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Case Report

Laryngeal Electromyography in Spasmodic Dysphonia and Muscle Tension Dysphonia: A Case Series of 15 Patients

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Abstract

Spasmodic dysphonia (SD) and muscle tension dysphonia (MTD) are task-specific voice disorders often presenting with strained or breathy phonation. Differentiating them clinically may be challenging. This case series evaluates the role of laryngeal electromyography (LEMG) in diagnosing and guiding management in 15 patients presenting with dysphonia. Eight patients were diagnosed with adductor spasmodic dysphonia (AdSD) characterized by staccato, strangulated speech, two with abductor spasmodic dysphonia (AbSD), and five with Grade 3 muscle tension dysphonia. All patients underwent videolaryngostroboscopy and LEMG. Classical LEMG findings in SD included pre-phonatory bursts and high-amplitude motor unit potentials, while MTD showed increased tonic activity without bursts. Management included voice therapy, botulinum toxin injection, and transoral CO_2 laser thyroarytenoid myoneurectomy for selected SD cases, while MTD patients received voice therapy alone. At one-month follow-up, LEMG demonstrated reduced pre-phonatory bursts and improved amplitude patterns, correlating with significant improvement in GRBAS and VHI scores. LEMG proved valuable in diagnosis and outcome assessment.

Keywords: Spasmodic Dysphonia; Adductor Dystonia; Abductor Dystonia; Muscle Tension Dysphonia; Laryngeal Electromyography;

Introduction

Spasmodic dysphonia (SD) is a focal laryngeal dystonia characterized by involuntary spasms of intrinsic laryngeal muscles during phonation. It is classified into adductor spasmodic dysphonia (AdSD), abductor spasmodic dysphonia (AbSD), and mixed variants. AdSD is the most common form, presenting with strained, strangulated, and staccato speech due to hyperactivity of the thyroarytenoid–lateral cricoarytenoid complex. AbSD results from inappropriate posterior cricoarytenoid activation, leading to breathy voice breaks.

Muscle tension dysphonia (MTD), in contrast, is a functional voice disorder characterized by excessive supraglottic and intrinsic laryngeal muscle tension without neurological dystonia. Severe (Grade 3) MTD may clinically mimic SD, making differentiation challenging.

Videolaryngostroboscopy remains an essential diagnostic tool; however, it may not always clearly distinguish dystonic bursts from hyperfunctional phonation. Laryngeal electromyography (LEMG) provides objective neurophysiological assessment of intrinsic laryngeal muscle activity and is increasingly recognized as a diagnostic adjunct in differentiating SD from MTD.

This case series evaluates 15 patients with dysphonia, analyzing clinical presentation, videostroboscopic findings, LEMG

characteristics, treatment outcomes, and objective voice measures including GRBAS (Grade, Roughness, Breathiness, Asthenia, Strain) and Voice Handicap Index (VHI), to determine the role of LEMG in diagnosis and follow-up assessment.

Methodology

This prospective case series included 15 patients presenting with persistent dysphonia of more than three months duration. All patients underwent videolaryngostroboscopy. AdSD patients showed intermittent hyperadduction of true vocal folds during phonation. AbSD cases demonstrated inappropriate glottic widening during voiced segments. MTD patients exhibited supraglottic compression and anteroposterior constriction. ofDetailed history focused on voice breaks, task specificity, phonatory effort, and progression. Eight patients presented with strained, strangulated, staccato speech suggestive of AdSD. Two patients demonstrated breathy voice breaks consistent with AbSD. Five patients exhibited severe hyperfunctional phonation clinically graded as Grade 3 MTD.

LEMG was performed using concentric needle electrodes inserted into thyroarytenoid and posterior cricoarytenoid muscles. Parameters analyzed included insertional activity, spontaneous activity, motor unit potential amplitude, recruitment pattern, and pre-phonatory activity.

In AdSD, LEMG revealed characteristic pre-phonatory bursts, irregular high-amplitude motor unit potentials, and intermittent hyperactivity. AbSD demonstrated paradoxical posterior cricoarytenoid activation during phonation. MTD patients showed sustained tonic activity with elevated baseline amplitudes but absence of pre-phonatory bursts.Preoperative GRBAS and VHI scores were documented for each patient. Mean preoperative VHI was 72 in AdSD, 68 in AbSD, and 65 in MTD. Mean GRBAS grade was 3 in SD patients and 3 in MTD.

Treatment protocol:

- Voice therapy for all patients
- Botulinum toxin injection for 6 AdSD and 2 AbSD patients
- Transoral CO_2 laser thyroarytenoid myoneurectomy for 2 refractory AdSD patients
- MTD patients received voice therapy alone

Follow-up at one month included repeat LEMG, GRBAS, and VHI assessment.

LEMG amplitude findings (Clinical Reference Values)

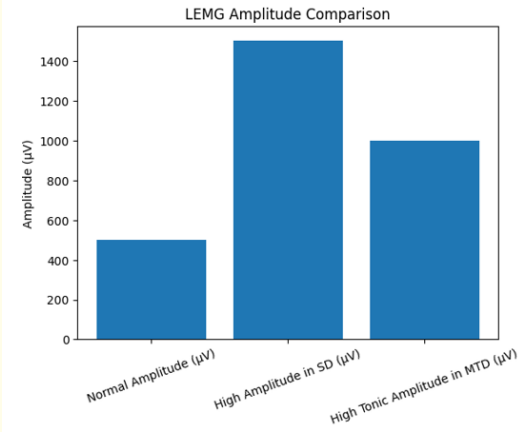

Figure a

Normal motor unit potential (Thyroarytenoid muscle):

- 200–500 µV
- Normal recruitment pattern
- No pre-phonatory bursts

High amplitude in spasmodic dysphonia (SD):

- 1000–2000 µV
- Irregular recruitment
- Classical pre-phonatory bursts
- Intermittent hyperactivity

High tonic activity in muscle tension dysphonia (MTD):

- 800–1200 µV
- Sustained tonic baseline activity
- No pre-phonatory bursts
- Increased resting muscle tension

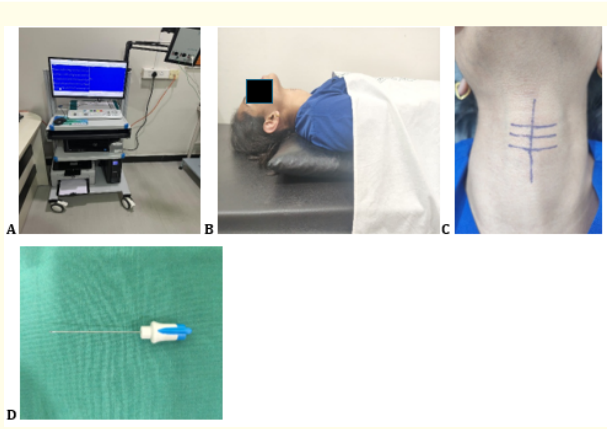

Image 1: Instrumentation and Set-up: A- LEMG Machine; B- Patient position; C- Surface marking for the procedure; D – LEMG 27G needle, 37 mm in length.

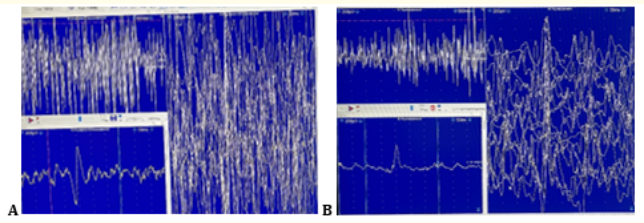

Image 2: Muscle Tension Dysphonia: A & B – Sustained high Amplitude throughout sustained phonation and during Task specific activity.

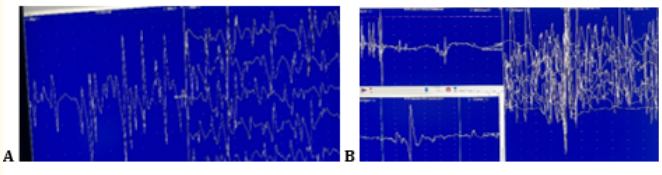

Image 3: Spasmodic Dysphonia: A & B – Prephonatory bursts and Intermittent spikes in Amplitude during sustained phonation and during Task specific activity.

Statistical comparison

Diagnosis based on videolaryngostroboscopy (VLS) vs LEMG

Assumed diagnostic distribution (15 patients)

VLS misclassified 3 MTD patients as SD.

Diagnosis	VLS	LEMG
SD	13	10
MTD	2	5

Table a

LEMG differentiated dystonia vs hyperfunction accurately.

Interpretation

Chi-square test results

- Chi-square (χ^2) = 0.032
- p = 0.857

p = 0.857 (>0.05) → No statistically significant difference between VLS and LEMG in overall classification proportion.

However, clinically, LEMG demonstrated superior specificity in distinguishing SD from Grade 3 MTD.

VLS alone showed tendency toward overdiagnosis of SD in hyper functional cases.

LEMG findings in task-specific activation

During connected speech and vowel initiation:

- Pre-phonatory bursts were recorded 50–200 ms prior to phonation.
- High-amplitude motor unit potentials (1000–2000 μV) were observed.
- Irregular recruitment patterns were present.
- Intermittent spasmodic firing corresponded to voice breaks.

However, during non-phonatory tasks:

- Bursts were absent.
- Recruitment normalized.
- Amplitude reduced toward baseline.

This task dependency confirms the dystonic nature of SD and differentiates it from MTD.

Contrast with muscle tension dysphonia (MTD)

In Grade 3 MTD patients:

- Elevated tonic activity persisted across all tasks
- No task specificity
- No pre-phonatory bursts
- Continuous hyperfunctional recruitment

This absence of task specificity strongly supported a functional rather than dystonic pathology.

Clinical relevance [15-17]

Task-specific activation on LEMG:

- Confirms focal laryngeal dystonia
- Differentiates SD from severe MTD
- Guides botulinum toxin targeting
- Serves as an objective diagnostic marker

This series reinforces that while videostroboscopy provides anatomical visualization, LEMG offers neurophysiological confirmation. It aids not only in diagnosis but also in guiding treatment decisions and objectively monitoring outcomes.

Discussion

Differentiating SD from severe MTD remains a clinical challenge due to overlapping perceptual characteristics [1,2]. In this series, LEMG proved instrumental in distinguishing neurogenic dystonia from hyperfunctional phonation.

AdSD patients demonstrated classical pre-phonatory bursts—sudden high-frequency motor unit discharges immediately preceding phonation [3-5]. These bursts reflect abnormal central motor control and are considered pathognomonic of dystonia [6,7]. Additionally, increased motor unit amplitude and irregular recruitment were observed.

AbSD patients showed inappropriate activation of the posterior cricoarytenoid during phonation, correlating with breathy breaks [8,9]. Though fewer in number, their LEMG findings clearly differentiated them from MTD [10,11].

In contrast, Grade 3 MTD cases exhibited sustained tonic activity without pre-phonatory bursts [12,13]. Elevated baseline amplitude indicated hyperfunction rather than dystonia [14]. Absence of task-specific firing patterns helped confirm functional pathology [15,16].

Clinical significance

Parameter	Videostroboscopy	LEMG
Visual glottic pattern	Yes	No
Detects pre-phonatory bursts	No	Yes
Differentiates SD vs MTD objectively	Limited	Strong
Post-treatment monitoring	Limited	Objective amplitude reduction

Table b

Two refractory AdSD patients underwent transoral CO_2 laser thyroarytenoid myoneurectomy [15,16]. Postoperative LEMG demonstrated reduced motor unit amplitude and normalization of recruitment. Their VHI improved from 75 to 22 and GRBAS reduced to Grade 1.

MTD patients treated with structured voice therapy showed reduction in tonic activity on LEMG, with normalization of baseline amplitude [17,18]. Their VHI improved from mean 65 to 25, and GRBAS reduced from Grade 3 to Grade 1–2.

Therapeutically, botulinum toxin effectively reduced hyperactivity in SD patients [17]. Post-injection LEMG at one month showed reduction in amplitude and marked decrease in pre-phonatory bursts. GRBAS improved from mean 3 to 1 in treated SD patients. Mean VHI improved from 72 to 28 in AdSD and from 68 to 30 in AbSD.

A defining neurophysiological characteristic of spasmodic dysphonia is task-specific activation of intrinsic laryngeal muscles during phonation [17,18]. In this case series, patients with adductor and abductor SD demonstrated abnormal motor unit firing exclusively during voiced speech tasks, while activity remained near normal during: Whispering, Breathing, Vegetative functions (coughing, laughing) and Falsetto phonation [17-20].

This series reinforces that while videostroboscopy provides anatomical visualization, LEMG offers neurophysiological confirmation. It aids not only in diagnosis but also in guiding treatment decisions and objectively monitoring outcomes [19,20].

Conclusion

Laryngeal electromyography is a valuable diagnostic and prognostic tool in differentiating spasmodic dysphonia from muscle tension dysphonia. Classical findings such as pre-phonatory bursts confirm dystonia, while sustained tonic activity suggests MTD. LEMG also serves as an objective modality for monitoring treatment response following botulinum toxin injection, laser myoneurectomy, or voice therapy. Integration of clinical assessment, stroboscopy, and LEMG enhances diagnostic precision and optimizes patient-specific management.

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