Etiology of vocal fold palsy

2006/09/27   R3 石堅
Recurrent laryngeal nerve paralysis: anatomy

- Medulla oblongata → vagus n. → jugular / superior ganglion in jugular foramen, anterior to jugular v. → posterio-medial to jugular v. → inferior / nodose ganglion, immediately inferior to jugular foramen.
  - → pharyngeal plexus, superior laryngeal nerve
- → recurrent laryngeal nerve descends with carotid artery
  - Left, 12 cm → mediastinum, crosses anterior to aortic arch → loops medially under aorta → ascends within tracheoesophageal groove.
  - Right, 5~6cm → loops around subclavian artery → not in tracheoesophageal groove until it approaches cricothyroid joint.

Recurrent laryngeal nerve paralysis: anatomy

- Left: nerve passes inferior thyroid artery
  - Anteriorly: 11~12%; Posteriorly: 50~55%;
  - Between distal branches: 33%
- Right
  - Anteriorly: 26~33%; Posteriorly: 18~25%;
  - Between distal branches: 50%
- Enter the larynx
  - Deep to inferior constrictor muscle, posterior to cricothyroid joint
  - Motor and sensory branch
    - 35~80% divide before entering the larynx.
  - Anterior: motor → ¼ to posterior cricoarytenoid muscle:
    - The sole abductor of the vocal fold.
  - Posterior: sensory → trachea, esophagus, pyriform sinuses.
- Blood supply:
  - Inferior thyroid artery, inferior laryngeal artery, the same vessels that feed the trachea and esophagus.

Mechanisms of nerve damage

- Risk in surgery of neck, chest, skull base.
  - Thermal damage, stretch, cutting, compression, vascular compromise.
  - Frequently not recognized at time of injury.
  - Potential for recovery: proportional to degree of injury.
  - Primary anastomosis is recommended if is cut.

- Slow-growing tumors that infiltrate or surround the nerve.
  - Compensation for paralysis → Symptoms may not be evident even in the presence of an immobile fold.
  - Thyroid tumors, paragangliomas, neurilemmomas, skull base meningiomas.

Etiology

- Asymptomatic paralysis: 30~50%.
- Incidence increases with the age:
  - Increased incidence of cancer and neurologic damage in elderly.
- Left recurrent laryngeal nerve is more frequently involved:
  - Longer course, especially within the mediastinum.
- Surgical etiologies
  - More frequent than tumor.
  - Anterior approaches to the cervical spine, carotid endarterectomy, thyroid surgery, skull base operation

Thyroid surgery

- The most common iatrogenic recurrent laryngeal nerve paralysis: 28% → 8~9% → 4.1%
  - permanent paralysis: 0.5~2.4%,
  - temporary paralysis: 2.6~5.9%.
- Benign thyroid neoplasms: 0.7%
  - 75% paralysis at pre-op, 89% recovery.
- Carcinoma: 8%, chronic lymphocytic thyroiditis: 5%.
- Risk ↑ when the nerve is not identified routinely during operation:
  - 18%, four times greater.
- Local scar formation ↑ → outcome ↓
  - thyroiditis, previous surgery, radiation.
- Plus neck dissection: 13%
- Mediastinal dissection.

Thyroid surgery

- Intraoperative monitoring
  - Fiberoptic observation of vocal fold motion through a laryngeal mask (LMA).
  - Intraoperative electrical stimulation of nerve.
    - Temporary paralyzed: 6.25%
    - No permanent paralyses.
- The nerve was identified and not sacrificed.
  - Post-op hoarseness: < 2 weeks.
  - Permanent paralysis: 0.3%
  - Small pneumothoraces (2%), intraoperative airway obstruction related to the monitoring procedure (5%).

Thyroid surgery

- Intraoperative electromyography (EMG) recording
  - Identify recurrent laryngeal nerve: 75~91%
  - Post-op temporary paralysis: 3.7%
  - Permanent paralysis: 3% → 1.85%
  - Vocal fold hematoma due to electrodes implantation: 7.7%
  - Advise in recurrent goiters, revision surgery, thyroid cancer surgery.
  - Monitoring doesn’t replace meticulous technique and judgment.

- Inflammation and edema of the recurrent nerve secondary to benign thyroid disease.
  - Thyroiditis, thyroid abscess.

Carotid endarterectomy

- Post-op vocal fold immobility: 2~6%
- Exact mechanisms: unknown.
  - Traction injuries from retractors, clamping injuries, interruption of neurovascular supply, compression injury, sharp dissection, electrosurgery.
- Staying close to the arterial wall during manipulation can minimize injuries.
- Pre-op laryngeal examination is suggested.

Anterior approach to cervical spine

- The most frequent complication of this procedure: 2~6%.
  - Predominance on the right side.
- 80% had full recovery within 1 year.
- Right recurrent laryngeal nerve:
  - Positioned higher in neck and had shorter loop as it recurred toward the larynx. approaches the larynx at 25°, whereas 4.7° of left.

Anterior approach to cervical spine

- Hypothesis (I): stretch-induced injury
  - Cloward retractor between larynx and carotid sheath.
  - Fully opened → nerve at 13% to 44% stretch.
    - 6% stretch / 1-hour: 70% action potential amplitude decrease
    - 12% stretch / 1 hour: blocked action potentials completely.
    - 15% stretch: complete intraneural ischemia.
    - 50 g of tension / 5 min: significant loss of function.

  - Temporary paralysis: 6.4% → 1.7% when the cuff was deflated during parts of the procedure.
  - Hoarseness correlated with cuff pressure, length of intubation time.

(I) + (II)

Anterior approach to cervical spine

Skull base surgery

- Vagal neuropathy from paraganglioma excision at skull base.
  - vagal paraganglioma, jugular paraganglioma
- Proximal vagal nerve injuries
  - Loss of laryngeal and hypopharyngeal sensation, loss of palatal sphincteric function → aspiration and dysphagia.
- Preoperative vagal paralysis: 30%.
- After surgery, another 25% developed paralysis.
  - even if the vagus was preserved.
- Standard neurophysiologic monitoring did not predict functional outcome.
- Neoplasms in the parapharyngeal space, infratemporal fossa, superior neck.
  - macroscopic preservation of vagus nerve
  - → no postoperative mobility disorders ~ recovered within 6 months.

Laryngeal Paralysis: Distinguishing Xth Nerve from Recurrent Nerve Paralysis Through Videoendoscopic Swallowing Study (VESS)

Fig. 1. Figures of the laryngopharynx during inspiration and swallowing in normal mobility of the laryngopharynx (a), in left recurrent laryngeal nerve paralysis (b), and in left Xth nerve paralysis (c). The left paralyzed pharynx is passively moving toward the right nonparalyzed pharynx during swallowing because of the asymmetrical squeezing of the constrictors between the right and left sides in the case of impaired unilateral motor pharyngeal branches such as occurs in this Xth nerve paralysis (c). The star indicates the midline of the posterior pharyngeal wall, which is moving during swallowing to the healthy right pharynx, in the case of left Xth nerve paralysis (c), in contrast to an isolated recurrent nerve paralysis (b). The arrows indicate the active movement of the laryngopharynx during swallowing and the empty arrows, the passive rotation of the left paralyzed pharynx to the healthy side (c). The circles symbolize the vocal fold immobility and/or the pharyngeal immobility.

Thoracic surgery and thoracic disorders

- Left recurrent laryngeal nerve
  - at risk for stretch or compression neuropathy from cardiac or aortic dilation or manipulation.

- Ortner syndrome
  - Uncorrected mitral valve stenosis, uncontrolled congestive heart failure → dilation of the left atrium → compressive injury.

- Aortic arch aneurysm / dissection
  - Nerve passes lateral to the ligamentum arteriosum → stretch injury secondary to aortic dilation.

- Left pneumonectomy or left lobectomy: 31%

- Open heart surgery: 1~2%

Thoracic surgery and thoracic disorders

- Few cases of right recurrent laryngeal nerve paralysis secondary to open heart procedures
  - trauma from central venous catheterization, esophageal retraction, median sternotomy putting both subclavian arteries under lateral traction, direct manipulation and retraction of cardiac structures and subclavian arteries.
- Extended radical esophagectomy for cancer: 45%
- Mediastinoscopy: 0.18~0.53%
- Transesophageal echocardiography (TEE):
  - not more common.

Neoplastic etiologies

- Tumors causing paralysis:
  - Thyroid (41%), lung (30%), esophageal (20%), mediastinal (4%).
  - Pulmonary or mediastinal: 80%
  - Non-laryngeal tumors: 17~32%
  - Prognosis: poor

- Nonsurgical treatment of malignancies of head and neck.
  - NPC R/T > 7000 cGy, +/- C/T.
  - Fibrosis, loss of vascularity around nerves in radiation field.
  - Vagal nerve paralysis: 0.6~26%, 0.5~10 years

- Neurogenic tumors
  - Vagal neurilemmomas: may be asymptomatic / compensated secondary to the slow growth of these tumors.
  - Paragangliomas: usually no paralysis, but their surgery can.

Endotracheal intubation

- 7.1 ~ 11%
  - Anterior branch of recurrent laryngeal nerve was compressed between lateralized arytenoid cartilage, thyroid cartilage, and inflated cuff.
  - Secondary to viral infections triggered after local trauma, such as herpes zoster.
  - Prolonged intubation: posterior commissure stenosis.

- Must be distinguished from arytenoid dislocation.

- Recovered spontaneously within 6 months.

Viral etiologies

- The least reported and least studied.
  - Positive viral titers ≠ neural involvement.
  - HSV, VZV, EBV, influenza virus, cytomegalovirus, HIV.

- Neural edema, loss of myelin, axonal disruption.
  - Direct viral injury or immunologic response.
    - Neural involvement occurs during the acute phase.
  - Resolve in weeks to months.

- Herpes-zoster virus
  - Nerve paralysis after recrudescence of virus following endotracheal intubations.

- Herpes simplex virus
  - Damage has been identified at the ganglion or nerve.

- Influenza-related paralysis may recover.
  - 1970 Japanese: high influenza A2 viral titers in an outbreak of idiopathic laryngeal paralysis

Drug-induced etiologies

- Lead, arsenic, alcohol intoxication: toxicity to CNS.
- Injections of local anesthetics into tonsillar fossae and during carotid surgery.
- Cisplatin: only one case report.
- Vinca alkaloids (vincristine/vinblastine)
  - The best known drugs to cause recurrent neuropathy.
  - M phase of cell cycle: bind to microtubules → affect axoplasmic flow → neuronal loss
  - Frequently follows peripheral / autonomic neuropathy.
  - May be dose dependent
  - Recover 4~6 weeks after cessation of the drug.
- Organophosphorous poisoning, secondary to pesticide use.
  - Transient paralysis by inhibiting anticholinesterase at neuromuscular junction.
  - “Intermediate syndrome”: muscle weakness and cranial nerve palsies with rapid onset of breathing difficulties.
  - Complete recovery: 4~18 days if ventilatory support is given.

Miscellaneous etiologies

- Diabetes neuropathy.
- $^{131}$I ablation after thyroid cancer surgery.
- Vagal nerve stimulation for seizure control.
- Jugular vein thrombosis.
- Central venous access procedures.
- Foreign body, NG tube insertion.
- Mediastinal irradiation.
- Catheter ablation of atrial fibrillation.
- Patent ductus arteriosus ligation / embolization.
- Cervical spine osteophytes.
- Neuromuscular disorder.

# Pediatric unilateral vocal fold immobility

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Neonates (&lt;6 months of age)</th>
<th>Children (&gt;6 months of age)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital</td>
<td>Idiopathic</td>
<td>Peripheral nervous system</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Central nervous system</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td></td>
<td>Arnold-Chiari malformation</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td></td>
<td>Hydrocephalus</td>
<td>Facioscapulohumeral myopathy</td>
</tr>
<tr>
<td></td>
<td>Brain stem dysgenesis</td>
<td>Spinal muscular atrophy</td>
</tr>
<tr>
<td></td>
<td>Kernicterus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Leukodystrophy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Encephalocele</td>
<td></td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Surgical trauma</td>
<td>Surgical trauma</td>
</tr>
<tr>
<td></td>
<td>Endotracheal intubation</td>
<td>Endotracheal intubation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vincristine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vagal stimulator</td>
</tr>
<tr>
<td>Malignancy</td>
<td>—</td>
<td>Thyroid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Laryngeal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Laryngopharyngeal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lymphoma</td>
</tr>
<tr>
<td>Trauma</td>
<td>Birth trauma</td>
<td>Blunt or penetrating trauma</td>
</tr>
<tr>
<td>Inflammatory</td>
<td></td>
<td>Charcot-Marie-Tooth disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Foreign body</td>
</tr>
<tr>
<td>Infectious</td>
<td>—</td>
<td>Whooping cough</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Encephalitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Polioencephalitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diphtheria</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rabies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Syphilis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tetanus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Botulism</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tuberculosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Epstein-Barr virus</td>
</tr>
</tbody>
</table>

**ETIOLOGY OF VOCAL CORD PARALYSIS**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Unilateral VCP</th>
<th>Bilateral VCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Infrequent</td>
<td>Common (ACM)</td>
</tr>
<tr>
<td>PNS</td>
<td>Infrequent</td>
<td>Common (myasthenia gravis)</td>
</tr>
<tr>
<td>Trauma</td>
<td>Common (thoracic surgery, ETT)</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>Common (skull base tumor)</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Infrequent</td>
<td>Infrequent (Guillian-Barre)</td>
</tr>
<tr>
<td>Cardiovascular anomaly</td>
<td>Common (VSD)</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Infrequent (chemotherapy)</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Genetic</td>
<td>Infrequent</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Common</td>
<td>Common</td>
</tr>
</tbody>
</table>

VCP = vocal cord paralysis; CNS = central nervous system; PNS = peripheral nervous system; ACM = Arnold-Chiari malformation; ETT = endotracheal intubation; VSD = ventricular septal defect

Andrew L., Ronald B., Kuppersmith, Marcelle Sulek, Ellen M. Friedman, *VOCAL CORD PARALYSIS IN INFANTS AND CHILDREN*, Otolaryngologic Clinics of North America, Volume 33 • Number 1 • February 2000
# Bilateral vocal cord immobility

**Table 1. Causes of bilateral vocal cord immobility**

I. Central nervous system disease
   A. Transient ischemic attacks
   B. Arnold-Chiari malformation
   C. Hydrocephalus
   D. Meningomyelocele
   E. Syringomyelia
   F. Status post head trauma
   G. Dystonia
   H. Drug-induced dyskinesia
   I. Myoclonus
   J. Parkinson-plus syndromes (multiple-system atrophy, Shy-Drager syndrome, progressive supranuclear palsy)

II. Neuromuscular disease
   A. Myasthenia gravis
   B. Multiple sclerosis
   C. Amyotrophic lateral sclerosis
   D. Guillain-Barre syndrome
   E. Myopathy
   F. Pseudotetanus (spasmophilia, hyperventilation)

III. Local
   A. Vocal cord fixation
      1. Arytenoid dislocation
      2. Cricoarytenoid arthritis, synovitis
      3. Tumors
   B. Laryngospasm associated with asthma
   C. Laryngospasm associated with gastroesophageal reflux disease
   D. Laryngospasm caused by inflammation after upper respiratory infection
   E. Trauma
      1. External neck (blunt or sharp)
      2. Intubation

IV. Peripheral nerve injury
   A. Neoplastic
      1. Neck
      2. Chest
   B. Iatrogenic
      1. Thyroid surgery
      2. Chest surgery
      3. Neck surgery
   C. Idiopathic

V. Psychogenic
   A. Conversion disorders
   B. Malingering
   C. Psychogenic + organic neurologic disease

Bilateral vocal cord immobility

Table 2. Most common causes of bilateral vocal fold immobility: Henry Ford Hospital (1985-1991)

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma (surgical/nonsurgical)</td>
<td>19 (21)</td>
</tr>
<tr>
<td>Nonlaryngeal malignancy</td>
<td>17 (19)</td>
</tr>
<tr>
<td>Thyroidectomy</td>
<td>16 (17)</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>14 (15)</td>
</tr>
<tr>
<td>Neurologic</td>
<td>12 (13)</td>
</tr>
<tr>
<td>Intubation</td>
<td>11 (12)</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>3 (3)</td>
</tr>
</tbody>
</table>

## Stroke and hoarseness

<table>
<thead>
<tr>
<th>Voice history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of symptoms</td>
</tr>
<tr>
<td>Sudden</td>
</tr>
<tr>
<td>Progressive</td>
</tr>
<tr>
<td>Aggravating/alleviating factors</td>
</tr>
<tr>
<td>Speaking</td>
</tr>
<tr>
<td>Singing</td>
</tr>
<tr>
<td>Stress</td>
</tr>
<tr>
<td>Prolonged phonation</td>
</tr>
<tr>
<td>Associated symptoms</td>
</tr>
<tr>
<td>Odynophonia</td>
</tr>
<tr>
<td>Other neurologic signs</td>
</tr>
<tr>
<td>Other systemic medical conditions</td>
</tr>
<tr>
<td>Character of symptoms</td>
</tr>
<tr>
<td>Quality of voice</td>
</tr>
<tr>
<td>Raspiness</td>
</tr>
<tr>
<td>Breathlessness</td>
</tr>
<tr>
<td>Strain</td>
</tr>
<tr>
<td>Flow</td>
</tr>
<tr>
<td>Decreased breath support</td>
</tr>
<tr>
<td>Decreased projection</td>
</tr>
<tr>
<td>Decreased volume</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td>Vocal fatigue</td>
</tr>
<tr>
<td>Decreased range</td>
</tr>
<tr>
<td>Loss of pitch control</td>
</tr>
<tr>
<td>Voice breaks</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Upper motor neuron and lower motor neuron signs in the larynx</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Upper motor neuron</strong></td>
</tr>
<tr>
<td>Cerebral</td>
</tr>
<tr>
<td>Spastic paresis of muscle groups</td>
</tr>
<tr>
<td>Decreased agility</td>
</tr>
<tr>
<td>Basal ganglia</td>
</tr>
<tr>
<td>Resting tremor</td>
</tr>
<tr>
<td>Dystonia</td>
</tr>
<tr>
<td>Rigidity</td>
</tr>
<tr>
<td>Cerebellar</td>
</tr>
<tr>
<td>Intention tremor</td>
</tr>
<tr>
<td>Dysdiadokinesis</td>
</tr>
<tr>
<td><strong>Nonspecific</strong></td>
</tr>
<tr>
<td>Myoclonus</td>
</tr>
<tr>
<td>Chorea</td>
</tr>
<tr>
<td><strong>Lower motor neuron</strong></td>
</tr>
<tr>
<td>Peripheral neuropathy</td>
</tr>
<tr>
<td>Isolated flaccid paresis or paralysis</td>
</tr>
<tr>
<td>Decreased agility</td>
</tr>
<tr>
<td>Normal coordination</td>
</tr>
<tr>
<td>Atrophy</td>
</tr>
<tr>
<td>Neuromuscular junction</td>
</tr>
<tr>
<td>Fatigability</td>
</tr>
<tr>
<td>Fluctuating abnormalities</td>
</tr>
</tbody>
</table>

Stroke and hoarseness

Table 3
Components of voice production and their roles

<table>
<thead>
<tr>
<th>Component</th>
<th>Nerves</th>
<th>Role</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphragm, lungs</td>
<td>Phrenic</td>
<td>Source generation</td>
</tr>
<tr>
<td>Vocal folds</td>
<td>X</td>
<td>Pitch, phonation</td>
</tr>
<tr>
<td>Supraglottis, pharynx, oral and nasal cavities</td>
<td>VII, X</td>
<td>Shape, resonance, “formant”</td>
</tr>
<tr>
<td>Lips, cheeks, mandible, tongue</td>
<td>V, VII, XII</td>
<td>Articulation</td>
</tr>
</tbody>
</table>

Table 4
Indirect effects on the voice in stroke patients

<table>
<thead>
<tr>
<th>Effect</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor pulmonary reserve</td>
<td>Sedentary, poor posture, poor inspiratory effort, physical deconditioning</td>
</tr>
<tr>
<td>Poor vocal effort</td>
<td>Poor pulmonary reserve, depression, medication effects</td>
</tr>
<tr>
<td>Chronic laryngitis, hoarseness</td>
<td>Gastroesophageal or laryngopharyngeal reflux (LPRD)</td>
</tr>
<tr>
<td>Endotracheal intubation</td>
<td>Arytenoid dislocation, laryngeal/subglottic stenosis, vocal fold paralysis,</td>
</tr>
<tr>
<td></td>
<td>tracheomalacia, laryngeal edema, laryngeal granuloma</td>
</tr>
<tr>
<td>Nasogastric intubation</td>
<td>Sinusitis, LPRD, chronic laryngitis</td>
</tr>
<tr>
<td>Pooled laryngeal secretions</td>
<td>Poor swallowing effort, medication effects, poor mucosal hydration, insensate larynx</td>
</tr>
</tbody>
</table>
Before the cause is labeled idiopathic, the possibility of neoplasm must be ruled out.

Idiopathic etiologies, by definition, have no obvious causes.

- The adequacy of the workup.

- 10~27% in English literature.

- 25.9~41.3% in the Japanese literature.

- Spontaneous recovery in 24% patients.

Cases of vocal fold paralysis

Before 1970
- trauma (including surgery): 32.8%
- neoplasm: 24.2%
- Idiopathic: 22.9%
- toxic/metabolic: 8.4%
- mechanical (intrathoracic compression): 7.8%
- CNS: 3.7%

After 1970
- neoplasm: 33.4%
  - 59% of these are lung primaries;
- trauma (including surgery): 31.5%
- idiopathic: 15.5%
- medical/inflammatory: 13.2%
- CNS: 6.4%

Protocols on evaluation of all possible causes of vocal fold immobility

- indirect laryngoscopy,
- chest and skull base plain films,
- barium esophagram and laryngogram,
- CBC, VDRL, blood sugar, ESR,
- lead and arsenic levels if exposed,
- thyroid function tests, thyroid radionuclide scan and uptake,
- direct laryngoscopy with arytenoid palpation,
- bronchoscopy with washings,
- esophagoscopy.

Cases of vocal fold paralysis

- Cause of paralysis was evident at time of presentation: 57%.
  - ~1/3 can be diagnosed on the basis of history of surgery or trauma alone.
- The remaining cases requiring further evaluation: 85% were neoplasm.
- Cost-effective evaluation
  - Low diagnostic yields → not recommended
    - CBC, ESR, VDRL, viral titers, skull films, barium esophagram, thyroid scan.
  - Chest films: 54% diagnostic yield.
    - up to 73% of mediastinal masses detected by CT are not seen on plain films.

Cases of vocal fold paralysis

- No other detectable lesion:
  - contrast CT scan covering the course of recurrent laryngeal nerve from the skull base through the aortic arch is indicated.
  - no mass lesion → idiopathic.

- Palatal or pharyngeal paralysis and/or other cranial neuropathies:
  - the other branches of the vagus nerve are involved. → a lesion of the main trunk of the vagus nerve high in the neck or in the skull base must be suspected.
  - gadolinium-enhanced MRI specifically targeting the base of skull and neck.
  - If MRI is negative, HRCT of the temporal bone/skull base for bony metastasis

Cases of vocal fold paralysis

- Weakness > 1 cranial nerve, not all ipsilateral.
  - MRI of the brain.
  - Neurologic consultation:
    - multiple sclerosis, Guillain-Barre’ syndrome....
- Dysphagia, highly suspected obstruction of the esophagus:
  - Esophageal carcinoma accounts for 1.8% of vocal fold paralysis.
  - Flexible transnasal esophagoscopy, esophagram,
  - Rigid esophagoscopy
    - direct tissue visualization and biopsy.

Clinical evaluation of vocal fold paralysis

- Voice: change in sound quality, capabilities or limitations.
- Strider.

Key features of the history
- Surgery: neck, chest or endotracheal intubation.
- Other neurologic symptoms: weakness, numbness
  - Neurologic disease: multiple sclerosis or Guillain-Barre’ syndrome.
- Voice, swallowing, and breathing symptomatology.
- Onset: gradual or sudden, persistency.

Subclassification of dysphagia: ≈ the site of lesion.
- no swallowing symptoms ~ difficulty only with thin liquids:
  - recurrent nerve paresis or paralysis.
- severe dysphagia ~ nasal regurgitation:
  - pharyngeal and palatal paralysis → high vagal lesion.

Other: age, concurrent medical problems.

Examination of voice and larynx

1. Speaking voice.
   - read a couple of sentences of a standardized phonetically balanced passage at conversational volume.
   - relatively normal at one end of the continuum and quite breathy, weak, and unfocused at the opposite end.
   - supraglottic phonation in compensation.
   - obligate falsetto registration.

2. Projected voice.
   - some UVFP have functional voices when speaking relatively quietly.
   - read in projected voice, as speaking to a crowd.
   - paralytic dysphonia: fluttering diplophonic sound, fewer words per breath, lack of commensurate.
   - palatal paralysis: hypernasality, nasal air emissions.

Examination of voice and larynx

3. Vocal range.
   - go higher or lower until the limits are reached.
   - some limitation at its upper extent, reduced air wasting.

4. The vocal manifestations of forceful cough.
   - nonpercussive cough, expiratory pseudo-wheezing.

5. Maximum phonation time (MPT).
   - take a maximal breath → phonate continuously at a given pitch (fundamental frequency if possible).
   - functional measure of glottic efficiency, but is dependent on pitch and pulmonary function as well.
   - vocal fold paralysis: MPT— generally <10s, often 2~5s.

Physical examination

- cranial nerve function.
- any mass lesions or scars.
- auscultation of the neck for bruits:
  - glomus tumor.
- Indirect mirror laryngoscopy
  - gross vocal fold mobility, pooled secretions.
  - the state of the laryngopharynx at baseline, without the influence of any preparation for endoscopy.

Endoscopic evaluation

- Videoendoscopy, Rigid endoscopy, Flexible endoscopy:
  - confirm diagnosis: vocal fold paralysis, fixation, arytenoid dislocation;
  - establish the likely site of lesion: hypopharyngeal, subglottic, and tracheal masses that may invaded the recurrent nerve;
  - endoscopic correlate of radiographic imaging along the course of vagal motor innervation: soft palate, pharynx, and larynx.

- Fiberoptic scope deep within the larynx
  - supraglottic and subglottic sensation, degree of stimulation needed to cause cough or gag.
  - markedly reduced or absent sensation suggests involvement of the superior laryngeal nerve.

Videoendoscopic swallowing study (VESS).
- Spray of 2% tetracaine into one nostril, flexible nasopharyngoscope.
- Upper surface of soft palate, symmetry of palatal elevation and lateral pharyngeal wall movement,
- Vocal tests of palatal function: velopharyngeal seal.
- The condition of pharynx and hypopharynx.
- Pharyngeal contraction: phonate at high pitch → symmetric horseshoe-shaped contraction with bulging of lateral and posterior pharyngeal wall.
Vocal folds evaluation

1. Rest position. (midline, paramedian, intermediate)
   - not correlate with the lesion location.
   - not correspond to the degree of vocal impairment.
   - atrophy and flaccidity of the vocal fold, over-adduct the opposite fold, constrict the larynx.

2. Position and direction of the vocal processes in relation to each other.

3. Symmetry of vocal fold contour.
   - asymmetric atrophy, more capacious ventricle, conus elasticus below the vocal folds.

4. Evidence of tissue loss (arytenoid divots) or synechiae in the posterior glottis.

5. Scarring along the cricoarytenoid joint capsules.
   - injury from prolonged intubation.

6. Subglottic and tracheal granulation, scarring, or stenosis.
   - increase suspicion of joint ankylosis.

Vocal folds evaluation during phonation

1. Mobility of the membranous part of the vocal folds as compared with the body and apex of the arytenoids.
   - Significant arytenoid apex movement without movement of the vocal folds proper suggests fixation caused by joint injury.

2. Glottic closure.
   - Incomplete: tissue loss/scarring or neurologic.
   - Limited mobility of vocal processes: rotate, medialize.
   - Vibrating mucosa may give the illusion of complete glottic closure.
   - Limited arytenoid adduction: cricoarytenoid joint ankylosis, sometimes arytenoid tissue loss → postintubation phonatory insufficiency.

3. Level of the match between vocal processes.
   - Vocal processes on the same horizontal plane → the arytenoids cannot have been dislocated.

4. Flaccidity of vocal fold structures.
   - Multiple brief phonations low in the range → lateral buckling.

5. Lesions in the trachea or bronchi, or a trail of blood.
   - A small trail of blood may be expected when the cricothyroid membrane puncture has been used.