Prognostic Value of Laryngeal Electromyography in Vocal Fold Paralysis

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Objective: To analyze the value of electromyography in predicting recovery from acute neurogenic vocal fold paralysis.

Study Design: Prospective case series.

Setting: University-based hospital of otorhinolaryngology–head and neck surgery.

Patients: Ninety-eight patients (56 women, with a mean age of 62.2 years; 42 men, with a mean age of 39.8 years) with 111 paralyzed vocal folds. The causes were varied, with thyroid surgery (53 cases) and idiopathic palsy (18 cases) being the predominant factors.

Intervention: Prognostication was based on electromyography performed no earlier than 14 days after onset of palsy. Findings were classified as neurapraxy, axonotmesis, and neurotmesis. Prognosis is inherent in this classification, since neurapraxy is presumed to resolve completely within 8 to 12 weeks, whereas axonotmesis is most likely to be followed by impaired vocal fold mobility.

Main Outcome Measures: Vocal fold mobility after 6 months.

Results: In 102 vocal folds, some palsy of various degree persisted after 6 months. Free mobility of the paralyzed vocal fold was restored in 9 cases. By means of laryngeal electromyography, defective recovery, defined as absence of completely free vocal fold mobility, was predicted correctly in 94.4% of cases (68/72). For complete recovery, prognosis was accurate in only 12.8% of cases (5/39).

Conclusions: The detection of neural degeneration by laryngeal electromyography allows the prediction of poor functional outcome with sufficient reliability in an early phase of the disease process. Conversely, the absence of signs of degeneration does not imply that complete recovery is to be expected.


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PATIENTS AND METHODS

LARYNGEAL ELECTROMYOGRAPHY

Basically, electromyography evaluates the integrity of the motor system by recording action potentials generated in the muscle fibers. All 5 major laryngeal muscles (thyroarytenoid [TA], lateral cricoarytenoid, posterior cricoarytenoid, interarytenoid, and cricothyroid) lend themselves for electrophysiologic examination. In this article, only electrophysiologic data obtained from the TA muscle will be reviewed.

The TA muscle was approached in the awake patient either transcutaneously or transorally. For the transcutaneous approach, which has been described in detail elsewhere, bipolar concentric needle electrodes 45 mm in length are inserted in the region of the cricothyroid membrane after cleaning of the skin with alcohol. To evaluate the TA muscle, the needle was inserted in the midline right above the cricothyroid notch, a small depression immediately above the cricoid cartilage. After passing through the cricothyroid membrane, the tip of the needle was angled to the affected side laterally and superiorly 30° to 45°. If the patient coughed, which indicated penetration of the airway space, the needle was withdrawn and repositioned. An increase in LEMG activity while the patient was phonating validated correct electrode position. In addition, standard verification gestures to assist in localizing the position of needle electrodes were used, as described elsewhere in detail. However, if no muscle activity was detectable, electrode displacement could not be discriminated from complete vocal fold paralysis by the transcutaneous technique.

RESULTS

At least 6 months after onset of palsy, completely free mobility of the paralyzed cord was restored in 9 (8.1%) of 111 vocal folds. Twenty-two vocal folds (19.8%) recovered substantial mobility, and 30 (27.0%), minimal mobility, while 50 vocal folds (45.0%) remained immobile.

Phonatory function was normal or near normal again in 19 (19.4%) of 98 patients. Forty-five patients (45.9%) had fair voices and 14 (14.3%) had usable voices, while 50 vocal cords (51.0%) remained immobile.

The LEMG was used in all 98 patients on 111 paralyzed vocal folds. The first examination took place an average of 27 days after onset of palsy. At this point, all diagnostic criteria should have developed to allow a diagnosis according to the Seddon classification, which implies a prognosis based on the expected clinical course. Thirty-nine patients were diagnosed as having neurapraxia, while 72 laryngeal palsies were characterized as axonotmesis. Neurotmesis was found in no case. Table 2 gives a cross-tabulation of electrodagnostic classification and final functional recovery. Calculated on this basis, the predictive value for transoral LEMG was 37.5% (15/40) vs 14.3% (3/21) for transcutaneous LEMG. As expected, the differences are not statistically significant due to the small numbers of patients.

There are at least 2 aspects to prognosis of vocal fold palsy: vocal cord mobility and phonatory function. Imperfect function of the intrinsic laryngeal muscles may be compensated for, with only minimal impact on vocal ability. It is

For transoral LEMG, bipolar hooked-wire electrodes were used. The hooks at the end of these thin flexible wires act as barbs, keeping the electrode in place once positioned in the muscle. For endolaryngeal application, a specially designed device was used: a curved metal cannula on a handle-piece hiding a hollow flexible needle that contains the hooked-wire electrode (Inomed Co., Teningen, Germany). The device was inserted into the endolarynx under endoscopic guidance, with the needle tip being secured in the applicator. Surface anesthesia of the oropharynx and endolarynx was induced before the procedure. When the applicator was positioned correctly above the mediobasal aspect of the vocal fold, the tip of the needle was pushed into the TA muscle. Because of the hooks at the distal end of the wire, the electrode remained in position when the applicator was withdrawn gently. This technique allowed better control over electrode position than the transcutaneous approach but was more time consuming, significantly more expensive, and technically more difficult. In addition, the majority of patients feel transoral hooked-wire LEMG to be less tolerable. Therefore, we used transoral LEMG only when a transcutaneous approach was not possible, ie, in patients with tracheostomy or with a nonpalpable cricoid. In this series, 91 patients were studied transcutaneously vs 7 transorally.

Regardless of technique, electrophysiologic evaluation was performed according to the following criteria. The use of LEMG allows for distinction between normal silent resting potential, voluntary motor unit potential, spontaneous fibrillation potential, and polyphasic reinnervation potential. The absence of any electrical activity either on electrode insertion or on attempted voluntary motion is called electrical silence. Normal voluntary action potentials are diphasic or triphasic and are extremely variable in

few data exist regarding validity, reliability, and reproducibility of clinical electrophysiology of the recurrent laryngeal nerve. It is the aim of this study to assess the prognostic significance of LEMG in patients with vocal fold paralysis in a prospective design.
amplitude (Figure 1 and Figure 2). Spontaneous fibrillation activity is defined as involuntary potential generated by a single muscle fiber, indicating axonal degeneration (Figure 3). However, this symptom of degeneration does not appear earlier than 10 to 14 days after injury. Polysynaptic motor units have 4 or more phases and herald nerve regeneration (Figure 4).

Electrophysiologic findings were classified according to Seddon's into neuapraxy, axonotmesis, or neurotmesis. For neuapraxy, the diagnostic criteria on LEMG are detection of a rarefied recruitment pattern or single action potentials on voluntary action without fibrillation activity and without spontaneous positive sharp waves. Axonotmesis is suspected when spontaneous activity, indicating neural degeneration, can be detected. Usually, this is the case no earlier than 10 to 14 days after onset of paralysis. Prognosis is inherent in this classification, since neuapraxy is most likely to resolve completely within 8 to 12 weeks, while axonotmesis is thought to have only a poor chance of recovery to a functional level. If reinnervation occurs after axonotmesis, usually it is associated with sequelae such as synkinesia caused by neuronal misdirection.10,11 The result is simultaneous activation of abducting and adducting intrinsic laryngeal muscles. For the same situation in facial nerve disorders, the appropriate term autoparalytic syndrome has been coined.9 Neurotmesis, representing complete destruction of the whole nerve structure over its full diameter, apparently cannot be expected to resolve at all. All LEMG examinations were performed with a computer-based electrodiagnostic system (NeuroScreen Plus; Jaeger-Toennies Inc., Hochberg, Germany). Signals were monitored by means of a speaker, bandpassed between 20 and 10,000 Hz, and stored in a database for later analysis.

Well known that phonation can be unaffected in patients who have 1 or more vocal folds that abduct or adduct only weakly.10 In our study, free mobility of the paralyzed vocal fold was restored in only 8.1%, while 46.9% of all paralyzed vocal folds improved at least to a certain extent. This is roughly consistent with a previously published large series of recurrent laryngeal nerve palsies of different causes,11 in which complete and partial recovery was observed in 21.8% and 10.9%, respectively, of 165 cases. In that study, spontaneous voice recovery to a good functional level was observed in 76% of cases, which corresponds well with the 65.5% found in our series. With vocal fold mobility restoration in 40% and voice recovery in 90%, a similar report,12 focused exclusively on outcome of recurrent laryngeal nerve injury caused by thyroid surgery, found a comparable relationship. For idiopathic palsies, a rate of less than 50% for partial and complete recovery was reported by Ward and Berci.13 Also in their study, the return of voice was satisfactory in a significantly higher percentage (59%). Obviously, for the patient, outcome in terms of vocal ability is of greatest importance. However, when the accuracy of a prognostic test is investigated, the outcome variable should reflect directly the system measured. Therefore, this analysis is focused on vocal cord mobility as a key variable for outcome determination.

The clinical use of LEMG has been advocated for diagnosis and prognostication of vocal fold palsy for more than 2 decades.10,14-16 Surprisingly, there are few reports on the usefulness of this technique in terms of reliability and prognostic accuracy. Min et al17 reported an impressive prognostic accuracy of 89% in patients with unilateral vocal fold palsy. Their study is seriously flawed for at least 2 reasons: first, the study population comprised 14 individuals only. Second, criteria for prognostication were defined very generously: a positive prognostic for laryngeal recovery was assumed when morphologic characteristics of the motor unit waveform were normal and significant persistent overall electromyographic activity as well as no electrical silence during voluntary tasks could be found. Obviously, this combination of favorable findings should be considered more an indication of a discrete lesion than an actual prognosis. Another study investigating 18 patients18 was not truly focused on the prognostic accuracy of LEMG. The authors of that study concluded cautiously that LEMG may be of prognostic value.

Our data suggest that detection of neural degeneration in LEMG allows for the prediction of poor functional outcome with high reliability in an early phase of the disease process. This can be extremely valuable for the timing of a definitive surgical intervention. In a study on patients with bilateral vocal fold palsy conducted by Eckel et al,19 the routine use of LEMG was helpful to decide on partial cordectomy at a comparatively early stage.

**PATIENT POPULATION**

In the electrophysiologic unit at the Department of Otorhinolaryngology, Head and Neck Surgery at University of Cologne, Cologne, Germany, 540 LEMG studies were performed between May 1, 1995, and September 30, 1998. For the 98 patients analyzed in this study, follow-up was complete during at least 6 months. By means of videostroboscopy, final vocal fold mobility was classified in 4 groups: free mobility when movement was normal compared with the unaffected side, substantial mobility when the affected vocal cord was moving in a clearly reduced but functional way, minimal mobility, when vocal folds moved in a markedly reduced way without obvious functional effect, and no mobility when the affected vocal fold stood completely still or showed only signs of passive movement. In 85 of these 98 cases, vocal fold palsy was unilateral, and bilateral immobility was present in 13 patients, for a total of 111 paralyzed vocal folds. The LEMG was performed on day 14 after onset of paralysis, the average delay was 27 days. For all cases, reliable and exact information on the beginning of the disorder was available. Fifty-six patients (37%) were women and 42 (43%) were men. Mean age was 62.2 years for women and 39.8 years for men. Data regarding cause of paralysis show that surgery involving the thyroid and neck contributed the greatest to the overall number. In the female patients, thyroid surgery was by far the single most important causative factor. In the male study population, thyroid surgery, followed by cervical vascular surgery and idiopathic lesions, accounted for the majority of vocal fold palsies. All 13 cases of bilateral paralysis were caused by thyroid surgery. Table 1 gives a detailed overview of nerve injury causes, separated for men and women.
As a consequence, tracheotomy was required in only 21% of the study population. On the other hand, the absence of signs of degeneration may not lead to the assumption that complete recovery is to be expected. This interpretation needs to be considered under various aspects to determine its validity.

STATISTICAL CONSIDERATIONS

To evaluate the validity of LEMG as a clinical test, the PPV and NPV, representing statistical standard tests, were calculated. In the concepts of PPV and NPV, the denominators are based on subjects with positive and negative test results. The formula for the PPV is (subjects with positive test and disease)/(all subjects with positive test). For the NPV, the general formula is (subjects with negative test and no disease)/(all subjects with negative test).

Thus in general, the prevalence of disease has a profound effect on the usefulness of a test. If the prevalence is low, the PPV of the test is low. Conversely, if the prevalence of disease is high, the PPV is high but the NPV is low. This is the situation we have with the data in this study. Most of the study population (102 of 111 vocal cords) had incomplete recovery of vocal fold mobility. An event occurring so frequently is much easier to predict than the rare condition, at least in our population, of complete recovery. Therefore, the PPV of 94.4% is not as good and the NPV of 12.8% is not as bad as they might seem at first glance.

TECHNICAL CONSIDERATIONS

There are no generally accepted standardized guidelines for LEMG, nor does a formal education exist for the laryngeal electromyographer. Even in centers with long experience in LEMG, the first question before data interpretation should be whether data were sampled in a technically appropriate and reliable manner. Since the ability and experience of the investigator are crucial, only a critical evaluation of the test routine can help to prevent systematic error.

Bipolar electrodes with both poles contained in the center core, as used in this study, have been demonstrated to have the highest recording quality. In LEMG, electrode displacement is probably the most critical point. When the transcutaneous technique is used, there are no clear-cut criteria to separate true electrical silence from electrode displacement. Transorally inserted hooked-wire electrodes allow better control of position by visualization with indirect laryngoscopy. However, since depth...
of insertion through the lamina propria of the vocal fold cannot be monitored, electrode displacement still can occur. In addition to the fact that in our department LEMG has been in clinical use in large numbers since 1986, we have another reason to believe in the technical accuracy of our study: during LEMG, the investigator is searching first for signs of neural degeneration, which is the most important symptom to establish a diagnosis. Frequent electrode displacement or wrong reading of the potentials recorded should lead to a great number of false-negative results. However, in our population, individuals who had been diagnosed as having neural degeneration actually had an unfavorable functional outcome. It was in the group without signs of degeneration where a surprisingly high number of vocal folds did not return to normal mobility. To blame undetected neural degeneration for these results would suggest that laryngeal nerve palsy is nearly always associated with neural degeneration. However, this is inconsistent with the published literature.17,18,23,24

**PATHOPHYSIOLOGICAL CONSIDERATIONS**

Laryngeal biomechanics and neural control are probably far more complex than anticipated until recently.25 Even in healthy subjects, considerable variation in firing patterns between motor units within the same muscle as well as in vocal fold movements producing the same speech sound could be demonstrated. Thus, our anatomic models serving as a basis for interpretation of LEMG data may be much too simple. There is increasing evidence of the existence of physiologically distinct compartments within the laryngeal muscles. As a consequence, detection of neural degeneration does not necessarily reflect a representative picture of the situation of the whole functional unit.26 Thumfart14 hypothesized early that degenerative and nondegenerative palsy may exist in parallel in the same vocal fold.

The outcome measure in this study was free vocal fold mobility, not a normal LEMG. A case was coded as showing complete recovery only when there was no sign of impaired mobility. Minor and minimal dysfunction of vocal fold movement is not exclusively attributable to a neural disorder. The cricoarytenoid joint, as a delicate counterpart in laryngeal movement, may also play an often neglected role in the pathophysiology of vocal fold palsy. It may be speculated that prolonged vocal fold immobility may lead to some kind of fibrosis of the cricoarytenoid joint. In theory, this might cause a persistent deficit in vocal fold mobility after neurogenic palsy even despite completely recovered neural supply. This model might explain elegantly the gap between the high PPV and low NPV calculated from our data. Patients with degenerative palsy do not regain undisturbed vocal fold mobility, as predicted by LEMG. However, for cases without signs of neural degeneration, there may still be a risk of developing cricoarytenoid fibrosis because of immobilization over several weeks. Although the clinical result is similar—the vocal fold cannot move freely—the underlying mechanism would be different. Its delicate anatomy,27 sophisticated physiologic characteristics28,29 and similar susceptibility30,31 to degenerative changes raise the question why the cricoarytenoid joint should be less prone to malfunction and fibrosis induced by prolonged immobilization than any other joint in the human body. Friedrich32 formulated this hypothesis, which may find strong support in our data.

**CONCLUSIONS**

In patients with vocal fold palsy of the lower motoneuron type, the detection of well-defined signs of neural degeneration on LEMG allows for the prediction of poor functional outcome with high reliability in an early phase of the disease process.

In the presence of signs of neural degeneration detected by LEMG, the decision for definitive surgical interventions such as thyroplasty or vocal fold augmentation in unilateral or partial chordectomy in bilateral vocal fold paralysis can be made safely at an earlier stage of the disease process. Thus, LEMG can be helpful to significantly shorten the process of voice rehabilitation.

However, the absence of degenerative alterations in LEMG does not necessarily indicate recovery to a normal or near-normal functional level. Hypothetically, these findings may reflect secondary fibrosis of the cricoarytenoid joint after prolonged vocal fold immobility of primarily neurogenic origin.

In summary, LEMG is a valuable tool in the workup of patients with vocal fold palsy. Since prognostic accuracy for favorable results is comparatively low, LEMG cannot replace clinical monitoring for at least 6 months or until complete recovery has been reached.

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**Table 1. Causes of Vocal Fold Paralysis by Sex**

<table>
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<tr>
<th>Sex, No.</th>
<th>M</th>
<th>F</th>
<th>Total</th>
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<tbody>
<tr>
<td>Idiopathic</td>
<td>8</td>
<td>10</td>
<td>18</td>
</tr>
<tr>
<td>Thyroid surgery</td>
<td>13</td>
<td>40</td>
<td>53</td>
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<tr>
<td>Cervical vascular surgery</td>
<td>7</td>
<td>0</td>
<td>7</td>
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<tr>
<td>Cervical spine surgery</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Neck surgery</td>
<td>3</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Chest surgery</td>
<td>6</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Skull-base surgery</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>42</td>
<td>56</td>
<td>98</td>
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</tbody>
</table>

**Table 2. Diagnosis at First Examination vs Actual Outcome**

<table>
<thead>
<tr>
<th>Electrophysiologic Diagnosis, No. *</th>
<th>Neurapraxy</th>
<th>Axonotmesis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete recovery</td>
<td>5</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>Recovery with sequelae</td>
<td>34</td>
<td>68</td>
<td>102</td>
</tr>
<tr>
<td>Total</td>
<td>39</td>
<td>72</td>
<td>111</td>
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*Total of 111 vocal cords in 98 patients.
REFERENCES


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