Secrets of Neurolaryngology

LISTEN CAREFULLY
LOOK CLOSELY

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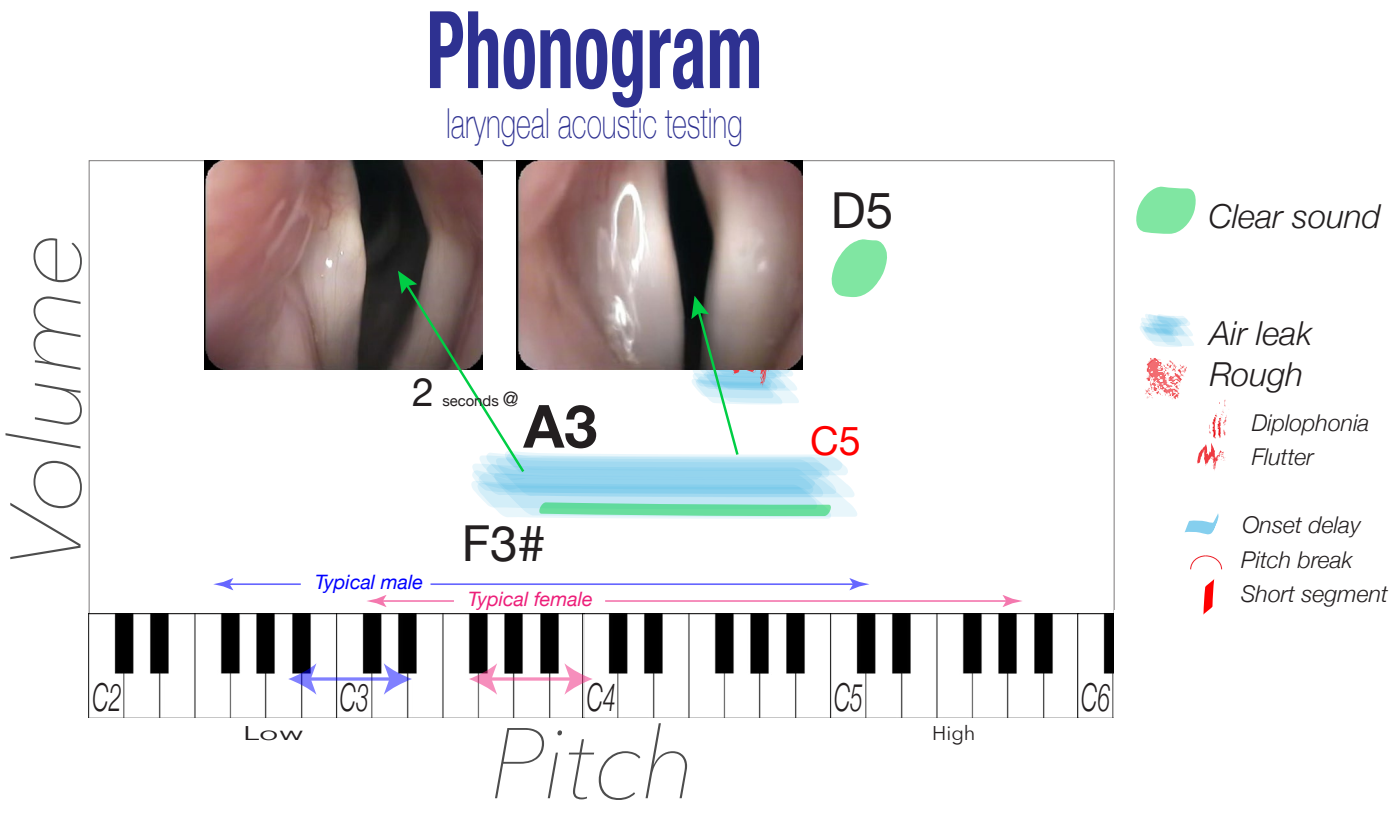

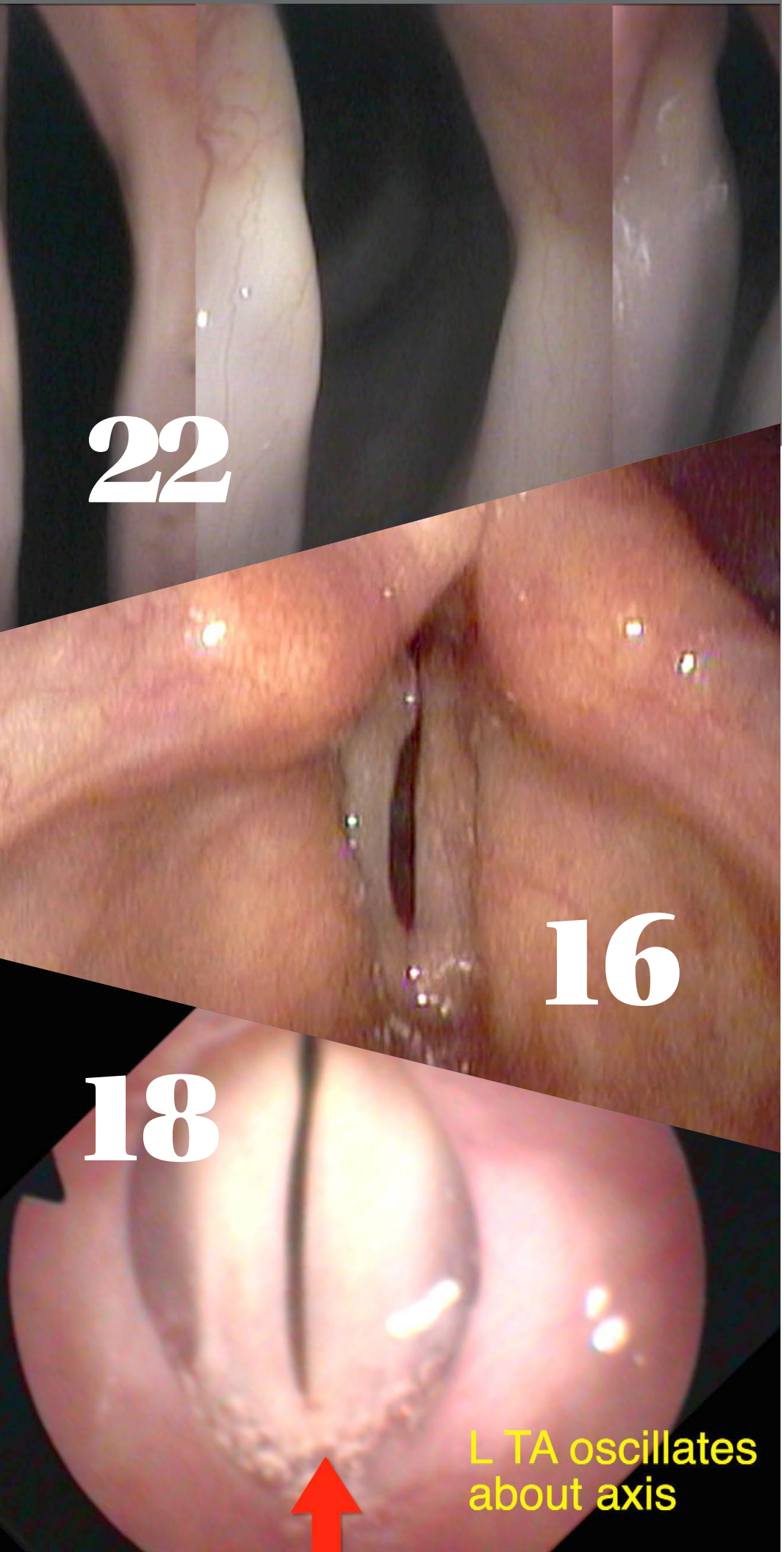

SUMMARY

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Visual Neurolaryngology

Becoming a visual neurologist of the larynx takes time to record motion, then observe and reason about the functions of each muscle of the larynx. It can be complex.

There are three muscles on each side of the laynx innervated by the recurrent laryngeal nerve and one muscle on each side innervated by the superior laryngeal nerve and one essentially midline muscle, so we could say there are 9 muscles of the larynx. If there is only a binary on and off function possibility for each muscle, then in theory we have a possibility of 9^2 possible configurations of the larynx or 81 different positions that the vocal cords could assume. Effectively there may be less than this because the muscles usually work in harmony on either side of the larynx.

However, after an injury, a muscle might just be weak and function partially, so now we have at least three possible configurations for each muscle. No innervation, partial innervation, complete innervation, so we could say 9^3 possible visual configurations of the larynx or 729 different configurations.

The nerve supply to the larynx is quite robust and after a nerve injury, nerve fibers will typically regrow back to the muscle(s). In this process of regrowth, nerve fibers from the brain bundled together may route input to a different muscle. Posterior Cricoarytenoid (PCA) brain fibers might grow to the Lateral Cricoarytenoid (LCA) muscle leading to LCA muscle contraction during inspiration, a very inappropriate time to contract.

Now each muscle might have at least 4 states: no function, normal function, partial function, function at an inappropriate time. We have at least 9^4 or 6541 different possible configurations. This number of possible laryngeal configurations is potentially very confusing to the examiner.

Consequently, an astute examiner benefits from focus and simplification. A video recording over several cycles of a function, including respiration and then phonation represents a combination of tools and techniques allowing this focus. Reviewing vocal cord motion slowly and repetitively, looking at one function repeatedly and comparing side to side, a pattern will usually become discernable. The same recording can be reviewed again looking at a different function or different area of anatomy.

Reviewing motion of different portions of the larynx separately will lead to an understanding of when the muscles, and hence nerves, are firing. You will develop a visual understanding of the neurophysiology of the larynx. Here are a few secrets for visually winnowing the complexity of movement down to a specific diagnosis.

Secrets of a neurolaryngeal examination

Observation

allow movement to happen repetitively.
isolate laryngeal functions.

FUNCTIONS
- respiration
- phonation

RESPIRATION
- inspiration
- expiration

PHONATION
- high pitch
- low pitch

then

- high pressure/flow/volume
- low pressure/flow/volume

Visual States

STABLE
- injury
  - paralysis
  - paresis
- recovery - reinnervation
  - normal
  - synkinesis
  - dyskinesis

UNSTABLE
- tremor
- spasm

MIMIC
- fixation
  - mucosal scar
  - joint ankylosis

Techniques

GET CLOSE
- topical anesthesia
  - improves view
  - doesn't impair movement

OBSERVE REPETITIVELY
- record multiple repetitions
- sniff to stretch cords
- lift arytenoids
- scope between false cords
- multiple perspectives

Visual diagnosis of laryngeal neurologic impairments is possible and, is a very accurate method for evaluating the neurologic status of the upper airway.

Findings

- Atrophy - TA, PCA, False cord
- Fasciculation - TA body, PCA body, LCA arytenoid
- Motion
  - Range of rotation - LCA, PCA (joint, scar)
  - Lengthening - CT (SLN, joint, scar)
- Oscillation - TA, CT
  - Compare high and low pitch
- Hyperactivation - LCA

Tools

- Endoscope
- Stroboscope
- Record audio & video
- Video editing software

EMG

Although EMG (electromyography) is commonly spoken of as the "gold standard" (if there even is one), visual examination offers a great deal - let's say a platinum standard.

Assumptions

Muscle action

- each muscle has only a single action,
- that action can be isolated,
- that action can be elicited and visualized.

Timing

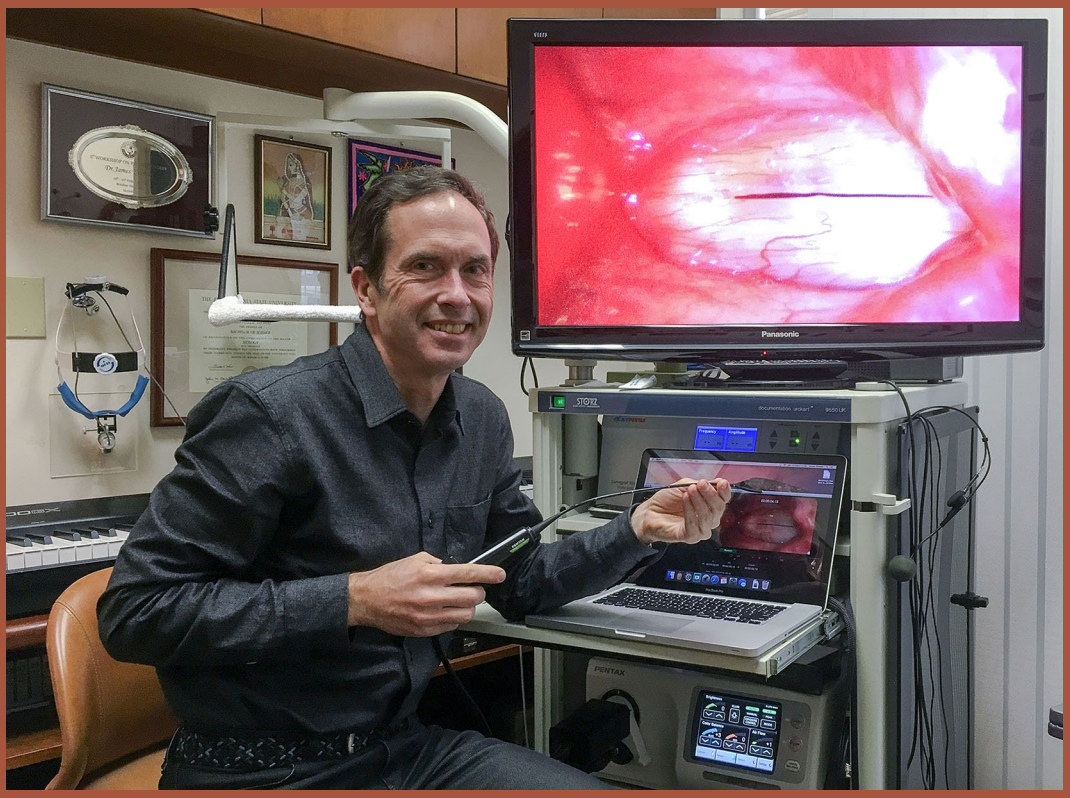
- each muscle has an appropriate time to contract,
- timing can be compared to the opposite side,
- timing can be compared to expected function,
  - inspiration, expiration, phonation
- inappropriate timing represents reinnervation by an opposing nerve branch.

Compensation

- the patient always attempts to compensate,
- removing compensation reveals pathology.

Recovery

- Degree of neural injury and patterns of regrowth determine timing of muscle actions.



VISUALIZING

Neurolaryngology: VISUAL EXAM

Careful listening
&
close observation
are precise neurolaryngeal
diagnostic methods,
and may supplement or replace
laryngeal EMG.

INTRODUCTION

The branching tree structure of the nerve

The first step in visual neurolaryngology is for the examiner to have a mental map of laryngeal anatomy. Understanding the treelike branching of the laryngeal nerves will allow us to work both forwards and backwards from clinical information we have. If we know where and how an injury took place along the path of the nerve, then we will know the findings and impairments that we expect see on an endoscopic examination. Likewise we can work in the reverse direction. Based on the functional impairments that are seen on an endoscopic examination, we should be able to predict where along the path of the nerve the injury took or is taking place.

The larynx is supplied by the Xth cranial nerve, the vagus nerve. There are two important anatomic locations along the path of this nerve for the examiner to think about.

The first location is where the Xth cranial nerve leaves the skull and heads towards the neck. It passes through a narrow opening along with two other cranial nerves, the XIth and the XIIth cranial nerves. From a diagnostic perspective, this bottleneck then is a location where an problem will likely affect not only the Xth cranial nerve but also the XIth and the XIIth cranial nerves. So in our endoscopic upper airway examination, if there are problems with the tongue (XIIth cranial nerve) or with lifting the arm and shoulder up (XIth cranial nerve), in addition to problems in the larynx, we will know to focus our attention on this narrow opening at the base of the skull.

The second diagnostically important location is where the Xth cranial nerve descends through the neck, giving off four branches to supply the throat and larynx. Knowing which of these branches are affected, will tell us how high the location of the injury is in the neck. If only single branch of the nerve is involved, we know the injury is taking place very near to the larynx. If multiple branches are involved, we know the injury is taking place further away from the larynx and closer to the brain.

The extended path of the left recurrent laryngeal branch of the Xth cranial nerve descending into the chest and wrapping around the aorta and left bronchus while the right typically remains recurrent in the lower neck is one of the most well known diagnostic asymmetries.

Since speech and voice overlap in function, assessment of the tongue, soft palate muscles and pharyngeal constrictors contribute to understanding laryngeal neurologic injury.

INTRODUCTION

Injuries & redundancy

A second principle of neurolaryngology is that the Xth cranial nerve, when injured, almost always tends to regrow and re-supply muscles with nerve input. Mild injuries and injuries close to an individual branch of the nerve will tend to recover with normal function.

More severe injuries and more proximal injuries, that is, injuries that are closer to the main trunk of the nerve, will result in regrowth with nerve pathways crossing to different branches. This will result in various degrees of mixed up function for the muscles of the larynx. Even in cases where there is a gap between the cut ends of the nerve, the cut neurons will often gradually grow through the intervening tissue and reconnect with the denervated muscles.

BILATERAL SYMMETRY

In many injuries of the larynx, we have a built in reference point. When one laryngeal nerve is injured, the other side continues to function normally and provides the examiner with a direct comparison by way of asymmetry in anatomy and function between the two sides. We can compare the structure on the left with the structure on the right. We can also compare the timing and range of movement on one side with the other.

COMPENSATION

There is redundancy in the muscles of the larynx. The supraglottis can constrict aiding closure when intrinsic muscles are weakened. This can bring the true vocal cords closer together although the supraglottic squeeze fatigues quicker.

There is more than one muscle to tension the vocal cords and raise pitch – thyroarytenoid and cricothyroid. Only the superior laryngeal nerve may be injured and the recurrent nerve compensates. When the recurrent nerve is injured, the superior nerve compensates.

When a branch on one side of the neck is injured, muscles from the branches on the other side help to make up for the loss. When one side is injured,compensate. Unilateral overcompensation fatigues quicker than normal.

Muscles

Some of the muscles innervated by the laryngeal nerves are so close to the surface mucosa that they can be nearly directly visualized. Others are deeper and only their effects can be visualized. Near direct visualization includes the ability to see the muscle contract and thicken or shorten. Atrophy and fasciculations can be visualized beneath the mucosa.

Indirect visualization is the ability to infer muscle contraction from joint and structure movement.

Some activities, such as respiration, are rhythmic and symmetric. Inappropriate timing and asymmetric motion can be utilized to infer innervation status.

Muscles observed during a neuroendoscopic evaluation:

- lingual muscles - genioglossus
- palatal muscles - levator veli palatini
- pharyngeal constrictors
- laryngeal muscles
  - thyroarytenoid (TA)
  - lateral cricoarytenoid (LCA)
  - posterior cricoarytenoid (PCA)
  - interarytenoid (IA)
  - cricothyroid (CT)

Near direct visual findings in the larynx include the following (along with the muscles in which they can be endoscopically observed):

Atrophy - TA, PCA
Fasciculation - TA, PCA

Indirect visual findings utilized to infer muscle function include the following (along with the muscles in which they can be endoscopically observed):

Oscillation - TA, CT
Tension - TA, CT
Range of motion - LCA, PCA
Vocal process resting postion - LCA, PCA
Lengthening - CT, PCA
Respiration
- Inspiration - PCA
- Expiration - LCA

Timing - inappropriate reinnervation
Compensation - unmasking needed

Visual findings

- Atrophy - TA, PCA, False cord
- Fasciculation - TA - body, PCA - body, LCA - arytenoid
- Motion
  - Range of rotation - LCA, PCA (joint, scar)
  - Lengthening - CT (SLN, joint, scar)
- Oscillation - TA, CT
- Hyperactivation - LCA, TA

Motion - Movement
slow & fast

Contraction --> movement

SLOW MOVEMENT

MUSCLE	EFFECT	ACTION	OBSERVE
TRANSLATION			
PCA	**ABduction**	Opening glottis	vocal process / posterior larynx
LCA (& TA - IA)	**ADduction**	Closing glottis	vocal process
CT	**tension (extrinsic)**	Elongate vocal cord	vocal process / anterior commissure

FAST MOVEMENT

OSCILLATION			
TA	**tension (intrinsic)**	oscillation rate	vocal margin amplitude
CT	**tension (extrinsic)**	oscillation rate	vocal margin amplitude
FLUTTER			
no muscle	flaccidity	flutter, Bernoulli effect	membranous margin

Larynx motion appears to be complex, at least on superficial examination. Some of the apparent complexity in reference articles derives from failure to discriminate among the various types of motion. I frequently read the statement, "The vocal cords don't move." I don't know whether the speaker is referring to the slow, translational movement of abduction and adduction or rapid, oscillation impairment (perhaps from intrinsic stiffness of the vocal cord). They usually are not referring to lengthening of the vocal cord, which is also a movement.

When I read in a report, "The vocal cords don't move," I also suspect that there is no distinction being made between a lack of abduction / adduction from:

- a lack of muscle contraction vs.
- inappropriate or simultaneous attempted abduction and adduction leading to no effective movement or only subtle translational movement

Additionally this subtle movement may be reduced but:

- appropriate or inappropriate in direction,
- appropriate or inappropriate in timing.

Impairment

Paralysis

Complete loss of voluntary movement

Paresis

Partial loss of voluntary movement. Normal timing and direction of motion

Crossed reinnervation

Synkinesis

Simultaneous stimulation of two muscles that cancels effective movement

Dyskinesis

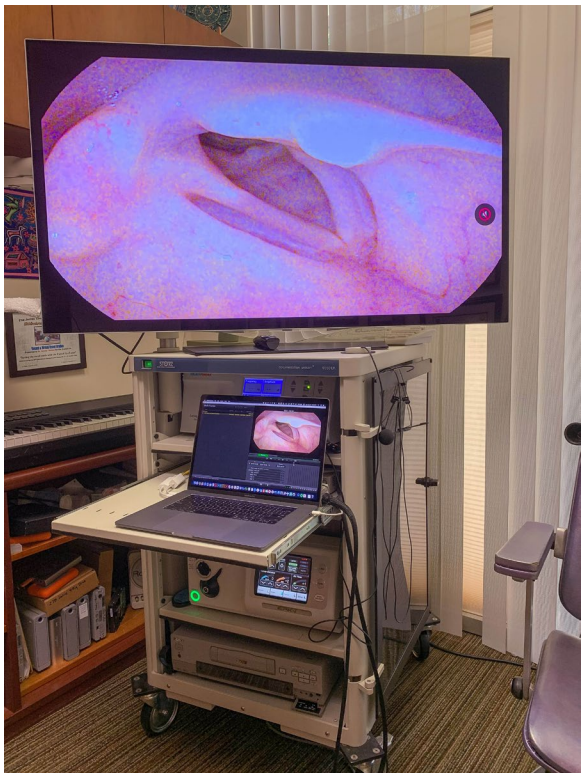
Contraction of a muscle leading to inappropriate timing of movement.

Paralysis may be the most frequently used term, yet it is the rarest condition. Shortly after a complete transection of the nerve is almost the only time I can be confident of a complete paralysis.

Paresis is a relatively common condition in which a muscle fails to completely contract. There is partial and appropriate motion.

Given the high incidence of nerve regrowth, synkinesis and dyskinesis are frequent findings.

Tools



Tools

- Video endoscope
- Digital recording
- Topical anesthesia
- Close exam
- Vocal manipulation

VIDEO ENDOSCOPE

The endoscope, video processor, and stroboscope are likely in almost every laryngology office. So too is some recording device, though too often the audio portion of the recording is left out.

A high-definition chip-on-tip endoscope improves visual quality greatly over fiberoptic scopes.

TOPICAL ANESTHESIA

Topical anesthesia is essential in some cases to see beneath the arytenoids or between completely compressed false vocal cords. Topical anesthesia with lidocaine does not alter vocal cord muscle motion. It only eliminates sensation temporarily, typically long enough to perform a detailed, close-up examination.

DIGITAL RECORDING

A digital recording device may also be common although some recording configurations are more difficult to use than a typical laptop with standard video software.

The ability to look at a video clip;

- repetitively,
- in varying degrees of slow motion,
- in forward and reverse,
- frame-by-frame and,
- AI software can slow video, remove recording artifacts, stabilize examiner unsteadiness and increase clarity of vasculature.

CLOSE EXAM

Close examination can sometimes be performed carefully during quiet respiration. However, true close examinations during phonation often require topical anesthesia.

Topical lidocaine, applied to the vocal cords allows the endoscope to lift the arytenoids out of the way and view the vocal processes in nearly all situations. The vocal process is the key guide to LCA and PCA muscle movement.

Topical lidocaine allows obstructing false cords to be moved laterally and true cord oscillation visualized directly in situtations of vocal weakness.

VOCAL MANIPULATION

Vocal manipulation is an incredible yet underutilized tool. Eliciting high and low pitch as well as high and low air flow assesses the function of the laryngeal muscles in different configurations of neural stimulation. Changes in position of the vocal cords and in the pattern of oscillation reveal weakness.

Observation

Take time

Observation

take time to record several repetitions of movement

FUNCTIONS

RESPIRATION

- inspiration
- expiration

PHONATION

- high pitch
- low pitch

then

- high pressure/flow/volume
- low pressure/flow/volume

Making a recording

It is easy to go for the gold. Spray the nose, put the endoscope in and look at the vocal cords for a lesion. Often the examiner's emphasis is on structure. However, laryngology is about function and structure plays one role in that function.

Photos primarily reveal structure. Observing function requires video. Value lies in observing the movement of structures over time and various tasks.

Although neurolaryngology exams take time, that time can be used efficiently. During the general ENT portion of the exam, as soon as one sees which nasal passage is more open, spray a mixture of a decongestant and topical anesthetic into that nasal passage. By the time the general exam is finished and patient's vocal capabilities recorded, the nose is well anesthetized. Patients are amazed how comfortable this exam is compared to a quick look endoscopy.

Taking time extends to the related cranial nerves. Before looking at the vocal cords, assess the movement of the tongue, soft palate and pharynx so they are not forgotten if there is obvious laryngeal nerve impairment.

Next, with the entire larynx in view, think about recording several cycles of function rather than "taking a picture" of the larynx. Recording several cycles of breathing seems like an eternity while recording, but when reviewing videos, 5-7 cycles of quiet respiration is quite minimal. And there is value in quiet respiration as well as a recording of active, deep respiration.

If one cannot see the vocal processes on an endoscopic overview, move the endoscope closer and again record several cycles of respiration from the vantage point of watching the movement of the vocal processes.

The same applies to stroboscopy. Recording a single pitch misses too much information. The patient is likely compensating for a problem at the chosen initial pitch. Record a range of pitches. Record a range of volume. Record both loud and soft sound production at low pitch and then loud and soft again at a high pitch. Recording a glide from low to high pitch and a glide from high to low pitch reveals additional information.

The ultimate goal is to record vocal cord vibration at the most impaired vocal quality, the "hoarse voice" that the patient is complaining about. The exam is not a test of how well the patient can sing, rather you want to capture the sound and image where sound is poorest in quality.

Endoscopy exam recordings typically range from about 40 seconds up to 4 minutes for more subtle problems. Stroboscopy exams are also typically 40 seconds up to 3 minutes of recording.

Reviewing the recording

At times, reviewing the same motion multiple times is warranted. Breaking down respiration into components will yield additional clues. For example, focus on inspiration only for several cycles. Then review the same recording of breathing cycles focusing on expiration only. Return and review just the right vocal process movement several times, sorting out range of motion as well as timing and comparing it to the opposite vocal process for range of motion and timing of motion when they are not synchronous. This component review of the video takes additional time, but reveals otherwise missed details.

After reviewing numerous recordings the examiner learns that several cycles of respiration allow better review of motion later. Recording a range of pitches during stroboscopy provides more detailed information. Consequently, on future examinations more details will be recorded.

FINDINGS

XI & XII cranial nerves
tongue
shoulders

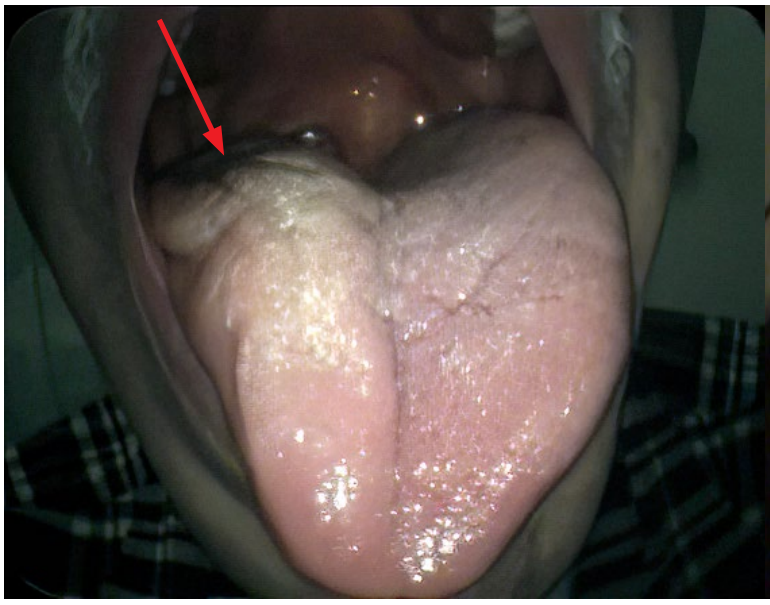
Tongue

CRANIAL NERVE XII

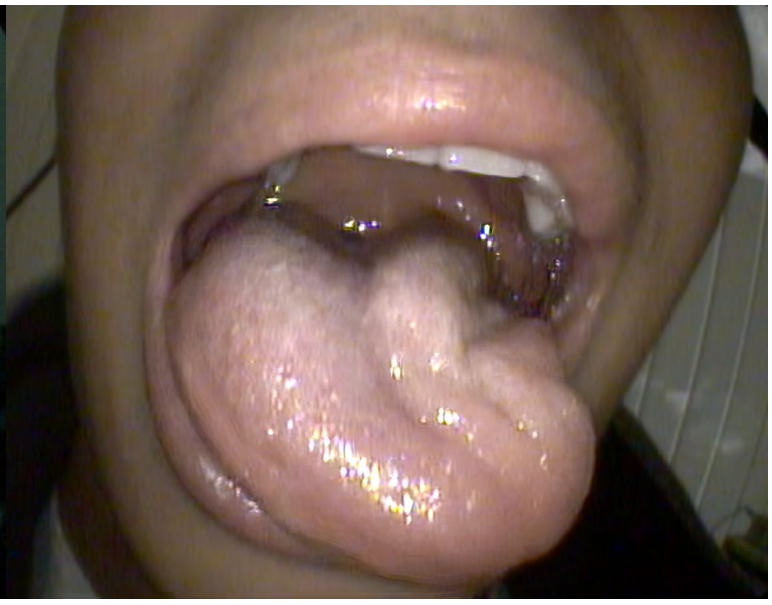
(SUPPLIES PALATAL LEVATOR)

ORAL VIEW OF TONGUE

- Observe for unilateral atrophy, fasciculations
- During tongue protrusion, the tip will deviate toward the weak side



Atrophy of the right tongue (arrow points to right side)



Protrusion of tongue – tip moves toward weak left side

Xth Cranial nerve palatal branch

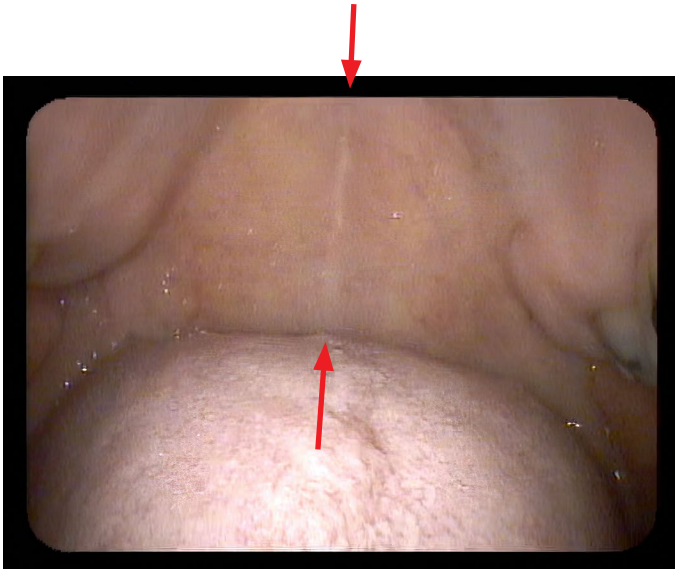
Palate

UPPER GANGLION BRANCH

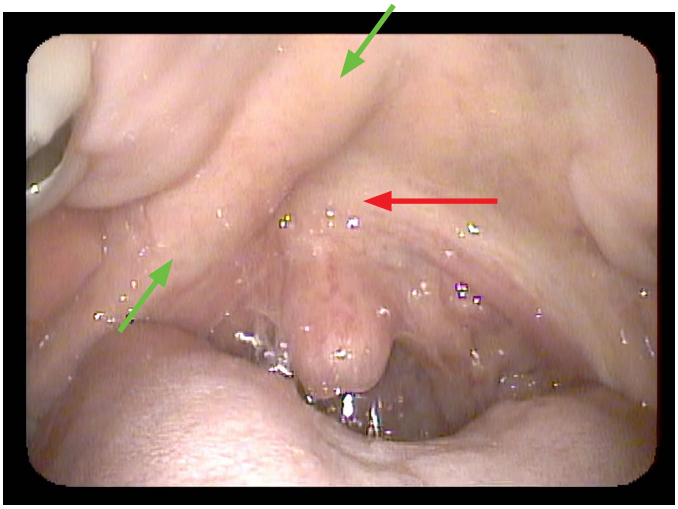
(SUPPLIES PALATAL LEVATOR)

ORAL VIEW OF PALATE

- Observe palate midline during /ah/. A lateral deviation is from weakness.
- Observe lateral indentation of soft palate during /ah/ from muscle contraction.



Median raphe of soft palate resting in midline (between arrows)



Contraction of only the right levator veli palatini muscle (green arrows). This pulls the uvula and the median raphe of the soft palate toward the right side (red arrow). The left side of the palate is paralyzed.

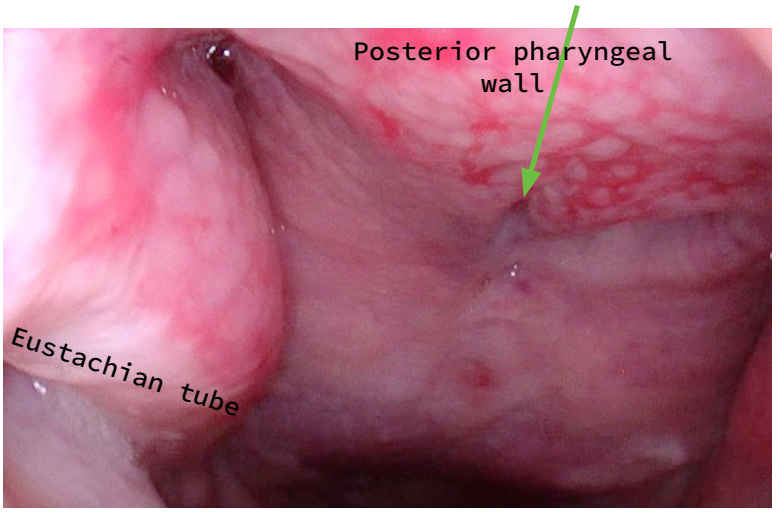
Xth Cranial nerve palatal branch

UPPER GANGLION BRANCH (SUPPLIES PALATAL LEVATOR)

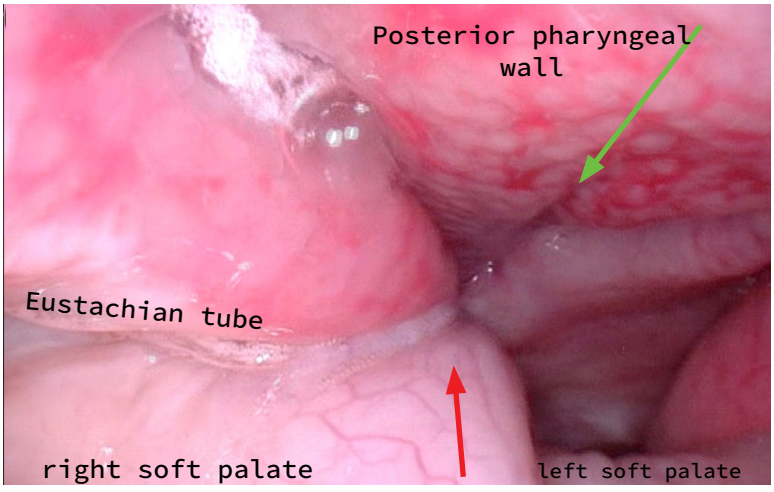
NASAL VIEW OF SOFT PALATE

- Both sides can usually be seen from one nasal passage
- Normal elevation is complete closure without air leak during plosives /pa/, /ka/, /ta/ and fricatives /sh/, /th/
- Incomplete closure represent levator veli palatini muscle weakness
- One side lack of complete elevation leads to a gap (lower photo) - a view from over the middle turbinate is most accurate
- Bubbles from secretions during phonation - plosives and fricatives is from weakness

Identifying palate weakness when it occurs simultaneously with recurrent laryngeal nerve weakness is important for the patient to realize the extent of injury - often the loss of voice is most obvious injury and may hide symptoms of nasal leak. If the voice is restored by some procedure, such as an injection augmentation, then the nasal air leak suddenly becomes very audible.



Endoscopic view of the nasopharynx from over the right inferior turbinate within the right nasal cavity. Posterior pharynx midline is at green arrow. The right Eustachian tube is visible. The soft palate is below the frame.



Endoscopic view of the nasopharynx from over the right inferior turbinate within the right nasal cavity during a fricative sound. The right soft palate has elevated and sealed against the eustachian tube and the posterior pharyngeal wall (red arrow). The left soft palate fails to elevate and leaks air up into the nose.

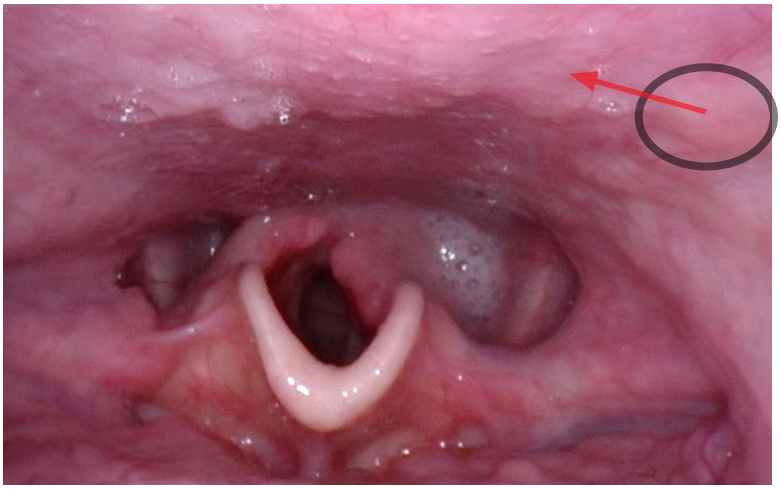
Xth Cranial nerve pharyngeal branch

Supra-laryngeal branches

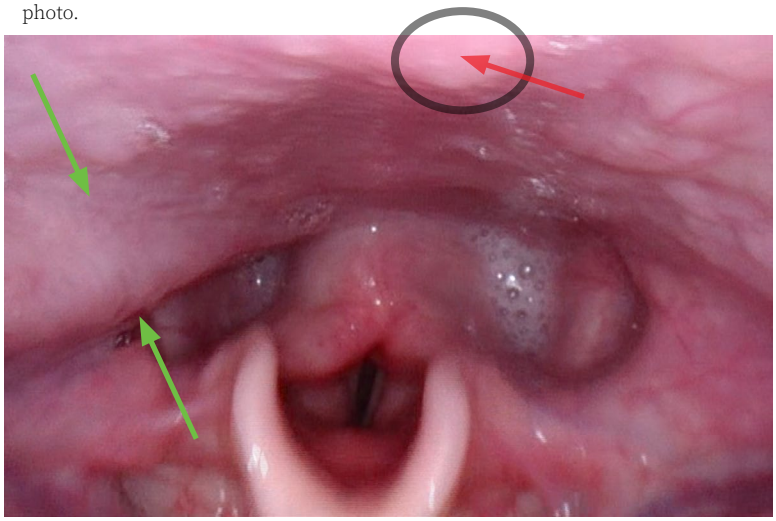
LOWER GANGLION BRANCH (SUPPLIES PHARYNGEAL CONSTRICTORS)

Position endoscope in upper pharynx, observe midline during a vocal pitch glide from a low pitch to a high pitch (or vice versa)

- Observe midline raphe which pulls toward strong side if there is injury
- Observe lateral pharyngeal squeeze from constrictors
- Pooling / open piriform sinus coincides with weakness as it tends not to empty during swallowing when the pharyngeal constrictors are weak.



Endoscopic view of the hypopharynx. Posterior pharynx is relaxed. There are pooled secretions in the left piriform sinus. Follow the circled gland in the next photo.



Endoscopic view of the hypopharynx at high pitch. Only the pharyngeal constrictor muscle on the right contracts (green arrows). There is no contraction present on the left. Secretions remain pooled in the left piriform sinus. The circled gland in the upper photo has been pulled over toward the midline from the right sided muscle contraction.

Superior Laryngeal Nerve
Cricothyroid muscle

SYMPTOMS

- Loss of power
- loss of upper range
- Loss of vocal control during singing (example: loss of easy pitch matching)

HISTORY

- frequently injured during neck surgery (thyroid, parathyroid, anterior cervical fusion)

DIFFERENTIAL DIAGNOSIS:

- Fixation vs nerve impairment: Bilateral SLN injury is relatively uncommon and symmetric bilateral motion impairment often represents scar or fixation rather than neurologic impairment

FINDINGS

VOCAL CAPABILITIES BATTERY:

- Limitation of the extent of upper pitch range compared to expected range
- Flatting and poor control in the upper vocal pitch range

ENDOSCOPY

- Vocal cord length does not change during attempted glide up in pitch in bilateral injury.
- vocal cord length changes minimally, less than expected in unilateral injury

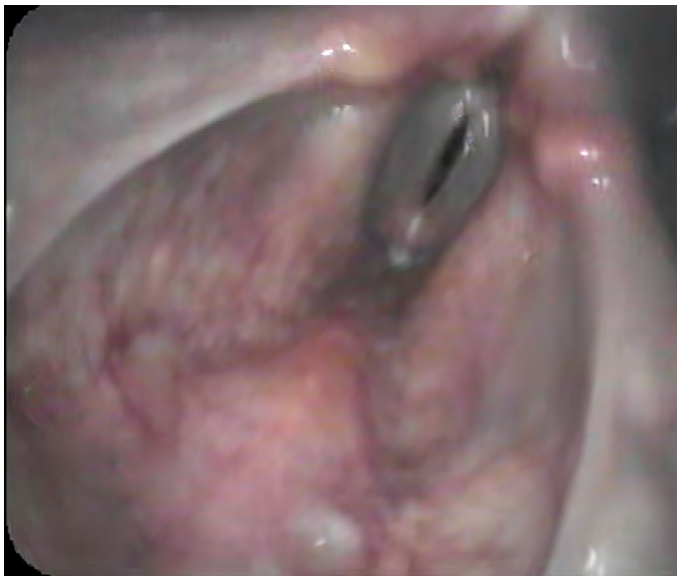
STROBOSCOPY:

- Symmetric oscillations at low pitch while
- Abnormal findings at high pitch (unilateral superior nerve injury)
  - Asymmetry of vocal fold vibration at high pitch present because differential tension is most evident when only one cricothyroid muscle contracts.
  - At high pitch the injured (less tense) cord oscillates lateral to it's axis while the normal cord oscillates symmetrically about it's axis
  - At phonatory onset and offset the weak cord has a central glottic gap

BILATERAL SUPERIOR LARYNGEAL NERVE IMPAIRMENT

51 year old male lost his voice 5 months ago after a total thyroidectomy. His voice gradually returned, but he now lacks power, notices early vocal fatigue, his voice doesn't seem as clear, his speaking voice feels lower in pitch and he can no longer reach any high notes.

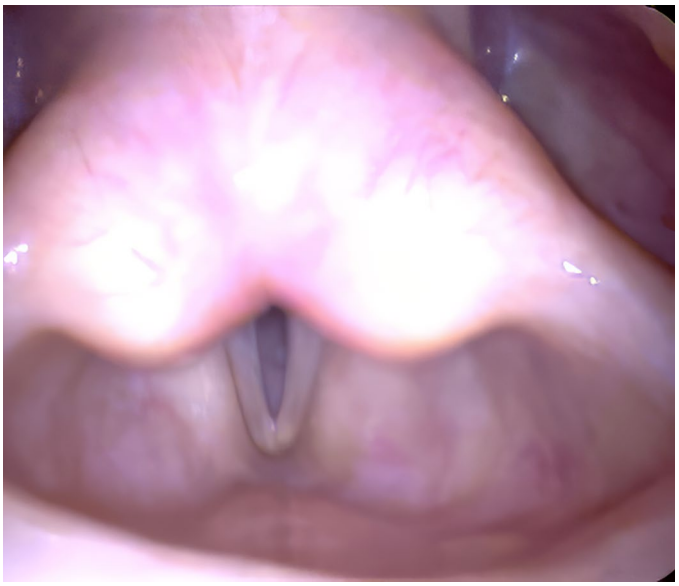
Vocal cords during stroboscopy. Open phase at low pitch of G2. Vocal cords are oscillating symmetrically. Note length of the vocal cords.



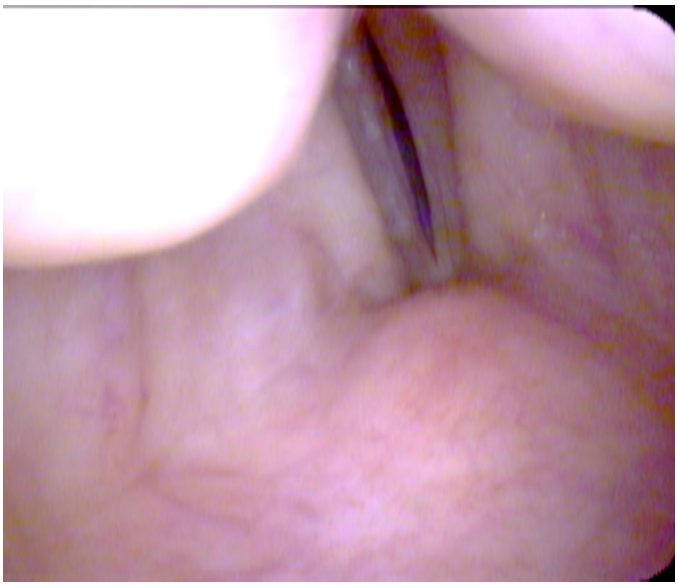
Vocal cords during stroboscopy. Open phase at his highest pitch of G3. Vocal cords continue to oscillate symmetrically. Their length has not changed and he should be able to reach at least an octave higher than this. Likely both superior laryngeal nerves were injured during the total thyroidectomy.

UNILATERAL SUPERIOR LARYNGEAL NERVE IMPAIRMENT

69 year old female with gradual vocal impairment noticed over about 4 years, affecting her singing voice quality.



Stroboscopic view during open phase of vocal cords in her lower range at a pitch of G3. Both cords oscillate symmetrically while likely only the thyroarytenoid muscle is active.



Stroboscopic view during open phase of vocal cords two octaves higher at a pitch of D5. The vocal cords are longer. The left cord oscillates much further laterally than the right suggesting that the left cricothyroid muscle is weak and cannot stretch the left cord as tight as the right.

Recurrent Laryngeal Nerve
Thyroarytenoid muscle

SYMPTOMS

- weak voice (hoarseness)
- delayed: reduced vocal range

HISTORY

- weak voice immediately after surgery (thyroid, parathyroid, carotid, esophagus, left chest surgery)
- cold symptoms with prolonged loss of voice

DIFFERENTIAL DIAGNOSIS:

- Bowing may mimic symptoms

FINDINGS

VOCAL CAPABILITIES BATTERY:

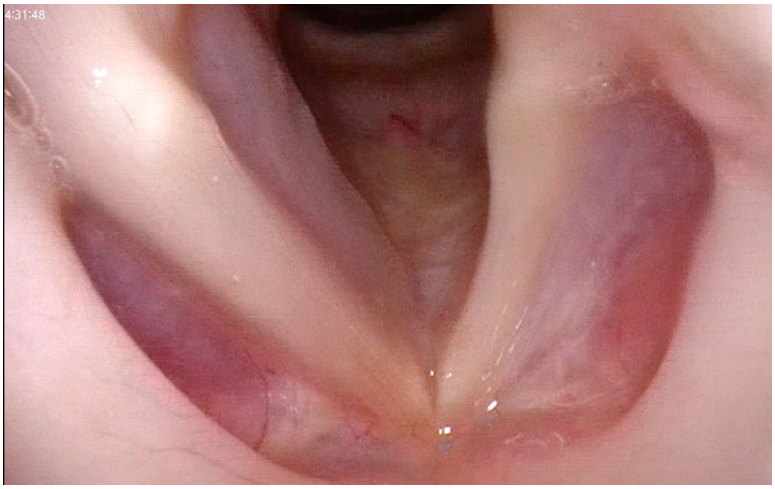
- loss of lower vocal range (CT provides some tension for upper range so less air leak at high pitch)
  - obligate falsetto - high comfortable speaking pitch
- white noise more apparent in lower range
- diplophonia in lower range from asymmetric true cord mass, asymmetric true cord tension
- delayed findings: may recover some tension from crossover nerve input

ENDOSCOPY

- focus on membranous true vocal cord
- bowing - rigid scope
- atrophy - flexible scope best for visualization
  - sometimes requires topical anesthesia to view vocal cord thickness or mass
  - Sniffing stretches the vocal cords and augments subtle atrophy.
    - Slow motion helpful for viewing thinning secondary to sniffing
    - Phonation first, followed by sniffing also augments thinness.
  - Can infer atrophy from enlarged ventricle
  - Inspiratory bernoulli effect if atrophic and if near opposite vocal cord
- fasciculations (visible on superior vocal cord surface as well as in subglottis)
- lack of tension

STROBOSCOPY:

- TA weakness is augmented by lower pitch
  - Low pitch removes cricothyroid compensation in partial paresis
  - If supraglottic squeeze is predominant only at low pitch - implies glottic incompetence more often than intentional hyperfunction
    - Topical anesthesia allows passage of endoscope between false cords for true cord view during phonation
- Oscillation
  - Low pitch
    - oscillates lateral to axis
    - Excursion amplified at low pitch
  - High pitch
    - CT keeps cords near each other
    - CT keeps cords in phase or almost in phase (patients tend to avoid diplophonia - especially during an exam)
- Augment abnormal vibrations by increasing subglottic pressure (volume and airflow). Visualized as
  - Flutter - random oscillation, or
  - Biphasic - one central node of oscillation with two segments



Close up exam reveals severe atrophy of the left thyroarytenoid muscle.

UNILATERAL RECURRENT LARYNGEAL NERVE IMPAIRMENT

73 year old female with sudden vocal impairment after total thyroidectomy 5 months ago.

Stroboscopy mid-phase at low pitch G3. Left cord is much further lateral from lack of tension.

Stroboscopy mid-phase at higher pitch G4. Left cord is still lateral from reduced tension, although the cricothyroid muscle pulls both cords closer to the midline as they stretch tighter..

Recurrent Laryngeal Nerve
Lateral Cricoarytenoid muscle

SYMPTOMS
- weak voice (breathy hoarseness)
- delayed: laryngospasms

HISTORY
- weak voice immediately after surgery (thyroid, parathyroid, carotid, esophagus, left chest surgery)
- cold symptoms with prolonged loss of voice

DIFFERENTIAL DIAGNOSIS:
- Bowing may mimic some symptoms
- Nonorganic dysphonia
- delayed: dyskinetic reinnervation may be confused with asthma

FINDINGS

VOCAL CAPABILITIES BATTERY:
- loss of lower vocal range (CT aids closure in upper range so less air leak at high pitch)
- elevated comfortable speaking pitch
- reduced maximum phonation time

ENDOSCOPY
- focus on vocal process position and orientation
- watch respiration for reduced range of motion (ROM) between inspiration and expiration
- delayed finding: vocal process midline, but immobile
- delayed finding: vocal process crosses midline from PCA innervation

ENDOSCOPY OR STROBOSCOPY
- watch vocal process closure during phonatory onset (adduction) in slowed down motion for speed of closure and range of closure.
  - view multiple closures to differentiate from nonorganic muscle tension where closure will be variable and intermittently complete
- Upper portion of arytenoid will often cover view of vocal process at end of closure, earlier if the PCA is out on the same side. Low speed video review very valuable.
- topical anesthesia greatly enhances view of posterior commissure during phonation - best view
- Observe for triangle of lateralized vocal process on weak side. Be cognizant of hyperclosure from opposite cord's vocal process
  - View at varying pitch (high, medium and low pitch)
  - lowest possible pitch reveals largest posterior gap triangle.
- with increased volume, arytenoid can be pushed laterally at low pitch in weak LCA, which partly corrects with pitch elevation as CT contraction compensates at higher pitch

STROBOSCOPY:
- no obvious oscillatory change unless other muscles are paretic. TA & LCA are frequently paretic, synkinetic or dyskinetic together

OTHER NOTES:
- View of rigid exam with small "apparently symmetric" posterior gap that changes to an asymmetric gap when viewed up close on flexible exam.

Recurrent Laryngeal Nerve
Posterior Cricoarytenoid muscle

SYMPTOMS
- stridor
- none

HISTORY
- injured during surgery (thyroid, parathyroid, carotid, esophagus, left chest surgery)
- cold symptoms with change in voice may represent viral nerve injury

DIFFERENTIAL DIAGNOSIS:
- nerve injury vs joint fixation

FINDINGS

VOCAL CAPABILITIES BATTERY:
- White noise during inspiration may be present from limited glottic opening

ENDOSCOPY
- Arytenoid tipped forward
- Posterior laryngeal fasciculations
- Atrophy of PCA area during sniffing
- No lateral motion of arytenoid during sniffing

Often branches to the LCA and TA muscles are also affected

OTHER NOTES:
- PCA neurons have a strong tendency to grow back to both the anterior and posterior branches of the RLN
- In more severe injuries, this often weights activation of the RLN during respiration toward continuous adduction of the recovered branch

REINNERVATION

NORMAL RECOVERY

- Occurs after a minor injury
- Restoration of complete range of motion
- Restoration of appropriate intentional motion

SYNKINETIC RECOVERY

- Occurs after a moderate injury
- PCA neurons split reinnervation to anterior adductor muscles and posterior abductor muscles
  - On many occasions the vocal cords on endoscopy appear immobile, resting near or a bit lateral to the midline,
  - or move slightly and movement may be appropriate in direction.
  - Tension is maintained during phontion, rather than flutter.
  - The reinnervated vocal cord can be thicker and tighter than the "normal" cord during phonation in the vocal underdoer or aged person with bowing of the "normal" cord.
  - Perhaps this represents a nearly 50:50 arrangement when abductor PCA neurons and adductor neurons reinnervate LCA and TA muscles.
  - There may also be a mix of abductor and adductor neurons reinnervating the PCA .
  - If normal recovery doesn't occur, this is the next best "ideal" spontaneous recovery.
  - That is, spontaneous reinnervation may not require intervention by surgery or therapy.

DYSKINETIC RECOVERY

- More severe injury
- Overgrowth of PCA (abductor) neurons to LCA muscle
  - Vocal process may be highly angled toward midline
  - Vocal process may cross the midline
  - Vocal process may move in the opposite direction of intention
    - medial during inspiration
    - lateral during phonation
    - lateral during expiration
- Dyspnea or stridor
  - May be light to very strong
  - Not due to weakness, it is hyper reinnervation
- May occur 10 – 20 years after injury
  - Often misdiagnosed as unresponsive asthma because of remote interval from injury
- Dyskinetic activity may increase with vocal use
- High rate of spontaneous laryngospasm
  - Responds to botulinum toxin injection into "paretic" cord.
  - Responds to reinnervation of anterior branch with ansa cervicalis with improved stability and steady tension during phonation

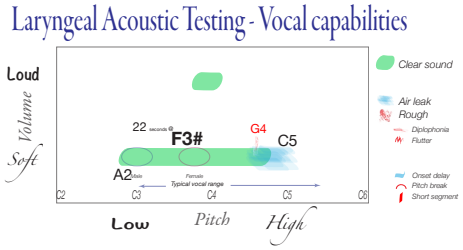
MAIN ISSUE IN BILATERAL RLN INJURY

- Initial symptoms after bilateral injury
  - Weak voice
  - Out of breath with speaking or physicial activity
  - Choking on water
- Symptoms after several months
  - PCA (abductor) neurons activate LCA movement toward midline
  - Louder voice, more dyspnea
  - More stridor
  - Increased frequency and intensity of laryngospasm
- Patients often learn to relax TA muscle and/or CTA muscle during inspiration
  - Shortens vocal cord
  - Allows central membranous cord to bow laterally
- Botulinum toxin injection
  - If placed into LCA muscle, tends to improve airway
  - If placed in TA muscle,
    - may precipitate functional airway obstruction
    - bernoulli effect moves membranous cord to midline during inspiration

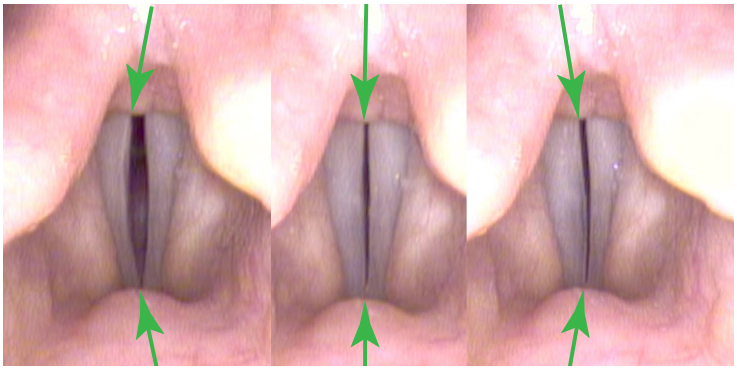
Infering tension from oscillation

Stroboscopy

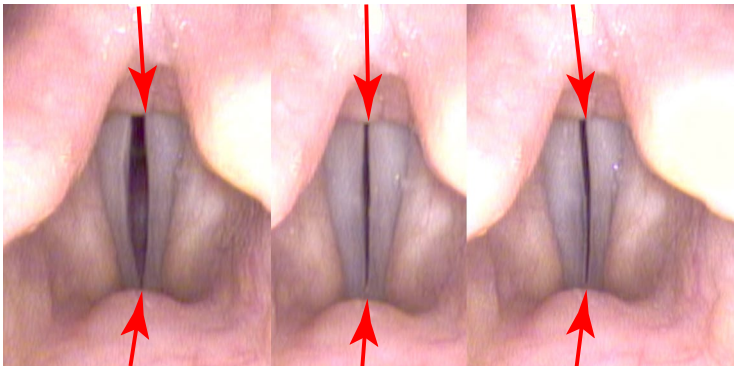
Two years ago Sally (50's) began losing her upper singing range. Her singing endurance dropped to where she fatigues after 15 minutes. She dropped from soprano and is even having difficult singing alto now.



Three selected frames from the stroboscopy exam at pitch G4 where her voice is straining to reach the note and she is leaking air.



If we follow the oscillation pattern of the right vocal cord (green arrows), the right vocal cord's central margin oscillates from lateral (left frame), to midline (middle frame) then crosses its axis (right frame) moving the vibratory margin medial to its axis. This cycle repeates over and over. The right cord is oscillating nearly symmetrically about its axis – because of adequate vocal cord tension.



If we follow the oscillation pattern of the left vocal cord in the same series (red arrows), the left vocal cord starts lateral (left frame), moves almost to midline but has a slight concavity (middle frame) and then return to a lateral to its axis position (right frame). This cycle repeats over and over.

The left vocal cord has insufficient tension relative to the right vocal cord and at this pitch (G4) never crosses its axis.

When she was examined at lower pitches, the vocal cords were symmetric in their oscillations, meeting in the midline.

In her lower range, when the cricothyroid is not activated, the tension in each vocal cord is the same leading to symmetric oscillations. As she increases pitch, the tension is greater on the right vocal cord (intact cricothyoid muscle).

This case represents a left superior laryngeal nerve paresis.

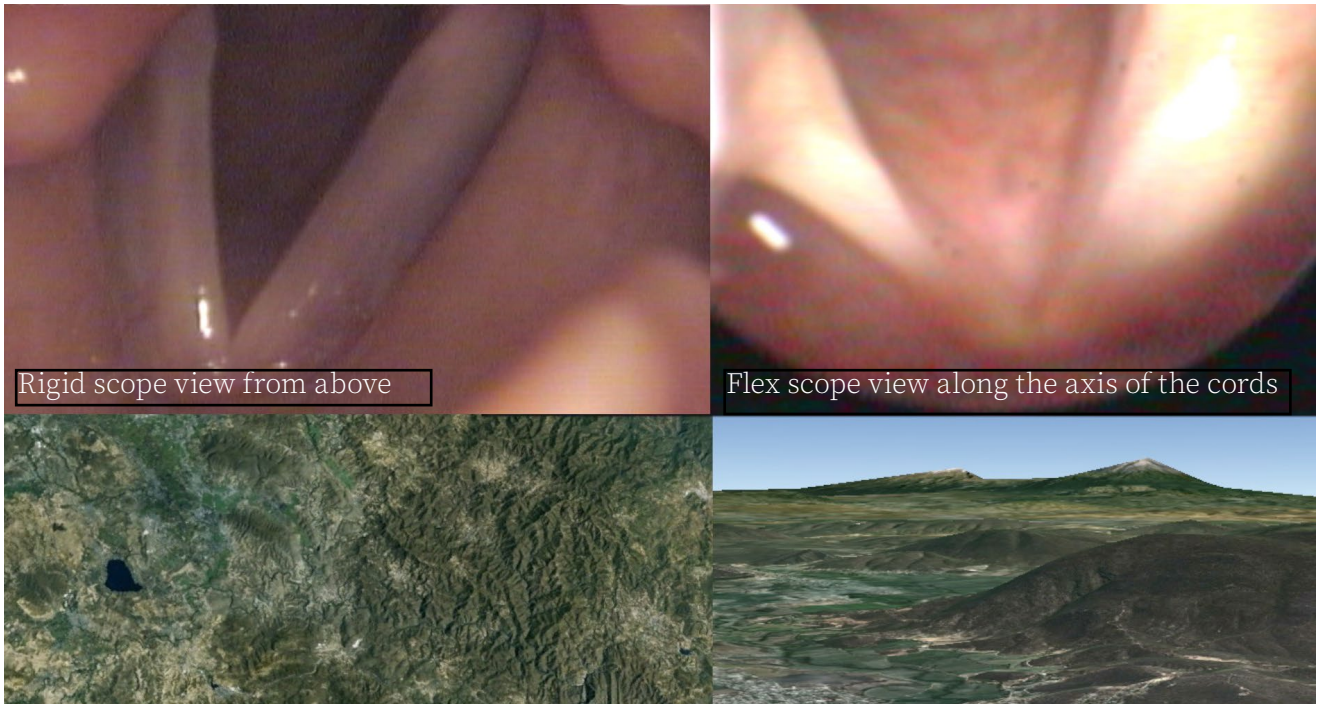
Using two endoscopes for perspective

Get super close

Position vs. Mass

Two endoscopes can offer different perspectives. The rigid endoscope view is high quality but usually more limited in terms of perspective.

A flexible endoscope can be passed into the laryngeal introitus and curved to approximate the axis of the true vocal cord giving a nearly parallel view. Often topical anesthesia is required to obtain this super close up view.



Satellite type view on a map
(Map Data: Google, © Cnes/Spot Image 2013)

Airplane type view: same region on a map
(Map Data: Google, © Cnes/Spot Image 2013)

Vocal cord position can explain some symptoms such as a weak or breathy voice when the vocal cords cannot approximate. But what if there is complete closure of the glottis from the moveable vocal cord approximating the immobile but midline vocal cord, yet the patient has an obviously diplophonic voice. A vocal cord resting in the midline does not explain diplophonia. However, the "airplane view" of the flexible endoscope reveals the difference in mass between the vocal cords and two vibratory structures of differing mass will tend to vibrate at different frequencies, consequently explaining diplophonia.

Dyskinesia & paresis
Paralysis – paresis – synkinesis

Dyspnea

Catherine had her thyroid gland removed 30 years ago. She developed a hoarse voice after the surgery suggesting her recurrent laryngeal nerve had been injured. Stretched, cut or somehow traumatized, the nerve stopped working after the surgery and one of her vocal cords could not move close enough to the other vocal cord to start vibrating with air flow, so she had no voice for several months.

Gradually her voice returned and eventually it recovered – or more likely it seemed to recover. She could no longer sing nor reach high pitches, but day-to-day, her voice was adequate for work. She accepted that as recovery.

Gradually over the past several years she started having sudden episodes of her breathing being cut off. She would be speaking and suddenly she couldn't breathe.

Every otolaryngologist who looked told her she had a paralyzed vocal cord, but that she still had "plenty of room to breathe." Several physicians told her that her episodes of difficulty breathing were from silent acid reflux and they placed her on antacids (didn't help).

We should ask, do her brief, sudden, "shortness of breath" episodes (dyspnea, usually with stridor) have anything to do with her previous surgical injury? Phrased another way, are laryngospasms related to nerve injuries?

The word paralysis means lack of mobility. It is true that her injured vocal cord does not obviously open or close with each attempted phonation or each attempted breath. There is the implicit assumption that since her vocal cord is not moving normally there is a lack of innervation – an error. The recurrent laryngeal nerve actually has such a strong propensity to regrow that even after excising several centimeters of the nerve, it still often grows back to the muscles in the larynx. When the nerve grows back, the major issue is not lack of nerve input, but lack of proper nerve input.

We can say that she is suffering from a laryngeal dyskinesia. Calling her injury a laryngeal dyskinesia implies different findings and different problems than laryngeal paralysis. A dyskinesia may be present whether or not there is any observed motion impairment, though usually there is some impairment of motion. After a nerve injury the problems that result are, to some degree, due to the degree of reinnervation, but even more due to inappropriately directed reinnervation.

In the ideal world, the injured nerve would regrow back to the muscle it used to control. In the most typical severe nerve injury about half the fibers end up going to their original muscle and the other half go to the opposing muscle. Consequently, the neurologically injured vocal cord appears to be immobile. The brain tells both muscles to contract simultaneously and the net effect is that there is no motion and nothing obvious happens.

That seemed to be the case with Catherine for many years, but something definitely changed in recent years. She began having the laryngospasms that cut off her breathing entirely for a seeming eternity (when in reality it was less than a minute, but when you can't get air, time subjectively moves slower).

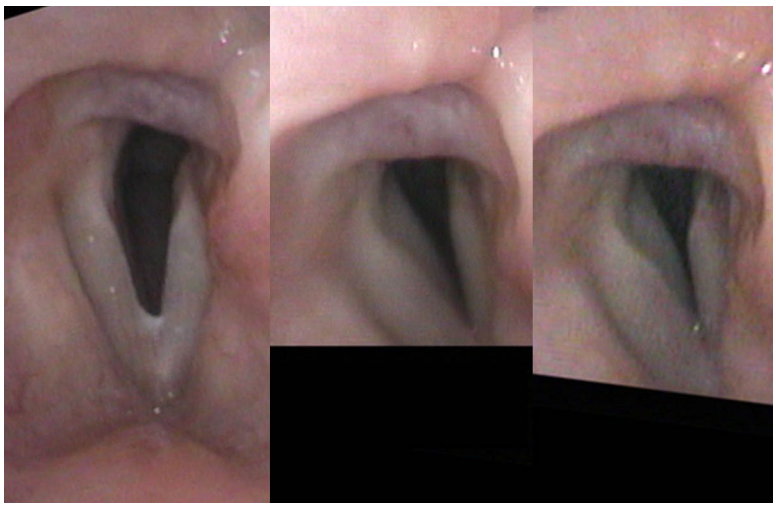
On her endoscopic exam, the healthy right vocal cord opened and closed appropriately, both during breathing and during sound production. She had quite a strong voice, though I would say that it actually had a strained or tight quality.

During the ultra-close portion of my endoscopic exam, I touched the left, non-moving or paralyzed cord lightly and it suddenly moved across the midline nearly closing off her airway. It wasn't paralyzed; it could move. It just did not move intentionally and appropriately during breathing or during phonation and it was trigger-happy. With even a small trigger, her left LCA muscle would spasm and move the left vocal process nearly all the way to the opposite cord.

This LCA spasm also increased gradually the longer Catherine spoke. With each phonation the right cord would touch the left side and the left vocal process would move further toward the right after each touch. Then, after resting her voice for a number of breaths, the left side would relax back toward its midline, resting position. (see photos below)

As both treatment and a test, I injected botulinum toxin into her dyskinetic (actually the opposite of paralyzed – hypercontracting), left vocal cord. Specifically, I placed the botulinum toxin into the TA and LCA muscles – the muscles that tense and move the cord toward the midline and closure.

Two weeks later, the opening in her larynx was larger while she was breathing. She could



Left: vocal cords at initial rest during inspiration.
Middle: after the stimulation of phonating, the left vocal process starts moving across the midline during inspiration. It remains in this position even during expiration.
Right: left vocal process hyperadducting during inspiration and narrowing the airway further after more vocal stimulation.

still make sound, though a little more softly. However, she could breathe better than she had in several years and she had not had any further laryngospasms since the injection. The paralytic effect of botulinum toxin lasted for three to four months and then the nerve connected to the muscles again and the left-side began to hypercontract again, which she could identify because of the increasing difficulty with breathing. Catherine returned to the office for another treatment every few months. After several injections, she asked if there was something more permanent that could be done.

The RLN splits like a tree into different branches. It is possible to cut only the branches that go to the closing muscles (the TA and LCA) and in effect, that is what we were doing chemically with the botulinum toxin. I suggested a surgery where we would cut the anterior branch of the RLN. Then, to prevent the original RLN from growing back as it had done 30 years ago after her injury, I would route a nerve from one of her neck muscles (the omohyoid) into the cut anterior branch suppling the TA and LCA muscles. If this new nerve sprouted fibers to the muscles before the old branch of the RLN, then she would have nerve input to these muscles during phonation (the omohyoid tenses during phonation). The TA and LCA muscles would bulk up, and even if the muscles didn't have a completely appropriate signal to move open and closed, they would hold tension during phonation and would not tend to inadvertently spasm nor tighten during breathing in.

After cutting the anterior branch of the recurrent nerve during surgery and sewing in the donor nerve, she had a weak voice for a month and then her vocal strength began to return. The left vocal fold ultimately positioned itself near the midline leaving a much larger opening than when I first met her and she could breathe easily (photo below). She no longer had laryngospasms that would cut her breathing off and her voice was less tight and strained. She could close the right vocal cord all the way to the left and had a strong and clear voice without the tight quality she had previously.

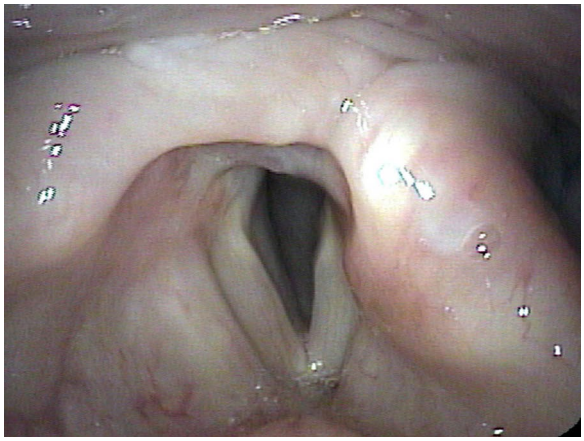
She has a reasonably open airway with the left vocal cord now stable and set in the midline during both inspiration and phonation and very importantly, no further laryngospasms.

A vocal cord's muscles after a nerve injury are seldom really paralyzed, even if there is no obvious easily recognized vocal process movement. The vocal cord doesn't lack movement though it may lack easily visible, intentional movement because of competing contractions. Also, it often has subtle, significant, inappropriate movement.

SUMMARY

After a nerve injury the vocal fold is more likely to move poorly and inappropriately (dyskinesia) than not move at all (paralysis).

For physicians who approach hoarseness after a surgical RLN injury with benign neglect, feeling that a hoarseness that recovers is inconsequential, they might wish to have their patients examined by a laryngologist to better understand their complication rate. A return to a normal voice might not mean a return to normal function.



Laryngeal opening 6 months after reinnervation on left from ansa cervicalis to anterior branch of the recurrent laryngeal nerve the left vocal process remains near the midline during respiration.

EXAMPLE CASE

Lateral cricoarytenoid paresis

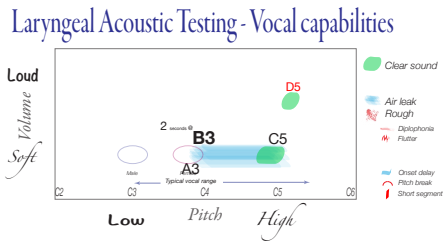
Weak voice - out of breath talking

At age 74, Stella was sitting in the hospital holding her grandchild and lost her voice. She thought it might be from the air conditioning. 6 months earlier she had completed radiation therapy to her chest for breast cancer. She cannot sing anymore and she is out of breath with talking. The first otolaryngologist told her nothing was wrong and that she had forgotten how to talk. She saw a second otolaryngologist about 8 times, trying antireflux medications lansoprazole and famotidine at various doses without improvement.

EXAM

VOCAL CAPABILITIES

Vocal capabilities testing was suggestive of a weakness although a nonorganic voice disorder could be considered in the differential diagnosis.



On vocal capabilities exam, she spoke with a falsetto quality. Her maximum phonation time was 2 seconds and she had a breathy quality throughout her vocal range except at high pitch, high volume where her sound was clear.

ENDOSCOPY

On endoscopy, the vocal cord architecture was normal.

Quiet respiration was observed.

The vocal processes were symmetric during inspiration. However, during expiration (above), the left vocal process did not move as far medial, nor rotate as medial as the right vocal process.

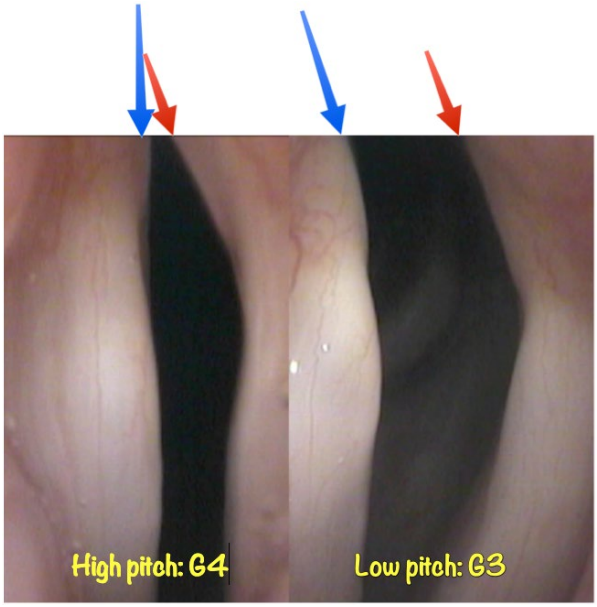
STROBOSCOPY

Stroboscopy was performed at her choice of pitch.

When allowed to phonate at any pitch (here vocal cords at phonatory offset at G4), her vocal cords align seemingly perfectly.

While her vocal capabilites supported her complaints, her visual exam didn't reveal an answer. Consequently, her larynx was topically anesthetized and the endoscope inserted beneath the vocal processes. She was then examined at varying pitches.

The tip of the vocal processes hold the key to assessing the functional status of the lateral cricoarytenoid muscle.



At high pitch the right vocal process is near the midline (blue arrow). The left vocal process has not rotated medially (red arrow), but the cricothyroid has pulled both vocal cords longer and thus straightened them, indirectly pulling the vocal processes closer together.

At low pitch the right vocal process is angled past the midline (blue arrow). The left vocal process has not rotated medially (red arrow) leaving a large gap for air escape. This gap is not visible unless the endoscope is placed beneath the arytenoids

CONCLUSION

She has an isolated left lateral cricoarytenoid muscle paresis. Her natural tendency is to use the cricothyroid muscle for compensation.

EXAMPLE CASE

Thyroarytenoid & Lateral cricoarytenoid paresis

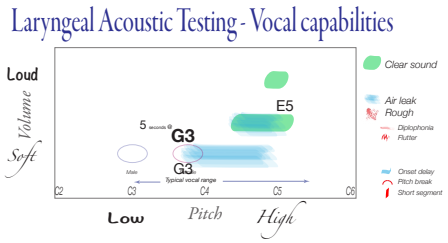
Weak voice - out of breath talking

At age 51, Melanie began to develop a raspy voice 4 days after hip surgery. She was told that she did not have an endotracheal tube in. Her allergist told her it was from reflux and treated her with omeprazole. Her heartburn went away and her voice remained poor. She saw an otolaryngologist who saw a vocal nodule and sent her to Speech therapy. She did not get any better.

EXAM

VOCAL CAPABILITIES

Vocal capabilities testing was suggestive of a weakness, most significantly in her lower vocal range.

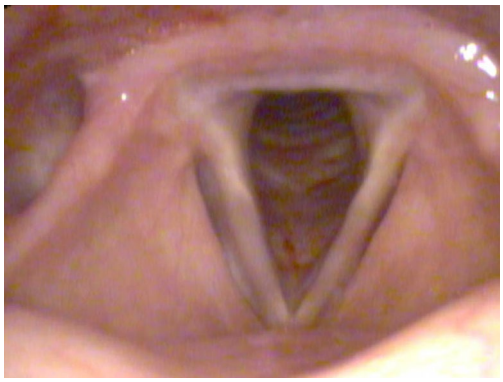


On vocal capabilities exam, she spoke with a lot of huskiness. Her maximum phonation time was 5 seconds and she had a breathy quality throughout her soft and low vocal range. At high pitch, high volume her sound was clearer.

ENDOSCOPY

On endoscopy, the vocal cord architecture was normal on a typical overview.

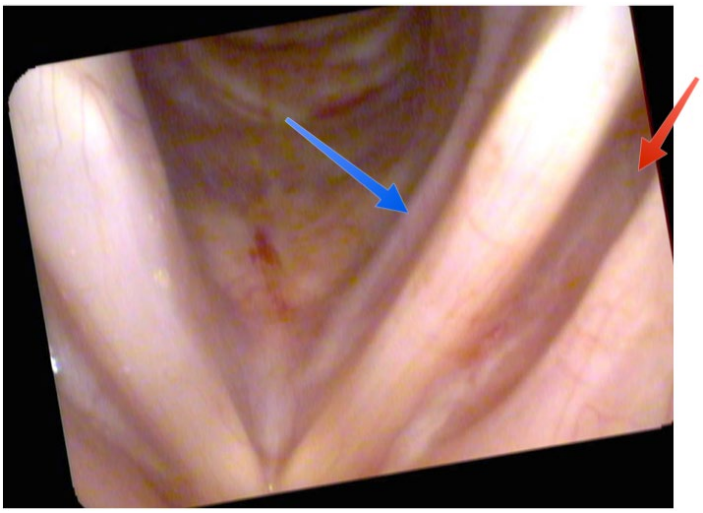
On close view during expiration the thickness of the vocal cords appears almost normal. But the left vocal process has not rotated medially as far as the right.



Inspiration

Expiration

Deep inspiration stretches a thin thyroarytenoid muscle.



Close up view of deep inspiration reveals concavity of left subglottis (blue arrow) and a large left ventricle (red arrow). There is less mass within the left thyroarytenoid muscle.

CONCLUSION

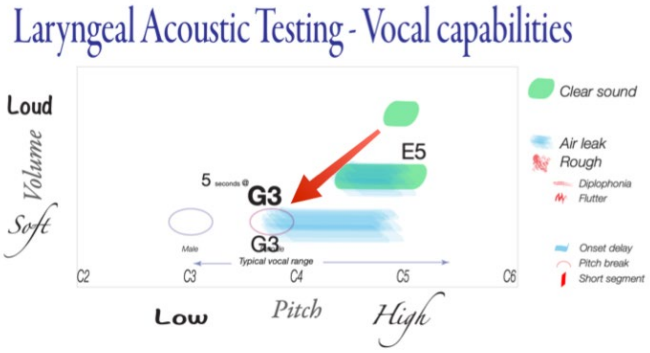
She has an left thyroarytenoid muscle and additional findings show it combined with a left lateral cricoarytenoid muscle paresis. Her natural tendency was to use supraglottic compression for compensation.

VOCAL CAPABILITES TESTING

Neuro- paresis

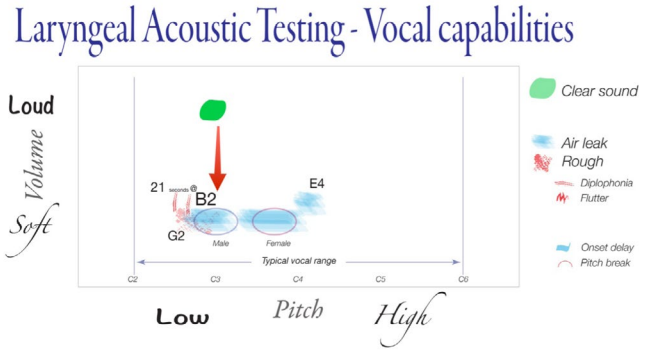
Nonparetic

Paresis TA, LCA



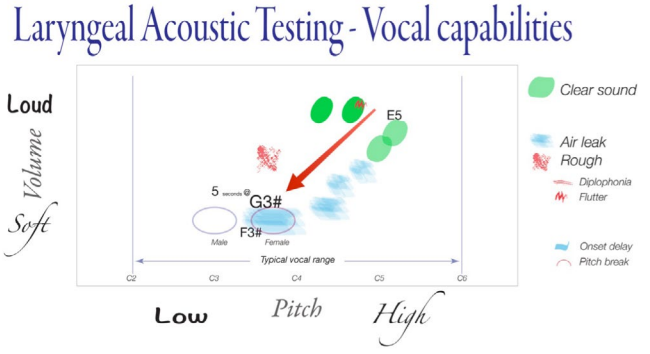
Low pitch, low volume leak, clear high pitch/vol.

Bowing



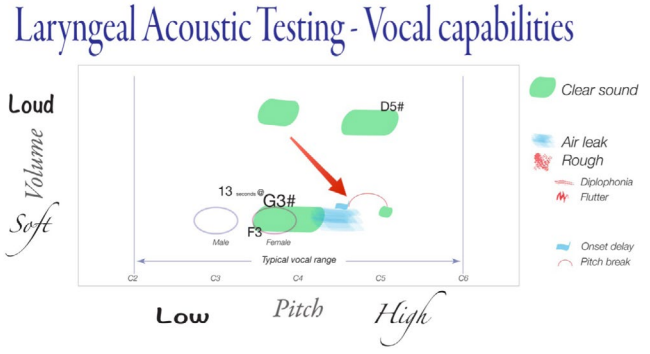
Low pitch, any volume region

Paresis LCA



Low pitch, soft volume region

Central swellings



High pitch, soft volume region

These patterns are the vocal signature for a different types of vocal impairment. Each pattern predicts what pathology will be seen during laryngoscopy and it suggests to the examiner what pitch and volume combinations are most likely to reveal the pathology to the examiner during laryngoscopy. The red arrows point toward the region of maximal vocal abnormal findings and in recurrent laryngeal nerve paresis, that is usually the low pitch, low volume area where there is no compensation from the cricothyroid muscle. Bowing resembles this pattern except that high volume, low pitch is typically quite clear. Vocal swellings have an entirely different pattern.