REVIEW ARTICLE

Electromyography of the posterior cricoarytenoid muscles: a consensus guideline

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Abstract

Purpose Since the introduction of transcutaneous-transcricoidal needle approaches, electromyography (EMG) of the posterior cricoarytenoid muscle (PCA) became easier to perform and teach. Among the Neurolaryngology working group of the European Laryngological Society, several centers have adopted PCA EMG as part of their routine EMG workup in vocal fold immobility collectively gathering long-term experience. The purpose is to give an update and an extension to already existing guidelines on laryngeal EMG with specific regard to PCA EMG.

Methods Consensus of all co-authors is based on continuous exchange of ideas and on joint laryngeal EMG workshop experiences over at least 7 years. A Delphi method of consensus development was used, i.e., the manuscript was circulated among the co-authors until full agreement was achieved.

Results Step-by-step instructions on how to perform and interpret PCA EMG are provided.

Conclusions Further research should include the establishment of normal values for PCA and thyroarytenoid muscle (TA) EMG as well as studies on the nature of some unusual activation pattern commonly seen in chronically lesioned PCA.

Keywords Posterior cricoarytenoid muscle · Laryngeal electromyography · Guideline · Review · Aberrant reinnervation

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Introduction

The recent progress in dynamic rehabilitation therapies for bilateral vocal fold paralysis (VFP) such as selective reinnervation or laryngeal pacing has drawn the function of the posterior cricoarytenoid muscles (PCA) more into the focus of neurolaryngologists. This triggered our interest in electromyography (EMG) of the PCA. The scope has widened over time and PCA EMG became part of routine laryngeal electromyography (LEMG) diagnostics in our neurolaryngology clinics.

Since introduction of LEMG by Feinstein [1] in the mid-1940s and Faaborg-Anderson [2] in the mid-1950s, different approaches for PCA EMG have been described. Initially, transoral approaches were used which later were refined with the use of hooked wire electrodes and special applicators [1, 3, 4]. Hiroto et al. used transcutaneously inserted needles, advancing the needle via the cricothyroid membrane, vocalis muscle (thyroarytenoid; TA) and lateral cricoarytenoid muscle (LCA) into the upper lateral part of the PCA [5]. A transcutaneous lateral approach as

described by Hillel [6] involves a manual rotation of the larynx with a more or less direct puncture of the PCA with the needle approaching around the cricoid and the inferior horn of the thyroid cartilage. This method is widely used for applications of botulinum toxin into the PCA. In recent years, most centers have switched to a more median transcutaneous-transcricoidal approach first described by Mu and Yang in 1990 [7] which is easier to learn, provides good needle support thanks to the cricoid cartilage, works better in obese patients than the lateral approach, and in our experience is less disturbing for the patient especially if used with local anesthesia.

In 2012, members of the Committee on Neurolaryngology of the European Laryngological Society published a consensus guideline mainly focused on TA EMG [8]. Now, the authors of this paper would like to concentrate on EMG of the only vocal fold opener, the PCA, summarizing our experiences with different approaches, useful maneuvers, pitfalls and signal interpretation. The recommendations laid out in this paper represent a consensus of co-authors from six involved centers in Germany, Austria, Hungary and Spain. There has been a long-term collaboration between these centers including efforts to standardize teaching of LEMG procedures and LEMG interpretation [9]. A literature review was done on Pubmed using the terms 'electromyography', 'larynx' and 'posterior cricoarytenoid muscle'. The manuscript was circulated among all authors in four rounds. The Delphi technique was used until a consensus was reached for all recommendations. These recommendations are also summarized in Table 1.

Indications and contraindications for PCA EMG

Since recurrent laryngeal nerve (RLN) lesions seem to affect different intrinsic laryngeal muscles to different extents [10–12], we recommend a comprehensive sampling of TA, PCA and cricothyroid muscle (CT) in patients who tolerate the procedure. In cases with vocal fold immobility and normal/near normal TA EMG, a pathologic PCA EMG can provide proof of an RLN lesion and thereby exclude mechanical fixations due to trauma or rheumatoid arthritis. In cases with abductor type or mixed spasmodic dysphonia, PCA EMG can help to identify which side is affected most. At the same time, botulinum toxin injection into the PCA can be guided by EMG [13, 14]. So far, there is no evidence for a possible prognostic value of PCA EMG in prediction of recovery of VFP.

Bleeding disorders/anticoagulant therapy in combination with high blood pressure or high risks of local infections are relative contraindications for PCA EMG. Bilateral VFP is no contraindication but post-procedure monitoring should be considered.

Equipment and needles

We recommend a multichannel EMG amplifier with three or more channels to include additional traces with a thermal element and/or thorax expansion belt for respiration detection as well as an audio track. These multichannel recordings enable a delayed or repeated independent analysis. We recommend the use of bipolar concentric needles for all transcutaneous sampling techniques. For better needle guidance and cartilage penetration they have to be sturdy enough, 75 mm × 0.65 mm (23 G), in women up to 40 years of age a 50 × 0.45 mm (26 G) needle may be sufficient (Fig. 1). With experience, the choice of needle may change. Numerous suppliers exist on the market. For transoral approaches, we recommend the use of bipolar hooked wire electrodes. A reliable and convenient system with a long, bent applicator can be purchased from Inomed, Emmendingen, Germany. For more elaborate explanations about the equipment, we would like to refer to the European guidelines on LEMG [8].

Transcutaneous-transcricoidal approach

The transcutaneous-transcricoidal approach to the PCA has become the preferred method in all participating centers, even for botulinum toxin injections (Figs. 2, 3). It is tolerated much better with the use of local anesthesia (sterile 2% or better 4% lidocaine or an equivalent). A 23 G cannula may be used to apply a small subcutaneous depot and about 2 ml in squirts in the subglottic laryngeal lumen, thinner cannulae may be used but tend to bend during inevitable patient swallowing maneuvers. For needle insertion through the cricothyroid lamina, it is best to choose the midline with slightly caudal orientation. Before injecting local anesthetic, needle positioning should be checked by air aspiration. Patient coughing should be encouraged between repeated squirts to spread the local anesthetic around the larynx.

With the larynx anesthetized, a 23 G (or 26 G) EMG needle is inserted in the same way through the cricothyroid lamina. Once the needle tip has reached the subglottic airway, a 'white noise' or 'air sound' becomes audible on the EMG machine. The needle has then to be orientated to the correct height and about 20°–30° laterally. The position of the cricoid lamina and, therefore, the position of the PCA compared to the cricothyroid notch can be quite variable. In most cases, a 90° angle between needle and neck skin line is best, for large high larynges, the needle should be orientated more cranially, and for small more cranially positioned larynges, the needle should be held more horizontally. The needle is then advanced to the

Table 1 Step-by-step recommendations

Step	Recommendations
1	Prearrangements:
	Constant reproducible conditions, room with optimal electrical shielding, regular control of equipment, an adjustable chair/examination couch for the patient are recommended
	Multichannel recording (EMG, respiration via thermal element and/or chest belt, sound)
	Amplifier settings: 100 µV/10 ms/div, filter 10 Hz–10 kHz (filter settings for respiration 0.02–20 Hz, for voice 50 Hz–1.5 kHz, necessary amplifications dependent on sensor)
2	Local anesthesia:
	About 2 ml of sterile 2 to 5% lidocaine (or equivalent), 23 gauge cannula, small subcutaneous depot, penetration of cricothyroid membrane, air aspiration (!), 2–3 small aliquots squirts, allowing patient to cough in between
3	Examination of TA and CT if necessary
4	Transcutaneous-transcricoidal needle insertion into PCA:
	Palpation of cricothyroid membrane, insertion of needle through it into subglottic airway, redirecting needle rectangular to the skin line or higher (higher in high male larynges) and 30° to the side of PCA to be sampled, advancing needle, with cartilage contact fine drilling movements, advancing needle slowly not more than 3–5 mm, listen for change of signal character (dull relative silence in the cartilage > crisp sounds in the muscle)
5	The sequence of *maneuvers* and *signal evaluation*:
	1. Insertion activity is difficult to discern, because the needle moves very little in the muscle, it should be graded as follows:
	(a) No activity
	(b) Normal activity (<300 ms)
	(c) Increased activity
	(d) Highly increased activity
	2. Quiet breathing (15–30 s)
	3. Forced inspiration (3 times)
	4. Normal effort, normal-pitch sustained phonation (3 times, long enough to judge on baseline and synkinesis)
	5. Swallow maneuver (1–3 times)
	Baseline is analyzed for *pathologic spontaneous activity*:
	If baseline is obscured by action potentials use phonation and swallow maneuver
	(a) No reproducible pathologic spontaneous activity
	(b) Little pathologic spontaneous activity
	(c) Moderate pathologic spontaneous activity
	(d) Dense pathologic spontaneous activity
	Agonistic muscle activity during inspiration/forced inspiration:
	(a) No activity
	(b) Single fiber pattern (1–3 different active motor units visible per field of view)
	(c) Severe decreased recruitment pattern (50% or more of baseline visible)
	(d) Mildly decreased recruitment pattern (less than 50% of baseline visible)
	(e) Normal/dense recruitment pattern (no visible baseline)
	Antagonistic muscle activity (synkinesis) during normal-effort phonation:
	(a) No antagonistic activity
	(b) Sparse antagonistic activity
	(c) Clear antagonistic activity (antagonist/agonist ratio of 0.65 or higher)
	(d) Strong antagonistic activity (more antagonistic activity than agonistic activity, antagonistic activity as dense as "mildly reduced" interference)
	If there is no change in activity during respiration or phonation but silence during swallow consider repositioning of needle (retracting slowly if too deep or new position)
	The *waveform of muscle action potentials* is also classified as:
	(a) Normal biphasic motor unit potential
	(b) Early (sometimes polyphasic) reinnervation potentials with low amplitude and long duration
	(c) Giant polyphasic reinnervation potentials with high amplitude and long duration
	(d) Myogenic polyphasic potentials with low amplitude but in many cases normal duration

mucosal surface of the cricoid lamina. The cartilage is penetrated by pushing and fine oscillating drilling movements until the relatively quiet and dull EMG sound from within the cartilage gets crisper. In some cases, a tactile loss of resistance can be felt. Cartilage resistance is important for orientation. A thick and rigid cartilage is a good indicator of correct needle position. If no cartilage resistance is encountered, the needle is usually too low.

Cartilage thickness rarely ever exceeds 3–5 mm. Avoid advancing the needle too deep as it may penetrate the hypopharynx or even the prevertebral soft tissues. In this case, the patient will complain of a strong neck pain or

Fig. 1 Selection of recording electrodes, top: long needle with hooked wires with transoral applicator below, bottom: two sizes of bipolar concentric needles, 75 mm x 0.65 mm (23G) with red handle, 50 mm x 0.45 mm (26G) with yellow handle
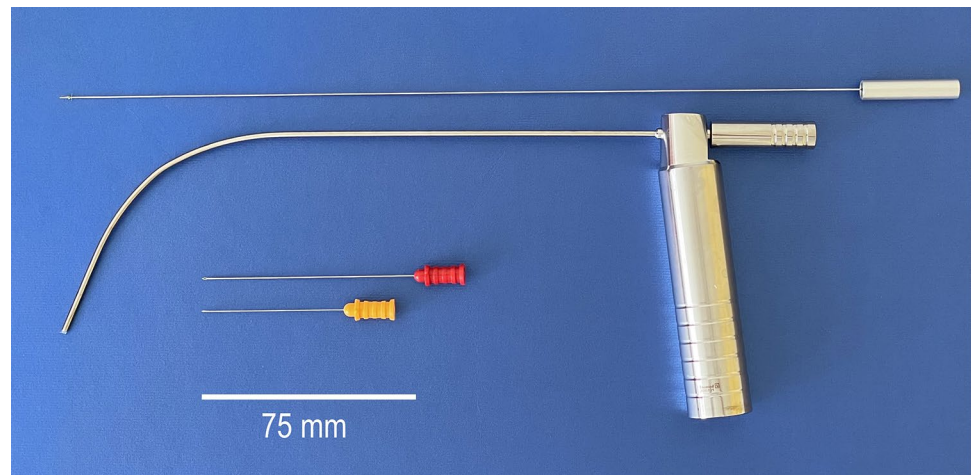

Fig. 2 Pathway of EMG needle with transcutaneous-transcartilagenous approach in a sagittal section of the larynx: the needle is orientated rectangular to the neck skin line or slightly upwards, after penetration of cricothyroid lamina a white noise/"air sound" becomes audible (1), within the cartilage the sound is dull/relative silence (2), thick cartilage indicates a good position, directly after the cartilage the PCA muscle signal is picked up, getting "crisp" within 1–2 mm (3); PCA in bright red; *cricopharyngeal muscle (CP) caudally continuing into the upper esophageal sphincter and esophageal musculature; between muscles is a thin layer of connective tissue and a vessel plexus indicated by blue and red dots; # prevertebral fascia
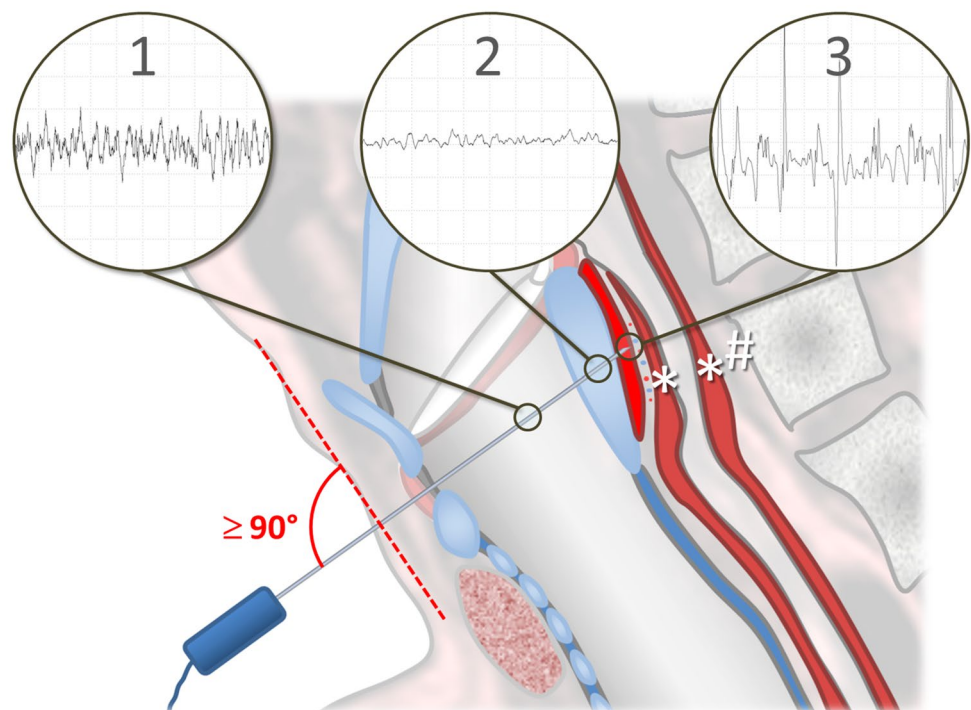
sudden urge to clear the throat. If this happens, the needle needs to be retracted immediately. The needle should not be advanced much further than 3–5 mm after cartilage is encountered, the overall insertion depth depends on the size of the larynx and thickness of the subcutaneous fat layer and varies between 35 and 45 mm.

For the beginner, it can be helpful to visualize the approach of the needle through the subglottic space with videolaryngoscopy. The applied local anesthesia usually helps to get a closer view.

Alternatively, the needle can be inserted in a more paramedian manner, through the cricothyroid membrane to stay submucosally during passage through the subglottic cleft and lower parts of TA before penetrating the cricoid plate towards the PCA. This approach has been preferred in Vienna. The lateral angulation must be reduced to compensate for the lateral insertion point. This approach is likely to be more tolerable for the patient because mucosal puncture with the needle can be avoided. However, it might be slightly more difficult for beginners since endoscopic control of needle guidance is not possible.

If used for botulinum toxin injection into the PCA, some needles may get blocked by small pieces of cricoid cartilage. Applying a slow and steady pressure allows the liquid to trickle past the cartilage plug in most cases. The extracartilagenous approach should be preferred in case of failure of the transcricoidal approach.
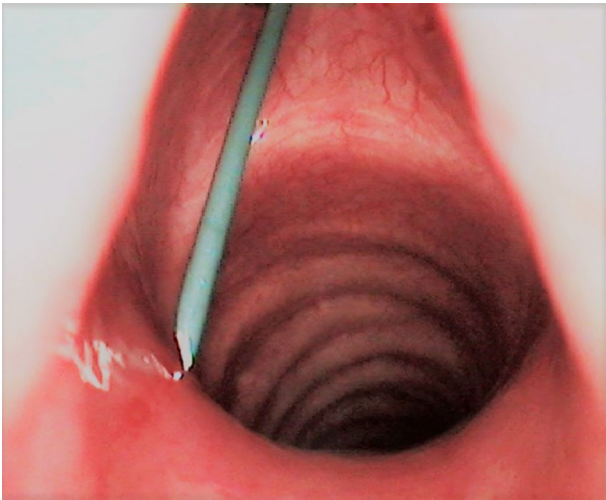
Fig. 3 Local anesthesia allows close up view with flexible endoscopy to monitor the approach of the needle through the subglottic air way towards the cricoid lamina, in this case a 23G botulinum toxin injection needle

Simultaneous LEMG tracing of two different laryngeal muscles is possible by leaving the transcartilagenous recording needle in place and using a second needle to probe another muscle.

Lateral transcutaneous approach

For this approach, the larynx has to be manually rotated to the opposite side as much as possible. The needle which might be slightly bent near the tip is then inserted toward the region of the cricothyroid joint which may be palpated as a slight notch. Once the dorsal border of the thyroid lamina has been reached, it has to be redirected towards the PCA with close contact to the cartilage either around the dorsal border of the thyroid lamina or, if approaching from below the joint, more in an upwards and medial direction. In some cases, it is possible to pass the needle above the joint in the small space between the cricoid and the inferior horn of the thyroid. Once the PCA has been reached it has to be held in place carefully for placement control and data recording.

This approach is widely used by others for botulinum toxin injections because there is no cartilage penetration and, therefore, no risk for needle blockage. However, twisting the larynx and scratching the perichondrium with the needle tip can be very uncomfortable for the patient. There is a certain risk for mucosal penetration into the piriform sinus or even puncture of main neck vessels. The needle has to be held in place for the whole recording. There are limits for this approach in obese patients and patients with low positioned larynges.

Transoral approach

This method is suited best for longer duration or multiple muscle multichannel recordings as well as needle placements in the operating theater. In an awake setting, it requires thorough local anesthesia of the oropharynx, tongue base and endolarynx. For the final intralaryngeal application of local anesthesia, a long, bent cotton wool swab may be used. This gives the examiner an impression whether the patient is ready to tolerate the coming procedure. Under laryngoscopic vision, with a 70° or 90° magnification optic rod, a pair of hooked wires are inserted postcricoidally and advanced for another 2 cm downwards. In general anesthesia, a regular straight suspension laryngoscope is used for electrode insertion into the same region.

The needle position cannot be corrected, hooked wires tend to be more expensive and signal quality is sometimes somewhat lower than with concentric needles.

Needle placement control and standardized sample recording of PCA

Correct needle position is indicated by an increase of activity during a forced sniff (the agonistic maneuver of the PCA) and a strong decrease of activity/cessation of muscle action potentials during phonation. If damage to the PCA innervation is advanced, no regular agonistic muscle action potentials may be visible. In those cases, pathologic spontaneous activity, audible as a typical very rhythmic 'rain on tin roof sound' may be the only indicator of correct needle tip placement in the target muscle.

According to the general LEMG guidelines [8], we recommend recording a period of quiet breathing (about 30 s), three forced sniff maneuvers, and three short sustained phonations. Phonation should be done with normal-effort and normal-pitch of the vowels [a:] or [i:], although the laryngeal elevation with [i:] may increase the risk of coughing during the examination. In addition to forced sniff and phonation, we recommend 1 or 2 *swallow maneuvers*.

For best identification of small signals, the EMG amplifier should be set to 100 μV per division with 200 ms flip time (10 ms per square). For a better overview, e.g., for evaluation of interference pattern (IFP) density/recruitment, amplifier settings can be changed to 500 μV per division and 2 s flip time.

Success rates and complications

Success rate and patient tolerance of PCA EMG using the transcutaneous-transcricoidal approach are similar to those for TA EMG. The use of local anesthesia, although a burden in itself, is key to improving acceptance and allows a repositioning of the needle if necessary. Most of the patients would

tolerate a follow-up examination for good reason although follow-up examinations are not regularly performed in any of the participating centers.

Minor complications comprise minor bleedings and varying degrees of discomfort which may last a few hours and is mainly felt during swallowing. Severe complications such as cricoid perichondritis, severe pharyngeal or prevertebral hematoma or prolonged bleeding are rare and may be related to additional risk factors such as immune suppressed conditions or increased risk of bleeding. The risk of glottic swelling/obstruction is lower than with TA EMG. However, in cases of bilateral VFP, inpatient patient monitoring should be considered.

Signal interpretation

Analysis of traces is performed in accordance with the proposed European guidelines [8]. In most participating centers, clinical registries to collect and analyze LEMG data have been established. For further information, evaluation forms and signal examples, we recommend checking out the website of the Neurolaryngology Working Group: www.lemg.org.

Usually, there is very little insertion activity discernible because the needle enters the muscle slowly and is steadied by the cartilage so that there is very little movement at the needle tip. Sometimes rotating the needle can elicit some artifacts similar to insertion activity. For signs of hyperexcitability/denervation, it is, therefore, best to rely on pathologic spontaneous activity.

Baseline There is always a round-shaped undulation of baseline which becomes visible during phonation or glottal stop. This background noise, probably originating from the cricopharyngeal muscle (CP), typically ceases during swallowing. As a result of background noise, low amplitude signals like pathologic spontaneous activity are less readily visible or audible than in TA (Fig. 4B, C). Thus, the swallow maneuver can help to uncover pathologic low amplitude signals (Fig. 6D).

Action potentials of a healthy PCA are somewhat larger than those of TA. They do exceed screen height in high-resolution EMG amplifier settings (Fig. 4A) and may reach 2.5–3.5 mA. Interference pattern (IFP) densities during maximum effort (forced sniff) are somewhat less dense than in TA (Figs. 4A, 5A). This is an expected finding due to the larger size of motor units compared to TA and the lesser number of nerve fibers innervating the PCA [15–17].

For assessment of acute or chronic (neurogenic) damage, pathologic spontaneous activity as well as signal density during agonist, antagonistic and swallowing maneuvers should be evaluated. There is no generally accepted normative data for PCA or TA EMG signal interpretation. Our recommendations are based on consensus agreement.

Pathologic spontaneous activity, as a sign of axonal damage and, therefore, poorer prognosis, becomes visible/audible 10–14 days after a nerve lesion. It is usually a low amplitude, short duration signal with a high rhythmicity, mainly in the form of positive sharp waves and fibrillation potentials. The small repetitive sharp signals are intermingled with steady background noise from the pharyngeal constrictor muscle (CP) which only ceases during a swallowing maneuver (Fig. 4B). In partial lesions, it might not be visible among the action potentials during respiration but is unveiled during phonation or swallowing. The sound is described as a very rhythmic 'rain on tin roof' sound. Pathologic spontaneous activity may be graded as absent–sparse/occasional–moderate–dense (screen filling). For an accurate estimate, multiple locations should be sampled, which is not feasible in most cases [8].

Interference pattern density The use of IFP density during maximum effort [18] as a recruitment test is considered unreliable by neurophysiologists but is established as the most feasible method in LEMG. Since increases in temporal recruitment (motor units firing with a higher rate) compensate for loss in spatial recruitment (actual reduction of number of active motor units), changes in interference pattern density become obvious in lesions involving more than 50% of nerve fibers [19]. Gradings do not correlate well with fiber losses. With the knowledge of these limitations, we use the same empiric grading as for the TA according to the above-mentioned guideline. We grade signals as 'dense' (no visible baseline), 'mildly decreased' (less than 50% of baseline visible), 'moderately/strongly decreased' (more than 40–50% of baseline visible), 'single fiber activity/pattern' (1 to 3 discernible motor units per 200 ms flip screen) and 'no voluntary activity'.

In acute and chronic RLN lesions, the differences in IFP densities between PCA and TA become more evident especially in iatrogenic nerve lesions. Compared to TA innervation, this seems indicative of a higher vulnerability and a lesser propensity for regeneration of PCA innervation [10, 11, 20–22].

Pathologic/synkinetic reinnervation Other than in TA, there is still no clear evidence of regular physiologic antagonistic activity in healthy PCA or acute RLN lesions during normal-effort normal-pitch sustained phonation in the majority of patients (Fig. 5A, B) [6, 11]. Therefore, any antagonistic activity found may be considered as synkinesis. However, there seem to be a few cases that do show such physiologic antagonistic PCA activation especially with raised pitch (PCA counter balancing the pull of CT onto the arytenoid). This has been previously observed by other groups too [6]. The rate this occurs is not known yet. Since it is not a constant feature, it would seem difficult to define cut off values as in TA EMG [23].
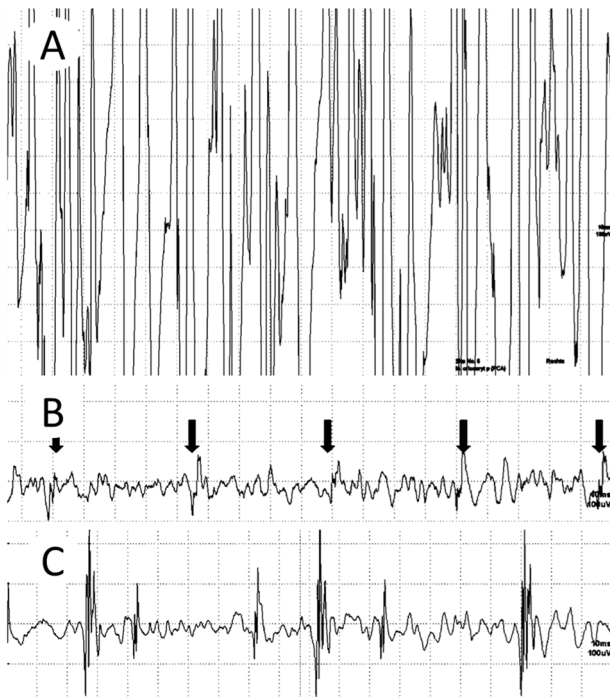
Fig. 4 PCA traces in high resolution (10 ms/100 µV per square): **A** a normal/dense interference pattern during respiration, action potential exceed height of the screen, no visible baseline, **B** during phonation an rounded undulating baseline caused by background noise of the CP becomes visible, any low voltage pathologic activity appears interspersed in it and has to be carefully looked for (black arrows), **C** in higher grade damages (single fiber pattern with small polyphasic action potentials) CP background noise is visible throughout the recording

The majority of patients with chronic RLN lesions (94.5% of cases with PCA damage longer than 1 year [11]) develop pathologic/synkinetic reinnervation either mainly during phonation (Fig. 5C) or more constantly during respiration as well as phonation (Fig. 5D). This synkinetic activity usually has lower amplitude than any residual orthotopic PCA activity.

In accordance with the above-mentioned guidelines and work on TA synkinesis done by Statham et al. [23], we recommend grading synkinesis as 'absent' (no antagonistic activity), 'mild' (just about noticeable activity up to an antagonistic/agonistic activity ratio of about 0.65), 'moderate'(antagonistic/agonistic activity ratio between 0.65 and 1) and 'strong' (antagonistic activity stronger than agonistic activity but at least as dense as'mildly decreased' IFP).

Healthy PCA is quiet during a swallow except for a short mid-swallow burst in a number of patients (Fig. 6B, C, white arrows). If present, synkinetic reinnervation is seen as activity during the entire swallow (Fig. 6D). Early/low amplitude synkinetic activity is best seen during a swallow because the baseline flattens due to the cessation of CP background noise.
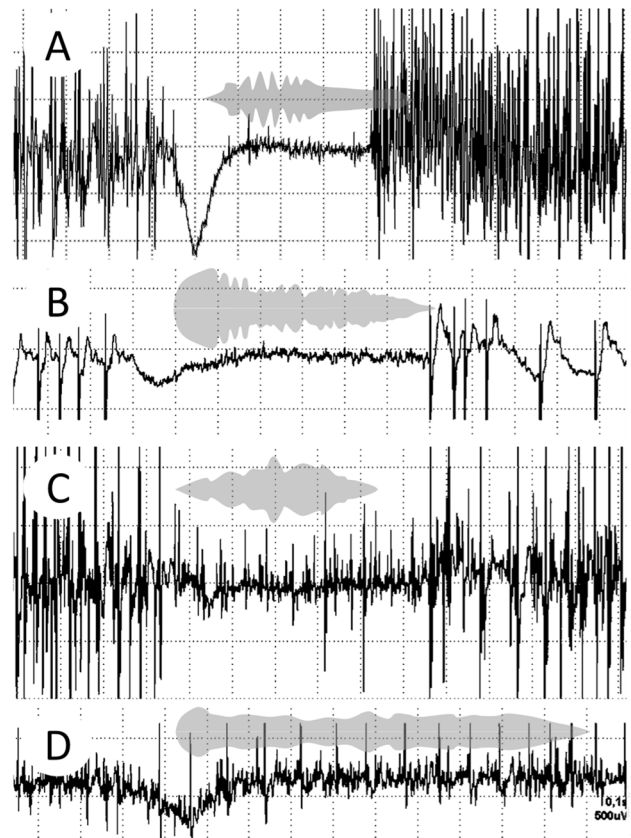

Fig. 5 Example PCA EMG traces during phonation in lower magnification (0.1 sec/500 µV per field, hull curve of sound as grey overlay above each trace): **A** normal PCA, no active motor units during phonation, only background noise and three artefacts are visible, **B** fresh RLN lesion with single fiber activity during respiration and background noise during phonation, **C** chronic VFP with mildly reduced interference during respiration and moderate dense synkinetic activity during phonation, synkinetic action potentials often have a smaller size, **D** chronic VFP with almost constant, smaller size PCA activity during respiration and phonation (and silence during swallow) which could indicate a reinnervation with nerve fibers originally innervating the CP

In some patients with chronic RLN lesions, the PCA has a more constant activity with very little variation during forced sniff or phonation (Fig. 5D). This activity usually stops during a swallow maneuver (Fig. 6A). This activation pattern of PCA is not visible in fresh RLN lesions but visible to some extent in about half of all chronic RLN lesions (in Gera 50%, 31 of 62 cases in a series with paresis of 6 months or longer, unpublished data). This activation pattern seems equal to the CP pattern [24]. The authors cannot distinguish between a misguided needle that samples CP instead and a pathologic reinnervation of the PCA by CP-related nerve fibers. However, since the CP receives nerve input via the RLN too [25], it seems not unlikely that this represents some form of synkinetic reinnervation too. Otherwise, this pathologic reinnervation could also arrive via intralaryngeal
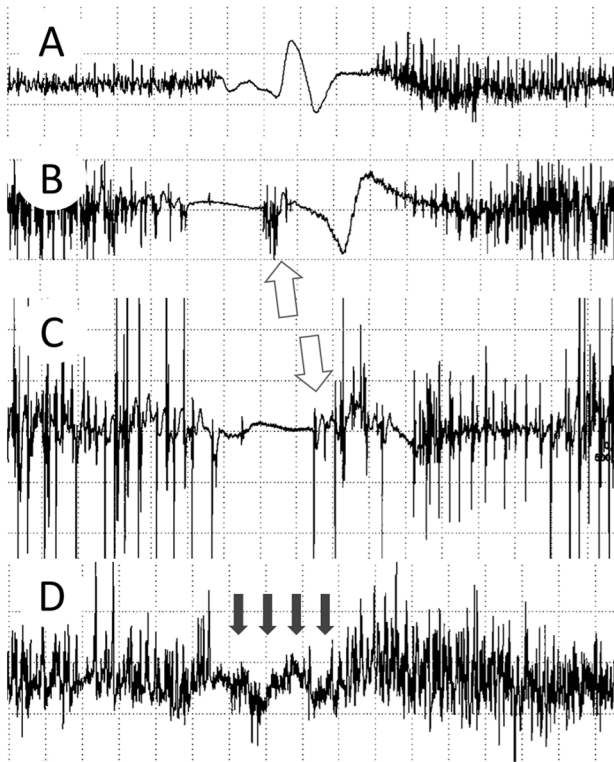
Fig. 6 Example PCA EMG traces during swallowing maneuver: **A** chronic VFP with more constant small size PCA activity during respiration and phonation, complete silence during swallow (including cessation of background noise) and a short intensification after the swallow, **B** and **C** different mid-swallow bursts (white arrows), note the otherwise absence of background noise, **D** throughout the swallow there is small size synkinetic activity (black arrows) in a chronic VFP

nerve anastomoses [26, 27], from the pharyngeal plexus or directly from the adjacent CP. There is no good term yet to differentiate it from the 'normal' synkinetic reinnervation by adductor nerve fibers. The term 'aberrant CP reinnervation' could apply considering the uncertainties of its nature and any possible reinnervation routes.

Conclusions

The PCA EMG approach as described by Mu and Yang or as modified in Vienna is clinically feasible with similar success rates as standard TA EMG. These techniques together with local anesthesia have enabled routine EMG examination of the PCA in neurolaryngology.

Apart from higher vulnerability of PCA innervation and a lesser propensity of PCA to reinnervate compared to TA [10, 11, 21], there are a few more differences between PCA and TA EMG to be aware of. First is the background noise from CP which makes it more difficult to identify pathologic

spontaneous activity or small polyphasic synkinetic activity. The antagonistic maneuver for PCA (phonation), the swallow maneuver as well as knowledge of size and the regular spacing of possible pathologic spontaneous activity potentials are helpful guides towards a correct interpretation of PCA EMG traces. Second, there are larger, but somewhat less dense muscle action potentials during maximum agonistic maneuvers of healthy PCA compared to healthy TA. However, no normative data for semiquantitative PCA or TA EMG evaluation exist yet. Third, there seem to be two patterns of pathologic reinnervation in cases with chronic VFP. One pattern resembles an antagonistic activation. In about half of the cases, there is a component of more constant activity of PCA throughout respiration and phonation with a cessation only during swallowing. This activation pattern does not occur in acute VFP and seems equal to the CP pattern.

Declarations

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