB
  - 18.9% SAL
    - 4.7% “clinically significant”
    - 3.9% required surgical intervention
  - DLB rarely indicated in most cases of LM

Bailey & Johnson 4th Edition, Chapter 78
Secondary Airway Lesions

  - 26 infants with FFL confirmed LM and met below criteria for MLB
- Presence of SALs documented

**Table 1** Indications for rigid endoscopy evaluation of children with diagnosis of laryngomalacia on FFL

1. Signs and symptoms
   - (a) Interference with activities of daily living, e.g., desaturation during feeding
   - (b) Persistence or progression of symptoms on close monitoring and review
     - (1) Biphasic stridor
     - (2) Retractions
     - (3) Hoarse cry
     - (4) Cyanosis
     - (5) Apneic spells
     - (6) Respiratory distress
     - (7) Failure to thrive

2. Clinical suspicion of presence of synchronous airway lesions such as previous intubation, cutaneous hemangioma
3. Presence of synchronous airway lesion(s) on flexible fiberoptic laryngoscopy
4. Abnormal airway radiographs

Yuen et al. (2006). IJPORL. 70, 1779—1784
Secondary Airway Lesions

- Only 2 SAL requiring intervention: both SUPRAGLOTTIC
- No adverse outcomes in pts who did not undergo MLB (sample size not reported)

Yuen et al. (2006). IJPORL. 70, 1779—1784
Secondary Airway Lesions

  - 228 infants with LM all underwent FFL, MLB under GA
  - 7.5 % SAL
    - Need for intervention not reported
  - SAL rate in LM is low, most lesions have minor significance

<table>
<thead>
<tr>
<th>Synchronous airway lesion</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tracheal bronchus(^a)</td>
<td>8</td>
</tr>
<tr>
<td>Stenosis of the left main bronchus</td>
<td>4</td>
</tr>
<tr>
<td>Stenosis of the right upper lobe bronchus</td>
<td>1</td>
</tr>
<tr>
<td>Tracheomalacia(^b)</td>
<td>5</td>
</tr>
</tbody>
</table>

Secondary Airway Lesions

- High incidence and increased clinical significance of SAL reported in severe LM pts or pts selected for SGP
  - Dickson et al (2009) 79%
  - Toynton et al (2001) 47%
  - Schroeder et al (2009) 58%
- How to decide when to MLB mild-moderate pts who are not yet candidates for SGP?
Complementary Studies

- Secondary lesions
  - High KV airway films
  - Airway fluoroscopy

Bailey & Johnson 4th ed. Chapter 75
Complementary Studies

- Aspiration
  - Chest X-ray for sequelae of chronic or acute aspiration
- MBSS
- FEES
- Salivagram
Complementary Studies

- GERD
  - Esophagram
    - Snapshot in time
    - Unknown acidity
  - pH probe/impedance
    - Gold standard in adults
    - Undetermined diagnostic criteria in infants and children
    - Needed pre-Nissen
  - Esophagoscopy with biopsy
- BAL
- Milk Scan
Complementary Studies

- OSA, PSG/NAP
  - Incidence in infancy controversial
  - Normative data in infancy is insufficient
  - Goldberg et al (2005)
    - 39 consecutive infants with OSA
      - Flex bronch
      - Laryngomalacia common etiology in <1yo and hypotonic pts.
Etiopathogenesis

- Anatomic Abnormality
- Cartilage Immaturity
- Laryngeal Sensorimotor Integration impairment
Anatomic Abnormality

- First proposed by Sutherland & Lack (1897)
- LM is a result of the exaggeration of an infantile larynx Omega epiglottis present in 50% infants without stridor (Richter 2008)
- Can persist after resolution of stridor in LM
Cartilage Immaturity

- Supraglottic prolapse due to “chondromalacia”

- Immature, abnormally pliable supraglottic cartilage

- Histologic Evidence
  - Shulman et al.: hypercellularity, dilated lacunae, paucity of cartilaginous matrix
  - Keleman: no histologic differences between cases and controls

Shulman et al (1976)
Sensorimotor Integration

- Pollitzer 1884 “nerve imbalance”

- Hypotonia/denervation
  - Thompson & Turner (1900)
    - Applied suction to denervated larynges found medial prolapse
    - Hypothesized cortical immaturity as an etiology in laryngomalacia

- Belmont and Grundfast (1984)
  - High prevalence of neurologic disorders in laryngomalacia pts 23%
  - Cadaveric dissection adult larynges
  - Proposed hypotonia of supraglottic musculature
Sensorimotor Integration

- Analogy to acquired LM
  - Sedation/GA can lead to “induced” laryngomalacia
  - Pts with CNS insults and develop “acquired” laryngomalacia
  - Cases of resolution of laryngomalacia with neurologic improvement
  - ? Model for congenital disease
Sensorimotor Integration

• The neurologic theory includes several proposed mechanisms.
  • General Neuromuscular hypotonia
  • Abnormal efferent pathway
  • Abnormal afferent pathway
  • CNS dysfunction

• Experimental Model: Laryngeal Adductor Response
Sensorimotor Integration

Mechano/chemoreceptors of superior laryngeal nerve located in AE fold

Thompson, DM. 2007. Laryngoscope 117 (Suppl 114). 1-33
Sensorimotor Integration

Generation of involuntary efferent response via vagus results in TVC adduction and swallow
LAR

Crossed Pathways. GA influences reticular formation first

Testing LAR

- Laryngopharyngeal sensory testing (LPST).
- Psychophysical test of LAR and sensation
- Intensity and duration calibrated air pulse to AE fold
- Sensory testing in adults: Threshold for behavioral detection
- Normative adult data: 2-4 mm Hg
Testing LAR

- Infants cannot perform psychophysical test
- Can determine threshold for glottic closure and swallow
- Normative pediatric data not available
  - Infants without neurologic dx or DD evaluated for UAO had average 4.3 mm Hg (Thompson, 2005)

Impaired LPST can be due to afferent, efferent pathways or CNS
Data from LPST

- Thompson (2007)
- 134 LM pts
- Performed LPST at 8-12 wk intervals
- Treatment (medical and surgical) non-randomized

Thompson, DM. 2007. Laryngoscope 117 (Suppl 114). 1-33
Data from LPST

- Thompson (2007)
- Higher thresholds in GERD pts
- No elevated threshold in neurologic pts

Thompson, DM. 2007. Laryngoscope 117 (Suppl 114). 1-33
Data from LPST

- Thompson (2007) Conclusions
  - LM severity correlated with laryngeal sensorimotor integration function
  - Maturation of function over time
  - Mechanism
    - GERD findings suggest it is sensory impairment
    - Lidocaine application worsens LM (Nielson, 2000)
    - Neurologic impairment itself was not related to elevated threshold
Management

- For mild and moderate disease = Expectant
  - Expectant
  - Treat suspected LPR/GER
  - Feeding modifications
  - Monitor for development of severe symptoms

- For severe disease = Surgical
  - Supraglottoplasty
  - Epiglottopexy
  - Tracheostomy
Empiric GERD Tx

- 70-100% of patients with LM have GERD
- H2 receptor antagonist (RA) or Proton pump inhibitor (PPI)
- H2RA: ranitidine 3mg/kg three times daily
- PPI: 1mg/kg daily
- If symptoms worsen ➔ 6mg/kg of ranitidine at night + 1mg/kg of PPI daily
Feeding modifications

• Pacing

• Thickening formula feeds

• Upright feeding position

• Small, frequent feeds
Evolution of Supraglottoplasty

- Variot (1898) first to suggest removal of excess AE fold tissue.
- 1920s surgical techniques introduced in literature
- Inglauer (1922) partial epiglottectomy with nasal snare
Evolution of Supraglottoplasty

Surgical Techniques Introduced, Tracheostomy remained mainstay

1920s → Hasslinger (1928) AEF resection using cupped forceps

→ Schwartz (1944) epiglottic wedge resection

1970s → Fearon (1971) epiglottic-to-tongue base adhesion

→ Narcy (1976) hyomandibulopexy

1980s → Lane (1984) microcup and bulluci scissors to trim AEF and supra-arytenoid mucosa
Evolution of Supraglottoplasty

Modern techniques and concepts emerged

1980s → Seid (1985) CO2 laser

  → Zalzal (1987) “epiglottoplasty” including lateral walls with microscissors

  → “Supraglottoplasty”

2000s → Zalzal (2005) microdebrider supraglottoplasty
## Indications for Surgery

<table>
<thead>
<tr>
<th>Absolute indications</th>
<th>Relative indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cor pulmonale</td>
<td>Aspiration</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>Difficult-to-feed child who has failed medical intervention</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Weight loss with feeding difficulty</td>
</tr>
<tr>
<td>Apnea</td>
<td></td>
</tr>
<tr>
<td>Recurrent cyanosis</td>
<td></td>
</tr>
<tr>
<td>Failure to thrive</td>
<td></td>
</tr>
<tr>
<td>Pectus excavatum</td>
<td></td>
</tr>
<tr>
<td>Stridor with respiratory compromise</td>
<td></td>
</tr>
<tr>
<td>Stridor with significant retractions</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Richter et al. (2008)
Contraindications

- Relatively uncommon
- Proceed with caution
  - Patients with comorbidities
  - Patients with multiple levels of airway obstruction
  - Higher risk of revision SGP and tracheostomy
- Postpone surgery till resolution of URI
Anesthetic Considerations

- Spontaneous breathing analgesia
- ETT in the nasopharynx, mouth
- Spray (1% lidocaine <2 years, 2% ≥ 2yrs)
- 0.5 mg/kg of decadron
- Intubation only for unstable patients or patients with poor pulmonary reserve

GOOD COMMUNICATION WITH THE ANESTHESIOLOGISTS
What is Supraglottoplasty?

- It is a surgery designed to treat LM that aims to trim the aryepiglottic folds and remove soft tissue, overriding the arytenoids.

- Begin with FFL, then full MLB.

- Watch during deep breathing for sites of collapse.

- Suction test (Polonovski, 1990).

- Microlaryngeal suspension.

Richter et al. (2008)
Surgical Set-up

Richter et al. (2008)
Instrumentation

Cold instruments

CO₂ laser

Microdebrider
SGP Instrumentation

- Cold Steel
- CO2 laser
- Microdebrider
SGP Instrumentation

- Cold Steel
- CO2 laser
- Microdebrider
SGP Instrumentation

- Cold Steel
- CO2 laser
- Microdebrider
  - AEF incision with microscissors
  - 800 hZ
  - Oscillating mode

From Groblewski et al. (2009) and Zalzal et al. (2005)
Post-operative care

- Intubation versus immediate extubation
  - Err toward intubation in <1yo (Richter et al., 2008)
- Monitored bed (continuous pulse ox) for overnight observation
- One or two doses of post-operative steroids
- Aggressive empiric reflux therapy
- Feeding
  - Generally can be started when infant is awake
  - Consider ST consult when pre-op aspiration exists
Post-operative care

- Follow-up FFL in 2-4 weeks for FEES exam and Q1, 3 months
- Monitor airway symptoms, apneic spells and feeding adequacy
- Stridor may persist for weeks to months
- Pts with persistent significant UAO
Complications after surgery

- 8%, relatively uncommon
- Increases with multiple comorbidites (>3)
- Site-specific complications most common
- 10% post-op lower respiratory infections
- 2-4% supraglottic stenosis – difficult to treat, so best is prevention
  - Avoid juxtaposed raw surfaces
  - PPI to reduce bridging intraarytenoid granulation
  - Avoid injury to pharyngeoepiglottic and glossoepiglottic folds
## Outcomes

Surgical outcomes from the most prominent series, with high patient numbers, of endoscopic surgical correction of laryngomalacia

<table>
<thead>
<tr>
<th>Authors</th>
<th>Method</th>
<th># Patients</th>
<th>% Success</th>
<th>% Revision</th>
<th>% Tracheostomy</th>
<th>Least successful patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polonovski, et al [31]</td>
<td>Scissors</td>
<td>39</td>
<td>79</td>
<td>3</td>
<td>2.5</td>
<td>GERD</td>
</tr>
<tr>
<td>Loke, et al [9]</td>
<td>Scissors &amp; CO₂</td>
<td>33</td>
<td>69</td>
<td>6</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>Reddy and Matt [58]</td>
<td>Scissors &amp; CO₂</td>
<td>106</td>
<td>93</td>
<td>5.1</td>
<td>11</td>
<td>—</td>
</tr>
<tr>
<td>Toyton and Saunders [13]</td>
<td>NR</td>
<td>100</td>
<td>94</td>
<td>—</td>
<td>1</td>
<td>Neuromuscular disease</td>
</tr>
<tr>
<td>Martin, et al [59]</td>
<td>Scissors</td>
<td>30</td>
<td>83</td>
<td>7</td>
<td>3</td>
<td>Vocal fold palsy</td>
</tr>
<tr>
<td>Lee, et al [34]</td>
<td>CO₂ laser</td>
<td>138</td>
<td>83</td>
<td>—</td>
<td>—</td>
<td>Cerebral palsy</td>
</tr>
<tr>
<td>Thompson [4]</td>
<td>Scissors</td>
<td>62</td>
<td>81</td>
<td>10</td>
<td>8</td>
<td>Neurologic disease cardiac disease congenital anomaly or syndrome</td>
</tr>
</tbody>
</table>

*From Richter et al. (2008)*
Aspiration

• Richter et al (2009)
  
  • FEES (n=50) and LPST (n=28) pre and post supraglottoplasty
  
  • Significant decrease in penetration and aspiration after SGP
  
  • No pts newly developed penetration/aspiration postoperatively

<table>
<thead>
<tr>
<th></th>
<th>Pre-op</th>
<th>Post-op</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penetration</td>
<td>88%</td>
<td>16%</td>
</tr>
<tr>
<td>Aspiration</td>
<td>72%</td>
<td>10%</td>
</tr>
<tr>
<td>LPST</td>
<td>8.23</td>
<td>4.44</td>
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</table>
Aspiration

  - Pts with persistent aspiration more likely to have:
    - Neurologic dx
    - CHD
    - Genetic disorder
  - Mechanism of LPST improvement?
    - Natural history vs. effect of surgery

<table>
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</tr>
<tr>
<td>LPST</td>
<td>8.23</td>
<td>4.44</td>
</tr>
</tbody>
</table>

Outcomes

- **Cold Steel**
  - Faster
  - Tactile feedback
  - No airway fires
  - Bloodier
  - Decreased precision

- **CO2 laser**
  - Less bleeding
  - Increased precision
  - Risk of Airway Fires
  - Longer OR time

No prospective trials comparing these techniques
### Table 4. Overall Results in 106 Study Subjects

<table>
<thead>
<tr>
<th>Main Outcome</th>
<th>Unilateral (n = 47)</th>
<th>Bilateral (n = 59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Success</td>
<td>45 (95.7)</td>
<td>55 (93.2)</td>
</tr>
<tr>
<td>Failure</td>
<td>2 (4.3)</td>
<td>4 (6.8)</td>
</tr>
<tr>
<td>Supraglottic stenosis</td>
<td>0</td>
<td>2 (3.4)</td>
</tr>
<tr>
<td>Aspiration</td>
<td>4 (8.5)</td>
<td>2 (3.4)</td>
</tr>
<tr>
<td>Contralateral procedure</td>
<td>7 (14.9)</td>
<td>0</td>
</tr>
<tr>
<td>Revision bilateral procedure</td>
<td>0</td>
<td>3 (5.1)</td>
</tr>
</tbody>
</table>

From Reddy & Matt (2001)
Epiglottopexy

- Indicated if the primary level of obstruction is a retroflexed epiglottis
- Commonly seen in infants with global delay, hypotonia & neurological disorders
- Tell parents that tracheostomy may be necessary
- Main risks are aspiration, supraglottic stenosis
Epiglottopexy

- Suspension of the patient
- Mucosa of the epiglottis is denuded with CO2 laser (1-10W) under microscopic guidance
- Additionally the epiglottis can be secured to the tongue base with 4.0 vicryl

Laser Epiglottopexy for Laryngomalacia
10 Years’ Experience in the West of Scotland

Andrew D. Whymark, MBChB, AFRCS; W. Andrew Clement, MBChB, FRCS; Haythem Kubba, MPhil, FRCS(ORL-HNS); Neil K. Geddes, MBChB, FRCS, CertGAM

Whymark et al. 2006
Epiglottopexy

Whymark et al. 2006
Indications for Tracheotomy

- Presence of > 3 comorbidities
- Severe sleep apnea
- Worsening symptoms after revision supraglottoplasty

From Bailey & Johnson. 4th Edition
Proposed algorithm for the treatment of mild and moderate laryngomalacia

Mild LM

1m FU + FL

2m FU + FL

3m FU + FL

FU @3m till resolution

Moderate LM

+ Acid suppression

+ Feeding modification

Symp worsen, persist

Complications

SURGERY

Adapted from Richter et al. (2008)
Proposed algorithm for treatment of severe LM

Severe LM
  ↓
Maximum acid suppression and SGP
  ↓
FU 2-4 weeks post op
  ↓
FU as recommended for mild/moderate LM

Consider PSG

Symptoms worsen

Revision SGP

Symptoms worsen

Consider tracheotomy

pH study and Nissen Fundoplication

Adapted from Richter et al. (2008)
Laryngomalacia Protocol

**Symptoms**
- **Grade I (mild):** Stridor +/- coughing with feeds
- **Grade II (moderate):** Stridor plus any of below:
  - Choking/gasping during feeds
  - Frequent regurgitation
  - Brief cyanosis
  - Brief apnea
  - Intermittent dyspnea with retractions
- **Grade III (severe):** Stridor plus any of below:
  - Failure to thrive
  - Cyanosis, dyspnea, or apnea requiring medical intervention
  - Pulmonary hypertension
  - Cor pulmonale
  - Pectus excavatum

*Grade III patients may initiate protocol after supraglottoplasty*

**Initial Visit:**
- MBS or FEES
- 6 weeks:
  - If previous study abnormal, or if persistent dysphagia, then MBS or FEES
- 3, 6, 12 months:
  - Same as above

**Initial visit:**
- If Grade II, pH probe
- 3, 6, 12 months:
  - If previous study abnormal, or if persistent symptoms, then repeat pH probe

**All visits:**
- Airway center form
- Shah assessment-
  - Stridor
  - Weight
  - Age at birth
  - Neurologic status

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Conclusions

- LM is the most common congenital anomaly of the newborn larynx.
- 80-90% of patients have a benign course.
- High pitched inspiratory stridor is the hallmark clinical presentation.
- Feeding difficulties and GERD are seen in 80-100% of patients with LM.
- History, PE and Flexible laryngoscopy aid diagnosis.
Conclusions

• Identifying patients who will benefit most from surgery is of paramount importance

• “Less is More” when performing surgery on the infant larynx

• Strict FU and reflux therapy
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Review Article

International Pediatric ORL Group (IPOG) laryngomalacia consensus recommendations


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e Department of Pediatric Otolaryngology, The Sydney Children’s Hospital Network-Westmead Campus,
Initial Presentation Algorithm

1. Consider CXR in infants where there is concern for aspiration and/or active pulmonary disease
2. Consider AP/Lateral airway films in infant whose clinical symptoms suggest a secondary airway lesion

Infant with inspiratory stridor

- FFL to confirm laryngomalacia or referral to otolaryngology provider

Consider more urgent otolaryngology referral for infants with:
1. Apnea
2. Cyanosis
3. Tachypnea
4. Failure to thrive
5. Difficult to feed despite acid suppression
6. Aspiration/pneumonia
7. Cor pulmonale

Suspected secondary airway lesion

- Evaluation under general anesthesia with laryngoscopy/bronchoscopy

If maintaining oxygen saturations on room air and no feeding issue, then outpatient management appropriate *see section 3

No indication of secondary airway lesion

If airway and/or feeding concern then consider admitting to the hospital or consider more urgent intervention *see section 3
Comprehensive Care Algorithm

Mild Laryngomalacia:
Inspiratory stridor with no other symptoms or radiographic findings suggesting secondary airway lesion

1 month symptom check, if stable or improving, can extend to 3-6 month symptom check

Moderate Laryngomalacia:
Cough, choking, regurgitation, feeding difficulty

Start acid suppression therapy and consider feeding therapy/swallow evaluation
*see section 4

Severe Laryngomalacia:
Apnea, cyanosis, failure to thrive, pulmonary hypertension, cor-pulmonale

Start acid suppression therapy and consider feeding therapy/swallow evaluation
*see section 4

Consider co-morbidities that place infant at high risk to do poorly:
1. Cardiac disease
2. Neurologic disease
3. Respiratory disease
4. Craniofacial dysmorphism

1. Laryngoscopy/bronchoscopy
2. Supraglottoplasty

*See section 5 for after care
Management of the difficult to feed infant algorithm

Start acid suppression therapy and consider feeding therapy/swallow evaluation.

- No clinical signs of aspiration
  - Infant falling off growth curve: Consider supraglottoplasty
  - Infant progressing on growth curve: Clinical follow up for symptom check

- Suspected aspiration
  - Consider CIJR if not already done for problems with oxygen saturation
  - FEES and/or VFSS
  - No aspiration on FEES/VFSS
    - Aspiration on FEES/VFSS with evidence of respiratory compromise:
      - Perform or refer for supraglottoplasty
        - See Severe Laryngomalacia
      - No evidence of respiratory compromise but aspiration on FEES/VFSS:
        - Feeding modification

Prior or concurrent to surgical management, neurologic work-up should be considered in infants with:
1. Hypotonia
2. Endoscopy with aspiration, pooled/frothy secretions, absent supraglottic sensation

If improves, continue treatment for moderate laryngomalacia as above
If pulmonary compromise or no improvement refer or perform supraglottoplasty
*See Severe Laryngomalacia
Post-surgical treatment algorithm and persistent LM

*Consider tracheostomy in patients with:
1. Multiple severe co-morbidities
2. Multilevel airway obstruction not amenable to surgical management
Recommendation for acid supression therapy

- Start with empiric PPI or H2RA
- If symptoms improve, increase dose of PPI or if on H2RA, consider changing to PPI
- Consider pH/impedance probe
- Consider Eryped as a motilin agonist

- Reduce either PPI or H2RA by 50% for 4-8 weeks and then stop
- Reduce remaining medicine by 50% for 4-8 weeks and then stop