Laryngeal Features of External Superior Laryngeal Nerve (ESLN) Denervation: Revisiting a Century-Old Controversy

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Background

The external branch of the superior laryngeal nerve (ESLN) innervates the cricothyroid (CT) muscle of the larynx, a vocal fold tensor “primarily” responsible for pitch elevation.
Effects of Unilateral CT Dysfunction: A Source of Controversy

For over 100 years, a controversy has existed regarding the laryngeal signs that should be considered pathognomonic of unilateral ESLN paralysis.

Myriad descriptions exist of the laryngeal behaviors ostensibly associated with unilateral ESLN denervation.

2. **Decreased longitudinal tension and length of the ipsilateral vocal fold with glottic insufficiency secondary to mild bowing** (Ward, 1977; Dursun, Sataloff et al., 1996).
3. **Sluggish ab- or adduction of the ipsilateral fold during repetitive phonatory tasks** (Heman-Ackah & Batory, 2003; Rubin et al., 2005).
4. **Asymmetrical, irregular, or aperiodic vocal fold vibration** (Dursun et al., 1996; Mendelsohn et al., 2007).
5. **Reduced vocal fold amplitude, and mucosal wave in some cases** (Dursun, et al., 1996).
To complicate matters, confusion surrounds whether voice and laryngeal manifestations reflect the effects of pure CT dysfunction, or alternatively, compensatory adjustments to prolonged denervation (and perhaps abnormal reinnervation).

To date, no consensus exists regarding the laryngeal features of acute or chronic, unilateral ESLN denervation.

Without improved recognition and differential diagnosis of unilateral ESLN denervation, it is impossible to discuss its prevalence, phonatory effects and/or treatment.
To address this longstanding controversy, we modeled “in vivo” acute, unilateral ESLN denervation by temporarily blocking the R. ESLN using lidocaine HCL (verified selective blockade with hooked-wire LEMGs).

By surveying a broad range of vocal tasks before and during the ESLN block, two specific aims were addressed:

1. to describe the salient phonatory and laryngeal features associated with acute CT denervation.

2. to identify a set of voice and laryngeal tasks that maximally provoke or expose ESLN dysfunction when present, thereby contributing to the development of improved clinical diagnostic protocols.
Methods

Participants
- 10 young, healthy adult males (non singers), no history of past or present voice disorder.
Hooked-wire LEMG Confirmation & Monitoring of Selective R. ESLN BLOCK

Pre-Block

During CT Block

MIC
R. CT
R. TA
Pre vs. During ESLN Block
Flexible Videolaryngostroboscopy (FVLS)

- Surveyed a wide range of vocal tasks/maneuvers, combining various pitch and loudness conditions that would permit comprehensive assessment of laryngeal function using Flexible videolaryngostroboscopy (FVLS).

- Tasks were included that would evaluate all claims made in the literature regarding laryngeal features associated with ESLN paralysis.
Specific Laryngeal Findings

Contrary to clinical reports...

- *no evidence of hypomobility/sluggishness* of the ipsilateral vocal fold, or a reliable pattern of axial rotation of the larynx during high pitch voice was observed.

- no evidence to support reduced vocal fold longitudinal tension/length, aryepiglottic fold length asymmetry, phase asymmetry, vocal fold plane differences, or glottic insufficiency, as diagnostic features of acute, unilateral CT dysfunction.
Instead, the analysis revealed a pattern of:

- deviation of the petiole of the epiglottis to the side of weakness (i.e., the right) in 60% of participants during a glissando up maneuver produced at normal volume.
Pre vs. During ESLN Block (M09)
Pre vs. During ESLN Block (M02)
Pre vs. During ESLN Block (M01)

Pre-

During Block
Pre vs. During ESLN Block (M04)
Pre vs. During ESLN Block (M05)
Pre vs. During ESLN Block (M10)
Example: Pre vs. During ESLN Block

Pre-ESLN Block
Is there clinical evidence to support the findings of the “in vivo” model?

- Acute vs. chronic?
- Paralysis vs. paresis?
- Isolated ESLN vs. combined with other branches?
- Any muscle asymmetry (RLN) vs. specific to ESLN denervation?
Cases of ESLN Denervation
Case #1: R. ESLN paresis

- 70 y.o., 8 mos. post surgical resection of C5-C6 vertebral body schwannoma (R. anterior approach).
- c/o weak voice, increased effort, and vocal fatigue.
- LEMG: “electrodiagnostic evidence of previous denervation and partial re-innervation suggesting axonal injury to right SLN”
R. ESLN paresis

Abduction

NPNL

Glissando Up
Video R. ESLN Paresis
Case #2 R. RLN & SLN paresis

- 40 y.o. physician.
- c/o 4 mos. Hx of increased phonatory effort, and vocal fatigue (all symptoms intensifying after extended use).
- o/e Essentially Normal Voice
- LEMG “increased complexity and amplitude of fast firing motor units in the right TA and, to a lesser extent, right cricothyroid...electrodiagnostic evidence of previous injury to right RLN (and SLN) with subsequent partial re-innervation.
Case #2 R. RLN & ESLN paresis

Abduction

NPNL

Glissando Up

Abduction
Case #2 Video
Case #3 Brainstem lesion

- 18 y.o. male
- At age 2, right hemiparesis, left vf paralysis (?recovered), hypernasality following removal of cavernous malformation of brainstem.
- Rebleed at age 16 with increased hypernasality.
- Palate deviates to right, VPI (left >right), hypomobile left vocal fold.
Case#3 L. Brainstem Lesion

Abduction

NPNL

Glissando Up
Case #3 Video
Additional Questions

- Does epiglottic petiole deviation occur in the presence of unilateral RLN paresis/paralysis?
- Does any asymmetric muscle activity within the larynx produce the marker, or is it specific to ESLN paralysis only?
Case # 4: 36 y.o. L. RLN denervation only (post PDA ligation @ age 19)
Case #4 Video
L. UVFP secondary to L. PDA ligation
Conclusions: ESLN Paralysis

Deviation of the petiole of the epiglottis to the side of weakness during GUNV may represent a valuable diagnostic marker of ESLN denervation.

Additional prospective studies are needed to validate the diagnostic precision of this marker.
References


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