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ORIGINAL ARTICLE

Surgical anatomy and neurophysiology of the vagus nerve (VN) for standardised intraoperative neuromonitoring (IONM) of the inferior laryngeal nerve (ILN) during thyroidectomy

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Abstract
Background Standardisation of the intraoperative neuromonitoring (IONM) technique is a fundamental aspect in monitored thyroid surgery. Vagal nerve (VN) stimulation is essential for problem solving, recognition of any inferior laryngeal nerve (ILN) lesions and prediction of ILN postoperative function. Issues that have been overlooked in the literature, particularly in terms of prospective approaches, are the topographic relationship of the VN with the carotid and jugular vessels as well as the neurophysiology of the VN and ILN that have been studied, with a prospective approach, in patients with various thyroid diseases.
Methods Cooperation with the Human Morphology Department resulted in the completion of a dedicated anatomy report, with the clear objective of providing a detailed anatomic and neurophysiologic description of the VN (n=263).
Results VN identification and stimulation was feasible in all cases and did not result in increased morbidity or operative time. Most VNs lay on the posterior region of the carotid ship (73%), i.e. the *P* position in accordance with our model. Mean amplitudes of EMG signals obtained from VN stimulation were 750±279 μV, lower than those obtained with direct INL stimulation (1,086± 349 μV).
Conclusion A better understanding of the variability in the VN may be useful not only to minimise complications but also to guarantee an accurate IONM.

Keywords Thyroid surgery · Neuromonitoring · Vagus nerve · Recurrent laryngeal nerve · Morbidity

Competing interest This report does not endorse any specific company.

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Introduction

The critical identification of the recurrent laryngeal nerve (RLN) during thyroidectomy has been accepted since the early 1900 as a safe method for achieving less vocal cord morbidity [1]. However, even in cases with no aberrant anatomy of the laryngeal nerve, RLN injury can occur. Additionally, in cases of central lymph nodes' clearance, thyrotoxicosis, retrosternal goitre and thyroiditis, it is often difficult to identify the nerve [2].

For this reason, intraoperative neuromonitoring (IONM) has been proposed and applied as an adjunct to standard visual identification of the RLN to prevent nerve paralysis [3]. Several studies have demonstrated the benefits of IONM in thyroid surgery [4, 5]. Barczyński et al. demonstrated that transient RLN paresis in patients with monitoring is reduced by 2.9% in high risk and 0.9% in low risk patients [4]. Thomusch et al. confirmed that IONM significantly decreases the rate of post-operative transient and permanent RLN palsies [5].

To define the proper procedure for IONM in thyroidectomy, parallel comparisons have been proposed between intraoperative electromyography (EMG) findings, anatomical structures, pre- and post-laryngoscopy and clinical practice to obtain a *standardisation* of IONM technique [6, 7]. Based on these comparisons, the standardised procedure is composed of several important steps to provide a sufficient overview of IONM technology [6, 7]:

1. Vagal nerve (VN) stimulation before thyroid dissection (*V1*)
2. RLN stimulation at initial identification (*R1*)
3. RLN stimulation at the end of thyroid dissection (*R2*)
4. VN stimulation after complete thyroidectomy and haemostasis (*V2*)

It was recently determined that VN stimulation optimises the IONM technology [7]. In monitor solving problems, *V1* is fundamental for verification of all system monitoring. *V2* is essential for recognising any RLN lesions and predicting nerve post-operative function accurately [6, 7].

Issues that have been overlooked in the literature include the surgical anatomy and neurophysiology of the VN and RLN. Herein, we present the normal baseline intraoperative EMG data for the VN and the topographic relationships of the VN with the carotid and jugular vessels, focusing especially on the standardisation of the IONM procedure to achieve the critical view of safety of the VN.

Materials and methods

Patients pre- and post-operative workup

The study was carried out prospectively in 140 consecutive patients who underwent thyroid surgery for various diseases. Exclusion criteria were history of previous neck procedures for any thyroid or non-thyroid disease, minimally invasive video-assisted thyroidectomy, advanced thyroid malignancy, pre-operative RLN palsy or intentional nerve transection. Patients were followed in collaboration with the Division of ENT surgery. Pre- and post-operative follow-up included direct laryngoscopy to check vocal cord mobility performed by an independent laryngologist at 24 to 48 h before surgery and within patient' discharge (usually at second post-operative day). Any reduction in the movement of the cord was recorded as post-operative cord paralysis. For those patients with documented post-operative cord palsy, repeated examinations were performed periodically at 1, 2, 4, 6 and 12 months after the operation until full recovery of vocal cord function was confirmed usually after logopedy. RLN palsy was defined as permanent when there was no evidence of recovery within 12 months of surgery.

The local institutional ethical review board granted for the study protocol. Participants provided informed consent.

Operative technique

Intubation was achieved with a low-dose short-acting neuromuscular blockade to provide adequate muscle relaxation for tracheal intubation (rocuronium 0.1/kg). [6-10] The Nerve Integrity Monitor (NIM-Response 2.0 System, Medtronic, Jacksonville, FL, USA) was used to record the amplitude of the EMG signal for the VN and RLN. Nerves were located, mapped and stimulated in the surgical field by the application of a sterile single-use pulse-generated monopolar stimulator probe (Medtronic, Jacksonville, FL, USA). Stimulation duration was set at 100 μs, current with a frequency of 3 Hz and the event threshold at 100 μV. The EMG signals were defined as *V1*, *R1*, *R2* and *V2* (see above) [7]. The monitor was set with a stimulation artefact suppression of 2.1 ms.

After the neck was fully extended, the correct endotracheal tube electrode position was assessed in a standardised manner by the following defined protocol [6-10]:

1. Pre-operatively
   (a) Monitor check function with impedance values of less than 5 kΩ
   (b) Impedance imbalance of less than 1 kΩ
   (c) Routinely repeat direct visualisation of true vocal cord mucosa—tube surface electrodes perfect well contact by a conventional or digital laryngoscope (Glidescope®) to ensure that the middle of the white marked region (the 30 mm exposed electrodes) was in contact with the true vocal cords (impedance values alone imply only good contact between the electrodes and the patient, so this is not necessary at the level of the vocal cords)
2. Intraoperatively:
   (a) Perform routinely ipsilateral V1 stimulation

In case of absence of initial *V1* signal, we assess some of the following [6-11]:

(a) *Laryngeal twitch assessment.* The post-cricoid region of the larynx during surgery is palpated through the posterior hypopharyngeal wall to sense posterior cricoarytenoid muscle contraction in response to ipsilateral RLN stimulation (i.e. the 'laryngeal twitch response') [11]. The following situations may be considered: (1) Laryngeal twitch is present, neural function is ensured and monitoring dysfunction is present (likely laryngeal electrode displacement: tube displacement, rotation). (2) If laryngeal twitch is not present, surgeon and anaesthesiologist should consider: (a) whether the stimulation current is being delivered (check monitor for current return), (b) whether ground

sternal electrodes are displaced, (c) whether the probe is malfunctioning and a new probe is necessary, (d) if the connections at connector box are solid, (e) whether a neuromuscular blockade has been administered and (f) if neural injury has occurred

(b) *Translaryngeal stimulation* on the midline thyroid cartilage, cricothyroid membrane and cricoids cartilage can be encoded to determine location within the larynx of exposed electrodes of the specific endotracheal tube. Surgeon should appreciate the vocal cords are located approximately 1/2 way down the thyroid cartilage. If maximum shunt stimulation occurs at the level of the cricothyroid membrane, anterior arch of the cricoid or lower, one assumes excessive endotracheal tube depth [6-11]

(c) *Repeat laryngoscopy* to check correct tube positioning

(d) *Contralateral VN stimulation*. If the contralateral vagus does not have a good EMG reading, a recording side (i.e. typically endotracheal tube-related) problem is likely and can be investigated as described above. If the contralateral vagus can be stimulated normally (i.e. the endotracheal tube is in good position), then there is a stimulator error on the ipsilateral side (including possible nerve injury)

In case of loss or reduced *V2* EMG amplitude with respect to the initial *V1* amplitude, we again verify accurately *a*, *b*, *c*, *d*. If nerve injury is suspected, RLN mapping is assessed with IONM to elucidate how and where the nerve was offended.

Technique of VN visualisation and stimulation

We identify the VN in the carotid sheath between the middle and lower pole of the thyroid gland at a very early stage of operation. The VN was identified and stimulated directly by dissecting the carotid sheath from just a 1-cm pouch. According to previous reports, the VN was routinely stimulated in the mid-neck with a current/intensity of *1 mA* [12]. The positive identification of the VN was mapped on the dedicated report sheet. If VN is not initially identify, probe-stimulation amplitude is increased to 2 to 3 mA to expedite VN identification in the carotid sheath. In detail, the location of the VN in relation to the common carotid artery (CCA) and internal jugular vein (IJV) was determined and classified as *A* (anterior), *P* (posterior), *Pj* (posterior to internal jugular vein) and *Pc* (posterior to the common carotid artery) configurations (Fig. 1a–d). The terminology used to designate VN and RLN is that of the Terminologia Anatomica [13]. To monitor any VN stimulation-derived systemic morbidity, electrocardiography, oximetry, end-tidal CO_2 and airway pressure were continuously monitored during surgery.

RLN identification and stimulation

The RLN was located by visualisation and/or placement of a stimulating probe at the tracheoesophageal groove. We tested and analysed the RLN with stimulation electric current/intensity of *1 mA* for definitive confirmation [12]. To optimise the RLN and VN response, we routinely dissected the nerves free from overlying soft tissue, in a strictly bloodless dry field [6-11]. The RLN was dissected meticulously to the entry of the larynx. During further thyroid dissection, the VN and RLN may have been checked again according to the needs of the surgeon. Thus, other VN and RLN amplitudes were obtained during the dissection (defined as *X1*, *X2*, *X3* etc.).

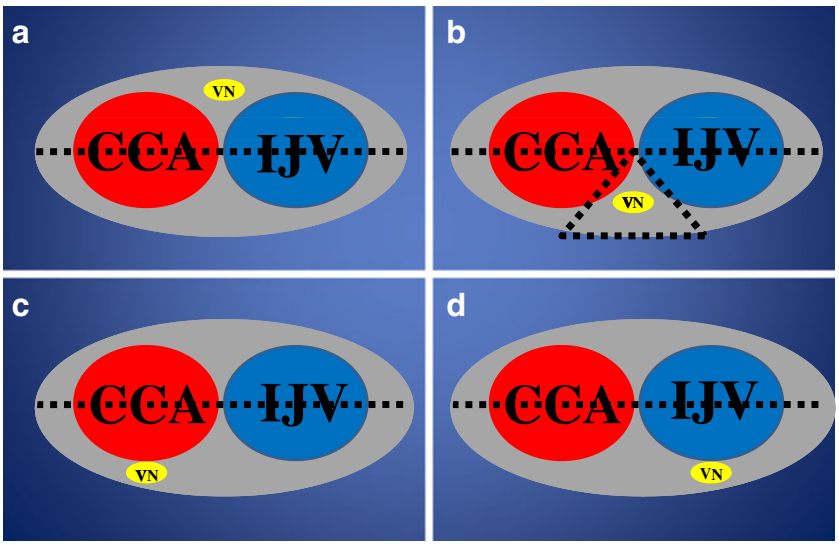

Fig. 1 **a** *A* location of VN; **b** *P* location of VN; **c** *Pc* location; **d** *Pj* location

Statistical analysis

Peak to peak amplitudes of evoked EMG activities were directly read on the IONM device screen with registration of the results. Only biphasic waveform was analysed. In cases of dichotomous variables, group differences were examined by non-parametric tests as needed. Statistical analyses were performed using the SPSS package, release 15.0 for Windows (SPSS Inc, Chicago, IL, USA). Significance was accepted at $p<0.05$. The validity of IONM was defined and calculated according to Chan and Lo [14]. The loss of IONM signal was defined as true positive (TP) when the RLN palsy was confirmed on post-operative laryngoscopic examination and as false positive (FP) when no such palsy was verifiable. An intact IONM signal was interpreted as false negative (FN) when followed by a post-operative laryngoscopic diagnosis of RLN palsy and as true negative (TN) with a normal post-operative laryngoscopic finding. Sensitivity was calculated as TP/(TP+FN), specificity as TN/(FP+TN), the positive predictive value as TP/(TP+FP), the negative predictive value as TN/(FN+TN) and accuracy as TP+TN/total number.

Results

Patients

Complete follow-up was available for all patients. Patients' details are presented in Table 1. Overall, 88% of the surgical procedures were total thyroidectomies (N=123), and 12% were hemithyroidectomies (N=17), providing 263 to be examined for VN and RLN (Table 2). The rate of RLN ramification in this study was 31% (N=81). No permanent palsy occurred. No bilateral vocal cord paralysis occurred. Only four nerves (1.5%) developed temporary palsy, which in all cases resolved within a 4-month range (1–4 months). Most patients (87%) experienced a 2-day hospital stay.

Table 1 Patients data, indications of surgery, type of surgery and selected operative details

Variable	Number	Percent
Age, year (median)	42 (19–87)	
Gender, M/F ratio	1/4	
Disease		
Non-toxic nodular goitre	82	57
Carcinoma	16	12
Graves' disease	23	17
Toxic nodular goitre	19	14
Procedure	140	
Total thyroidectomy	123	88
Lobectomy	17	12
+Central compartment clearance	8	6
Operative details		
Estimated blood loss, ml	51±25	
Operative time, min	96±21.6	
Thyroid volume, ml	62±25	

Table 2 Details of NAR

Variable	Number	Percent
Total no. of nerves at risk	263	
One trunk RLN	181	69
RLN ramification	82	31
Non-RLN	0	0

Relationship of VN with IJV and CCA

Most VNs were found in the posterior region of the carotid sheath (96%). The *P* location of the VN (Fig. 2) was the most common configuration observed on either side (77% right side and 73% left side) followed by the *Pc* (15%) and *Pj* (8%) localisations in accordance with our scheme/model. Only 11 (4%) cases of *A* location were observed overall.

Neurophysiology of the VN and RLN

Before thyroid resection

VN identification and stimulation were feasible in all cases and did not result in any local and/or systemic morbidity. The mean response amplitudes were 750±279 μV for *V1* and 1,086±349 μV for *R1*. Amplitudes were significantly lower for *V1* than for *R1* ($p<0.05$). In fact an improvement of nerve amplitude was observed in 82% of nerves at risk

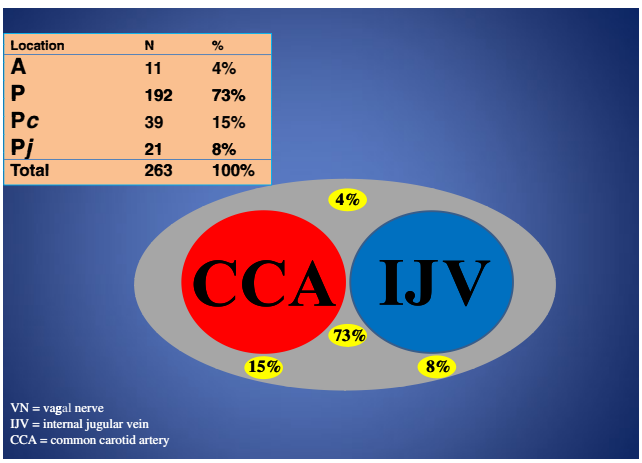

Fig. 2 Location of *VN*

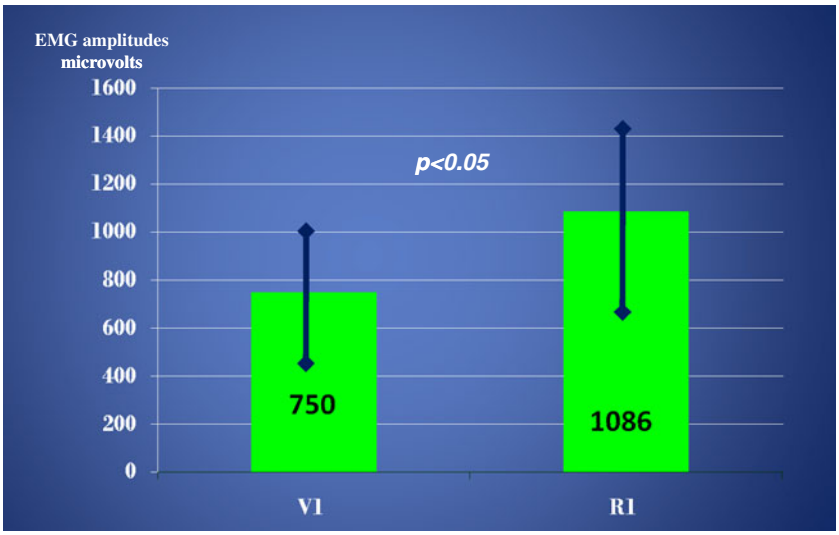

Fig. 3 Mean V1 and R1 response amplitudes (N=263). Overall, an improvement of amplitude from V1 to R1 was observed in 82% of cases

(NAR) from *V1* to *R1* (Fig. 3). Response amplitude values of *V1* or *R1* were not related to gender ratio, age, side, VN distribution, gland volume, treated disease or RLN ramification (p>0.05).

Amplitudes during and after thyroid resection

Overall, the mean response amplitudes of R2 and V2 were 1,201±301 and 1,222±203 μV, respectively. In this study, there were 257 (97.7%) nerves with improved or unchanged V1, R1, R2 and V2 signals, and all cases showed normal vocal cord function post-operatively (true negative). No false negative cases (i.e. RLN palsy diagnosed on post-operative laryngoscopy with intact IONM signal) were noticed.

Four (1.5%) nerves experienced loss of EMG signals with documented RLN palsy (true positive) at laryngoscopy performed 24–48 h post-operatively (Fig. 4). The loss of signal was identified as a total loss of the primary normal biphasic stimulation waveform with amplitude response stimulation reduced to less than 100 μV. The causes of nerve injury were elucidated with the application of neuromonitoring. Using the probe stimulator to map all of the cervical courses of the RLN proximally and distally, we found that the RLN was injured in the last 2 cm of its course in three quarters of these cases and >2 cm from its end in a quarter of cases. In three quarters of nerves, it was difficult to judge the nerve injury with only visualisation. Three traction injuries and one thermal injury were identified, i.e. three type I injuries and one type II injury.

Two remaining cases (0.7%) experienced a reduction in response amplitudes during thyroid dissection (Fig. 5) without a complete loss of signal (as defined above) and conservation of the biphasic waveform (false positive). The response amplitudes decreased in these patients during dissection compared to before excision: One patient experienced as 71% reduction in amplitude (*R2* 806 μV, *X*

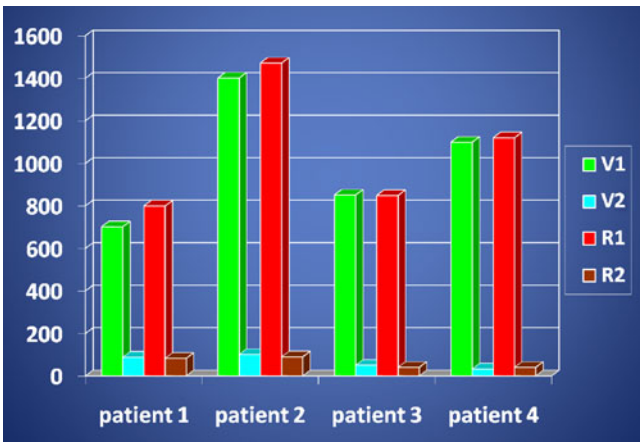

Fig. 4 Response amplitudes in nerve injury (N=4)

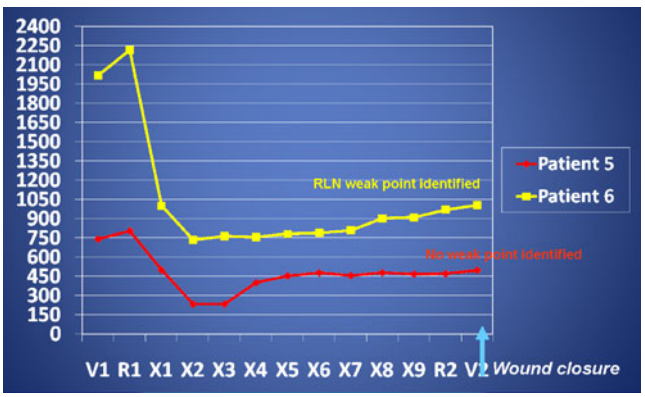

Fig. 5 Amplitudes in patients with reduced-weak signal during thyroidectomy but *no post-operative RLN palsy* (N=2). Patients experienced a conservation of the byphasic waveform without a complete loss of signal and an improvement of signal before wound closure (signal re-entry)

234 μV and *V2* 499 μV) and other patient experienced a 67% reduction in signal (*R2* 2216 μV, *X* 734 μV and *V2* 1005 μV). A weak point in the RLN was found in the last patient. In both patients, a nascent improvement of nerve function with enhancement of amplitudes was observed before wound closure (Fig. 5) in comparison to the lower amplitude obtained during dissection. Post-operative laryngoscopy in these two patients was negative. The calculated accuracy of IONM was 99.2%.

Discussion

VN distribution

Variations in the relationship between the VN, IJV and CCA have never been reported previously to the best of our knowledge. We provide an anatomic basis and demonstrate the reproducibility of our scheme for the identification of the VN within its course in the carotid sheath in the neck. In accordance with our model, the posterior locations (*P*+ *Pj*+*Pc*) of the VN in relation to the CCA and IJV were the most frequent configuration observed (96%; Figs. 1 and 2). The CCA and the IJV together shoulder in the VN, which stays at an angle behind these two vessels encased in a carotid sheath in about 76% of cases (*P* configuration according to our scheme).

The results of our study raise several other important points that could minimise complications while identifying or stimulating the VN. If the surgeon does not initially identify VN, or is not confident with carotid sheath dissection as in the case of reoperative surgery, a large goitre, endoscopic thyroidectomy or a hostile neck, VN identification may be expedited with an increase in amplitude to 2 to 3 mA without initial blind dissection of the carotid sheath. In fact, the cervical segment of sympathetic trunk is just behind the vagal nerve (but outside the carotid sheath): In the case of *P*, *Pj* and *Pc* VN location, the surgeon must pay attention not to injure these sympathetic nerves.

A second observation concerns the *A* location of the VN. The *A* configuration of the VN was rare (4%) in this report. Technically, in the *A* location, the VN may be stimulated only by simply applying the stimulator to the carotid sheath, without dissection (usually in patients with scarce fatty areolar tissue). Moreover, the rare *A* configuration of the VN may explain the rare condition of a non-RLN that arises anterior to the CCA that has been rarely described in the literature.

Finally, further clinical application of our reproducible VN scheme distribution may be useful in other interventional settings, such as direct stimulation of the vagus nerve for real-time monitoring systems, drug-resistant epilepsy therapy, depression, anxiety, Alzheimer disease or diagnostic purposes [15, 16].

Possible limitations of the proposed VN scheme include the fact that the VN was stimulated by dissecting the carotid sheath from only a 1-cm pouch; thus, a less-than-full vision and anatomic description of all the full VN course in the carotid sheath was presented. Other limitations include traction and manipulation of the carotid sheath during surgery, gross lateral metastatic lymph nodes that may displace the VN, tortuosity, previous neck procedures, kinking and coiling of the CCA, carotid sheath stuffed with fatty areolar tissue, rotation of the patient' head and variations of the IJV and CCA position [13].

VN and RLN neurophysiology

In this preliminary report, the overall mean response amplitudes were 750±279 μV for *V1*, 1,086±349 μV for *R1*, 1,201±301 μV and 1,222±203 μV for R2 and V2, respectively. In patients with no complications, the mean baseline s of R1, R2, V2 were higher than those V1. Possible explanation for this includes the fact that intubation was achieved with a short-acting neuromuscular blockade to provide adequate muscle relaxation for tracheal intubation; thus, the different timing of stimulation between V1 and R1, R2 and V2 (about 15–20 min) may result in increased amplitudes for R1, R2, V2 compared to V1. Moreover, the swelling of the larynx and vocal cords due to intubation during operation provides a better contact between vocal cords and the specific EMG tube for monitoring.

During monitored thyroidectomy, we observed three evaluative events:

1. Improved or unchanged V1, R1, R2 and V2 response amplitudes (97.7% of NAR) and all cases showed normal vocal cord function post-operatively.
2. Loss of signal (specifically an amplitude response stimulation of less than 100 μV) plus total loss of the primary normal biphasic stimulation waveform with documented RLN palsy at laryngoscopy performed post-operatively (1.5% of NAR). Thus, EMG signal changes through direct RLN injury.
3. Weak signal (0.7% of NAR), i.e. reduction of response amplitudes during thyroid dissection (without a completely loss of signal) in comparison with initial *V1* or *R1* signal amplitudes values. Interestingly, signal amplitude was diminished about 70% with conservation of the biphasic waveform and an improvement of nerve function with enhancement of amplitudes ('signal re-entrance') before wound closure in comparison to the lower amplitude obtained during thyroid dissection. Post-operative laryngoscopy in these patients was normal.

In all cases of total signal loss and in one case of weaker signal amplitude, stimulation control of the RLN by means of probe stimulator produced a positive EMG response distal to the injury. However, no EMG response was found when the RLN stimulation was performed proximal to the nerve injury.

Additional studies need to define the exact relative quantitative contributions and other possible parameters associated with neural injury (only four patients developed RLN injury with vocal cord palsy). Amplitudes during IONM may vary significantly within patients because of variations in several factors: type of anaesthesia, endotracheal tube position, manipulation on the trachea, exact stimulation probe-nerve contact, RLN preparation (degree of fluid in the surgical field and nerve ensheathed in fascia), type of stimulation probe (mono vs bipolar) and temperature in the environment.

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