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Laryngeal EMG: Preferential Damage of the Posterior Cricoarytenoid Muscle Branches Especially in Iatrogenic Recurrent Laryngeal Nerve Lesions

Gerhard Foerster, MD &; Andreas H. Mueller, MD

**Objective:** Laryngeal electromyography (LEMG) of the thyroarytenoid (TA) muscle alone may not be sufficient in all patients to characterize or prove a recurrent laryngeal nerve (RLN) lesion in cases of vocal fold immobility. LEMG of the posterior cricoarytenoid (PCA) muscle may provide additional information.

**Study Design:** Retrospective review.

**Method:** Between 2008 and 2016, 339 patients in our laryngeal paralysis clinic were examined by transcutaneous needle TA-LEMG and, if tolerated, by PCA-LEMG. LEMGs were rated and compared according to criteria of the European Laryngological Society. Etiology was categorized as iatrogenic, noniatrogenic, or malignancy related.

**Results:** A total of 282 out of 339 patients had a partial or complete RLN or vagal nerve lesion: 178 iatrogenic, 74 noniatrogenic, and 30 because of nerve involvement by malignancies. Of paralytic vocal folds, 35.7% had normal or near-normal TA innervation, whereas corresponding PCA traces (if present) were pathologic in 94.6%. Comparing pairs of TA and PCA-LEMGs in paralysis of less than 4 months duration showed a predominance of PCA branch injuries in iatrogenic lesions (71.7%), while in noniatrogenic lesions this was less pronounced (44.4%). In the few malignancy cases, there was an almost even distribution. Synkineti reinnervation was earlier in iatrogenic RLN lesions.

**Conclusion:** PCA-LEMG was better in proving an RLN lesion than TA-EMG alone. Our findings suggest etiology-dependent differences in the TA/PCA lesion pattern. To confirm this, larger sample sizes are needed. A preferential damage to PCA innervation in iatrogenic lesions could be relevant for further improvements of intraoperative neuromonitoring.

**Key Words:** Larynx, EMG, PCA, TA, vocal fold paralysis, damage pattern, etiology, neuromonitoring, synkinesia.

**Level of Evidence:** 4.

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**INTRODUCTION**

Laryngeal electromyography (LEMG) was pioneered in the 1940s and 1950s. Improvements led to some wider use in the 1970s. Transoral techniques widened the diagnostic scope, but for routine use transcutaneous mono- or bipolar needle techniques remained standard due to better signal quality, shorter procedure times, and lower costs. To reduce the burden of the procedure on patients, it became common to limit the examination to the thyroarytenoid (TA) muscle. Data on LEMG of the posterior cricoarytenoid (PCA) and other intrinsic laryngeal muscles is very limited in the literature, apart from some fundamental studies describing normal physiology. In the 1990s, interest declined as the impact on clinical decision making was felt to be low, and LEMG primarily became used for guiding the injection of botulinum toxin in spasmodic dysphonia. However, there is evidence for LEMG to predict defective healing of laryngeal nerve palsy. The good negative predictive value of LEMG in such cases was demonstrated in a meta-analysis. Reliable prediction of a low probability of recovering purposeful vocal fold movement is useful for the timing of potentially irreversible surgical interventions.

The ability to reliably predict recovery of vocal fold motion seems more difficult and remains within the level of chance, although newer concepts of integrating both qualitative and quantitative LEMG analysis are promising better results in recovery prediction. In the last 5 years, there has been renewed interest in LEMG and neurolaryngology due to developments such as selective reinnervation techniques and laryngeal pacing.

Our own interest in laryngeal EMG intensified with screening patients for laryngeal pacing because we wanted to know the innervation status of the larynx and especially of the PCA, our target muscle for the functional electrical stimulation. We have seen a considerable number of patients with vocal fold immobility who have a normal or near-normal TA-EMG. In many cases, conducting PCA-EMG revealed damage of the PCA branches of the recurrent laryngeal nerve (RLN). Most of these cases seem to occur in iatrogenic RLN lesions.
We undertook this study to define the incidence of normal or near-normal TA-EMGs in patients with vocal fold immobility and to demonstrate the value of adding PCA-EMG to identify RLN lesions in such cases. We also looked at the relative value of adding PCA-EMG to evaluate vocal fold immobility based on the presumptive etiology of the lesion.

MATERIALS AND METHODS

A total number of 339 patients were seen in our laryngeal paralysis clinic (384 LEMG sessions) between January 2008 and December 2016. All LEMGs were performed using 50 mm needles (Teca Elite, Natus Neurology, Middleton, Wisconsin, U.S.A.) with a four-channel EMG machine (Neuropack M1, Nihon Kohden, Tokyo, Japan). In 2013, we started using multichannel recordings including channels for a respiratory belt; a thermistor to register nasal breathing; and a microphone (Atmos, Lenzkirch, Germany) to record vocal maneuvers, commands, and comments. All data were analyzed and classified in accordance with the European Laryngological Society consensus article on LEMG. According to this guideline, interference pattern (IFP) density during maximum effort is classified and numbered as normal/dense (class 4), mildly reduced (class 3), moderately/strongly reduced (class 2), single fiber activity/pattern (class 1), or no activity (class 0). Lower values correspond to more severe nerve branch damage.

Pathologic spontaneous activity, defined as regularly spaced, low-amplitude, short-duration potential units that are generated by a single-muscle fiber indicating axonal degeneration, were evaluated as absent (0), sparse (1), clear (2) or dense (3).

A pathologic reinnervation is often referred to as synkinesis and is defined/recognized as an increase in activity during an antagonist maneuver (i.e., increased activity in the TA muscle during a forced sniff or in the PCA muscle during phonation) and was graded in the same way as pathologic spontaneous activity. Data sets often were incomplete, sometimes because not all muscles could be traced but often due to patient comfort and compliance. PCA-EMG was not enforced, especially in cases for which TA-EMG gave sufficient proof of a paralysis.

The data set of 402 TA-EMGs and 209 PCA-EMGs (counting each examined muscle) was divided in different ways to answer different questions:

1. PCA-EMG proof of paralysis: any etiology, any duration, only immobility/paralysis patients with “near-normal” or normal TA-EMG (95 of 266 TA-EMGs with corresponding 56 PCA-EMGs). An LEMG tracing was considered normal or near-normal if the IFP-density was class 4 or 3 with absent or sparse pathologic spontaneous activity and absent or sparse synkinesis.

2. Comparing TA/PCA lesion pattern in different etiologies: all TA- and PCA-EMGs with less than 4 months duration (171 TA- and 86 PCA-EMGs), averages of all applicable traces (Fig. 1), distribution of IFP classes in different etiologies (Fig. 2), and at different time intervals for iatrogenic lesions (Fig. 3) as well as pairs of ipsilateral TA-PCA-EMGs (Fig. 4). In patients with repeated LEMGs of a paretic vocal fold, only the first sufficient study was analyzed. Including patients who presented within 4 months allowed us to accumulate an adequate sample size while avoiding distortion of results by regenerative processes, which clinically become more obvious 4 months after the onset of the paralysis.

3. Onset of synkinesis: TA- and PCA-EMGs fitting in the following time intervals: < 4 months, 6 to 8 months, and > 12 months (numbers in Fig. 5).

RESULTS

Out of 339 patients, 282 had a partial or complete unilateral or bilateral RLN or vagal nerve lesion (37 with partial paresis, 245 with immobility). Paralysis was iatrogenic in 178 patients, noniatrogenic in 74, and due to RLN involvement by malignancy in 30. The remaining 57 patients had other findings or were examined to rule out a nerve lesion.

Ninety-five (35.7 %) out of 266 recorded TA muscles in immobile vocal folds showed normal or near-normal

![Fig. 1. Laryngeal EMG means of interference pattern density classes in RLN paralysis of different etiologies. IFP classes in RLN paralysis of less than 4 months duration (n = 146). Comparison between TA- and PCA-EMG in iatrogenic, noniatrogenic, and malignancy-related paralysis. Class 4 IFP: normal/dense pattern; class 3 IFP: mildly reduced; class 2 IFP: moderately/strongly reduced; class 1 IFP: single fiber activity/pattern; 0 IFP: no activity. Lower values indicate more severe nerve branch damage. The difference between TA- and PCA-mean was significant in iatrogenic lesions (P < 0.01, Mann-Whitney U-test). EMG = electromyography; IFP = interference pattern; PCA = posterior cricoarytenoid; RLN = recurrent laryngeal nerve; TA = thyroarytenoid.](image)

All data were collected retrospectively and processed with Excel 2010 (Microsoft Corporation, Redmond, Washington, U.S.A.). Statistical analysis (Mann-Whitney U-test for independent samples) was carried out using SPSS 23 (IBM Corp., Armonk, New York, U.S.A.). P values were calculated for two-sided tests with the level of significance set at P < 0.05.

![Fig. 2. Distribution of interference pattern density of TA and PCA electromyographies. Recurrent laryngeal nerve paralysis with less than 4 months duration. Noticeable difference between TA and PCA, especially in iatrogenic paralysis. Darker colors mark lower interference pattern density, i.e., stronger denervation. iatr. = iatrogenic; malig. = malignancy; PCA = posterior cricoarytenoid; TA = thyroarytenoid.](image)
innervation in terms of interference pattern density, spontaneous activity, and synkinetic activation. In 56 of these cases, PCA-EMG recording was successfully performed and revealed RLN paralysis as the cause of vocal fold immobility in 53 (94.6%).

Two of the three cases with near-normal TA- and near-normal PCA-EMGs were derived from patients with longstanding iatrogenic lesions (48 and 350 months duration). In both cases, PCA-EMG patterns were abnormal, showing more constant activation rather than the typical variation with respiration. The third case had underlying malignancy and could not be solved.

In paralysis of less than 4 months duration, the average IFP density class of PCA was lower than that of TA, particularly in iatrogenic paralysis, which was highly significant, $P < 0.001$ (Fig. 1). The distribution of IFP classes is depicted in Figure 2, and the extent of the differences in ipsilateral recorded TA–PCA-trace pairs is illustrated in Figure 4. In iatrogenic paralysis, the IFP density class is lower in the PCA compared to the ipsilateral TA in 71.7% of patients; 17% are equal; and only 11.3% have a higher IFP density class in the PCA. In noniatrogenic paralysis, this is much less obvious: the ipsilateral PCA-IFP density class is lower in 44.4%, equal in 44.4%, and higher in 11.1%. The number of data sets in noniatrogenic paralysis was much lower ($n = 18$), and the differences were not significant $P = 0.098$ (Fig. 4).

In persisting iatrogenic paralysis, there were sufficient data sets to depict IFP density class distributions by duration of the lesions. Figure 3 shows that innervation/IFP density class improves over time but the principle differences between TA and PCA remain.

Synkinesia almost was absent in paralysis of less than 4 months duration. In the group, the percentage of muscles with synkinetic activation quickly rose after 6 months duration of paralysis in iatrogenic paralysis, with little further increase after 1 year or longer and little differences between TA and PCA. In noniatrogenic paralysis, development of synkinesia seems delayed relative to iatrogenic lesions. There was not enough data to estimate when synkinesia peaks in noniatrogenic paralysis.

**DISCUSSION**

In one-third of all immobile vocal folds, there was no substantial TA lesion (35.7%); thus, a TA-EMG alone would not have explained the immobility. If recorded, PCA-LEMG demonstrated significant RLN pathology of the PCA branch in the vast majority of these cases. Depending on the criteria, it was 53 of 56 cases (94.6%) if considering IFP density, PSA, and synkinesis—or 55 of 56 cases (97.8%) if also considering other pathologic PCA activation patterns.

In other words, PCA-EMG appears to be more sensitive than TA-EMG in detecting partial or unequal lesions of the recurrent laryngeal nerve. However, PCA-EMG is not always tolerated. Looking on those 95 near-normal TA-EMGs for which we needed additional proof for a paralysis, we only had it in 56 cases, that is,
58.9%. Although our success rate improved over time (about 80% in 2016), we were not able to reach the same success rate as in TA-EMG. An overall success rate in PCA-EMG of about 60% seems not to advocate the method. However, alternative methods cannot replace a needle EMG. Surface electrodes are not able to reach the PCA, suffer from crosstalk of neighboring muscles, and are unable to detect pathologic spontaneous activity. Endoscopy to an extent also can describe lesion pattern in later stages, with the exception of the PCA. Standard imaging (computed tomography and magnetic resonance imaging (MRI)) can detect total atrophy in later/advanced stages of vocal paralysis. Newer 3 Tesla MRI imaging with analysis of fat content of vocal folds may improve this. But this does not help in early paralysis and in the prediction of prognosis.

There are good reasons not to abandon TA-EMG. First of all, TA-EMG technically is easier to perform; traces are easier to interpret; and well-preserved TA innervation appears to be a good prognostic factor for voice rehabilitation. This is worth knowing because, from our own experience, the innervation status of the TA not always is deducible from laryngostroboscopy. Finally, most published studies on the prognosis of recovery of gross vocal fold motion are based on TA-EMG data.

Prediction of recovery of vocal fold motion in cases of well-preserved TA innervation usually remains within the level of chance. Only Smith et al. were able to define criteria that were strict enough to predict a positive outcome in a small case series. Our data instead suggests that it is difficult to conclude on the condition of the PCA from the condition of the TA-innervation. Analysis of PCA-EMG might improve prognostic accuracy in the future. However, on first impression our data does not imply a simple answer. Further studies are needed to thoroughly correlate clinical outcomes with specific PCA-EMG findings.

Two methodic weaknesses should to be mentioned: First, there could be an investigator bias because these are raw clinical data without any blinded reevaluation. However, we do not think this is the case because we mainly discovered the differences retrospectively. Second, there is a lack of normal IFP density values for TA and PCA-EMG. This could distort a comparison of TA and PCA-EMG traces. From own experience and what is known from anatomical studies, the PCA motonic units are larger and fewer. However, this should not cause significant shifts in our coarse IFP grading system, and as a systematic error it likewise should affect paralysis cases of all etiologies.

We found a tendency toward predominant damage to PCA innervation in iatrogenic lesions (Figs. 1–4). Several explanations for this are conceivable:

1. The majority of iatrogenic cases of RLN paralysis were due to thyroid surgery, which is done with TA neuromonitoring in our referring centers. The finding could reflect a better protection of TA innervation during surgery. However, the monitoring used was not continuous, and the predominance of PCA damage was not limited to thyroid surgery cases; thus, this cannot be the only explanation.

2. Dionigi et al. identified and quantified different mechanisms of RLN trauma during thyroid surgery. The most common were traction (71%, mainly transmitted via Berry's ligament), thermal injuries (17%), and compression (4.2%), with different rates of recovery. Accidental transection was the rarest cause (1.4%). Traction applied to the RLN could have heterogeneous effects on different branches, exposing those to the PCA, which leave the nerve stem first and at a steeper angle, to greater shear forces than those to the adductors. RLN damages from carotid artery or spinal surgery should share similar pathophysiologic mechanisms.

3. The compensatory reserve of the PCA innervation could be less. Motor units of the PCA are larger and fewer than those of the TA, thus, even if the same fraction of nerve fibers were injured, the impact of it could be greater because the potential for reorganization/terminal sprouting is less.

4. A combination of these or unknown factors could be the cause of preferential damage to PCA innervation in iatrogenic lesions.

The use of intraoperative neuromonitoring of the RLN (usually TA monitoring only) has long been regarded as optional in thyroid surgery because it had not been shown to consistently decrease the rate of permanent paralysis in primary thyroid surgery compared with rates of experienced surgeons not using neuromonitoring. However, the quality and success of neuromonitoring has improved a lot with development and consequent implementation of standards.

<table>
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<tr>
<th>TABLE I. Demographic Data of the LEMG-Examined Patients.</th>
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<td><strong>Patient Variables</strong></td>
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<tr>
<td>Patients (female/male)</td>
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<tr>
<td>Age [years], (median)</td>
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<tr>
<td>Side of paresis (right/left/bilateral)</td>
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<tr>
<td>Duration of paresis [months], (median)</td>
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<td>Repeated LEMGs</td>
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LEMG = laryngeal electromyography.

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In noniatrogenic paralysis, the difference between residual PCA and TA innervation did not reach significance (Figs. 1 and 2) but may exist, larger studies are needed. The underlying pathomechanism of such a difference, if confirmed, seems even less clear. The number of PCA recordings in tumor-related nerve lesions in our study was limited, and results should be interpreted with caution.

The time of onset of pathologic synknetic reinnervation indicates the time a lesioned nerve needs for regeneration and should correlate with the distance from the lesion to the innervated muscle. According to our data, there seems to be a clear difference in the time of onset of synkinesia in iatrogenic and noniatrogenic RLN lesions, with delayed appearance of synkinesia in noniatrogenic lesions suggesting a more proximal lesion site (e.g., closer to or at the level of the vagal nerve stem) (Fig. 5). Again, the number of noniatrogenic data sets was small. To more clearly understand the time course of pathologic innervation, it would be necessary to have more data giving the possibility to differentiate between more time periods.

Figure 3 illustrates the fact that regeneration processes occur despite persisting vocal fold immobility. There is a shift toward denser interference patterns both in the TA and in the PCA over time. However, the difference between the muscles seems to persist, which partly might be explained by greater initial damage of PCA nerve branches but could also be due to preferential TA reinnervation as described by Woodson in a cat model with complete RLN transection. The reason for this could be related to differences in expressions of different neurotrophins.

Another unexplained finding is the higher incidence of left-sided nerve lesions in noniatrogenic, nonmalignancy-related RLN and vagal lesions (Table I). Whereas in iatrogenic lesions the right-sided approach of spinal surgeons and mediastinal involvement in malignancies account for the side predominance, there is no plausible explanation for the left side in noniatrogenic lesions. We did not see frequent left-sided cardiac failures, which might explain this (Ortner’s syndrome). May be lesions of the right and shorter nerve remain “subclinical” more often and therefore present less often.

Further evaluation of the prognostic value of PCA-EMG and the significance of synkinesia with larger studies and high-quality recordings are needed. We hope to achieve this in the future with an European LEMG registry using equal evaluation criteria and including training with new examiners.

CONCLUSION

Inclusion of PCA-EMG improves the diagnostic value of LEMG. However, the relevance of PCA-EMG for prognosis has yet to be shown. This study suggests preferential damage to PCA-nerve fibers in RLN lesions, particularly in iatrogenic lesions. In these cases, traction on the nerve is to be understood as the predominant mechanism of injury. Intraoperative neuromonitoring of the TA muscles alone may miss a partial RLN injury in some cases.

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