Arq Neuropsiquiatr 2004;62(3-A):741-744

LARYNGEAL ELECTROMYOGRAPHY IN MOVEMENT DISORDERS

Preliminary data

Paulo A.T. Kimaid[1], Elizabeth M.A.B. Quagliato[1], Agrício N.Crespo[2], Aline Wolf[2], Maura A. Viana[1], Luiz A.L. Resende[3]

ABSTRACT - This study describes preliminary laryngeal electromyography (LEMG) data and botulinum toxin treatment in patients with dysphonia due to movement disorders. Twenty-five patients who had been clinically selected for botulinum toxin administration were examined, 19 with suspected laryngeal dystonia or spasmodic dysphonia (SD), 5 with vocal tremor, and 1 with Gilles de la Tourette syndrome (GTS). LEMG evaluations were performed before botulinum toxin administration using monopolar electrodes. Electromyography was consistent with dystonia in 14 patients and normal in 5, and differences in frequency suggesting essential tremor in 3 and Parkinson tremors in 2. The different LEMG patterns and significant improvement in our patients from botulinum toxin therapy has led us to perform laryngeal electromyography as a routine in UNICAMP movement disorders ambulatory.

KEY WORDS: laryngeal electromyography, movement disorders, botulinum toxin.

Eletromiografia laríngea e distúrbios do movimento: dados preliminares

RESUMO - Este estudo descreve dados preliminares de eletromiografia laríngea (LEMG) e tratamento com toxina botulínica em pacientes com disfonia associada a distúrbios do movimento. Foram estudados 25 pacientes, 19 com distonia laríngea ou disfonia espasmódica, 5 com tremor vocal e 1 com síndrome de Gilles de la Tourette. LEMG realizada com eletrodos monopolares, antes da administração de toxina botulínica, foi compatível com distonia em 14 pacientes (normal em 5), sugeriu tremor essencial em 3 e Parkinson em 2. Os diferentes padrões de LEMG e melhora considerável obtida com administração de toxina botulínica instituíram LEMG como rotina no ambulatório de distúrbios do movimento da UNICAMP.

PALAVRAS-CHAVE: eletromiografia, laringe, toxina botulínica.

Dysphonia is a speech problem caused by abnormalities in the sound generator, such as laryngeal structures[1]. The production of sound requires fine and precise motor synchronism of the laryngeal muscles; this is often affected in movement disorder patients[1]. Laryngeal electromyography (LEMG) is a useful diagnostic method for motor unit disorders[2-8]; it can be used to establish the cause of laryngeal immobility[9-11] and help guide the needle to the correct location for botulinum toxin administration in laryngeal hyperkinetic movement disorder patients[12-14].

We describe preliminary LEMG pattern data from 25 patients complaining of dysphonia attending UNICAMP movement disorders ambulatory whose clinical examinations and laryngeal endoscopies suggested vocal fold hyperkinetic movement disorders.

METHOD

A total of 25 patients complaining of dysphonia were seen at UNICAMP movement disorders and laryngology ambulatories between January 2000 and January 2003. After consent from the ethical medical committee for human research, all patients were submitted to clinical and endoscopic evaluation, and selected for botulinum toxin treatment due to the diagnosis of hyperkinetic vocal fold movement disorder. Patients with normal LEMG or Parkinson's disease tremors were not treated with botulinum toxin.

LEMG was performed before botulinum toxin administration, with patient in seated position, slightly inclined back support, head supported and slightly extended, arms

Serviços de Neurologia[1]; Otorrinolaringologia[2] Faculdade de Ciências Medicas, Universidade de Estadual de Campinas (UNICAMP) Campinas SP, Brasil; Serviço de Neurologia[3] Faculdade de Medicina do Estado Paulista, Botucatu SP, Brasil.

Received 24 November 2003, received in final form 11 February 2004. Accepted 20 March 2004.

Dr. Paulo Andrade Teixeira Kimaid - Rua Barata Ribeiro 552/61 - 13023-030 Campinas SP - Brasil.

hanging beside the body, and shoulders and cervical musculature relaxed.

Records were made using a four-channel Nihon Kohden Neuropack μ, with 112dB minimum common mode rejection ratio, 0.6 μVrms noise level, and 1000 MΩ input impedance. Filter band-pass was set to 10 - 10000 Hz, sensitivity to 200 μV/cm, and analysis time to 50 ms/cm. After skin asepsia with alcohol, a monopolar electrode was inserted by percutaneous technique for laryngeal muscles approach[13-15]. The *tireoaritenoideus* muscle (TA) was studied in all patients and *cricoaritenoideus posterioris* muscle (CAP) in just 2 with abduction spasmodic dysphonia (SD).

Botulinum toxin, 2.5U of Botox®, diluted in 0.9% saline solution was administered unilaterally in TA muscle of all adduction SD patients, 3 with essential tremor (ET), and 1 with Gilles de le Tourette Syndrome (GTS). For 2 abduction SD patients, botulinum toxin was unilaterally administered in CAP muscle. Botulinum toxin was not administered in 2 patients with Parkinson tremor or the 5 normal LEMG patients. The 0-10 scale of improvement was used for treatment evaluation[16]. Improvement was considered significant between 8-10, mild between 5-7, poor between 2-4, and insignificant or without improvement between 0-1.

RESULTS

Of the 25 patients, 18 were female and 7 male, ages were between 21 and 86 years (mean 51.4). Endoscopy and clinical examination were consistent with SD in 19 patients, vocal tremor in 5, and GTS in 1. LEMG results were consistent with SD in 14 (12 adduction and 2 abduction), normal in 5, vocal tremor in 5 (3 essential tremor and 2 Parkinson), and GTS in 1. Results are shown in Table 1.

All patients with adduction SD showed increased TA muscle rest activity with irregular bursts during muscle contraction. In abduction SD patients, this abnormal muscle activity was found in CAP. Tremor was not observed in SD patients. Essential tremor was diagnosed in 3 patients with normal TA activity at rest and rhythmic bursts of 8 - 11 Hz muscle activity during sustained phonation. Tremor was bilateral in these patients. The 2 patients with Parkinson tremor had rhythmic bursts of 5-6 Hz muscle activity at rest that disappeared with phonation; one patient was unilateral on the right hand side, the other bilateral. The GTS patient had sporadic subtle bursts of involuntary muscle activity independent of voluntary phonation.

Botulinum toxin was administered to 18 patients: 14 with SD, 3 with essential tremor and 1 with GTS. In 14 SD patients treated with botulinum toxin, 9 showed significant improvement, 3 mild, and 2 poor. The 2 patients with poor results had abduction SD. In 3 patients with essential tremor, 1 showed significant improvement, the other 2 mild. In the GTS patient improvement was significant.

DISCUSSION

The most common movement disorders affecting laryngeal muscles were reviewed by Brin et al[1]. They were classified as hypokinetic, hyperkinetic, and mixed according to muscle abnormality[1]. LEMG can help differentiate patterns in abnormal muscle activity and identify the muscles to be treated with botulinum toxin[1,6,12-14,17].

Although tremor is the most common movement disorder, the most frequent type of hyperkinetic laryngeal movement disorder encountered in our service is SD; this is similar to other authors[1,6]. Spasmodic dysphonia patients commonly display normal function at rest with abnormal involuntary adductor (adduction SD) or abductor muscle (abduction SD) co-activation during phonation[1,6,17,18]. LEMG showed increased muscle activity in the apparently normal muscles at rest; during phonation co-activation manifested as bursts of adductor or abductor muscle activity.

In this study, as in literature, the SD group were predominantly of adduction type[1,12-14,17]. Although 25 to 30 % of patients presented tremor[1], no tremor was found in our SD patients. In the past SD was thought to be a psychogenic disorder, but Aronson[18,19] reported no difference between SD patients and normal subjects using psychiatric tests. Interestingly we found 5 patients with mildly elevated rest activity in TA muscles which was interpreted as psychogenic dysphonia. Brin et al.[1] considered psychogenic dystonia as a form of secondary dystonia with dystonic phenomenology and psychiatric etiology. They also considered it a rare condition because patients were referred to a psychiatrist and not a movement disorders service. We suggested speech therapy to our patients before any other type of treatment. In accordance with many authors[1,12-14,17] the majority of our SD patients showed improvement after botulinum toxin administration; results were poor in abduction SD. Brin et al.[14] reported an average of 50% improvement in abduction SD patients.

Tremor is a rhythmic oscillatory movement, such as in the laryngeal sound generator, with relatively stable periodicity[1,20,21]. After Ardran et al.[22], other authors have studied vocal tremor using LEMG[1,19,23]. In ET patients, LEMG generally shows

Table 1. LEMG findings and botulinum toxin treatment in patients with movement disorders.

Case	Sex	Age	Diagnosis before LEMG	LEMG findings	Improvement with Botox^{R} (2,5U)
1	F	58	SD adduction	Increased TA rest activity. Bursts with phonation	8
2	F	57	SD adduction	Increased TA rest activity. Bursts with phonation	9
3	F	38	SD adduction	Increased TA rest activity. Bursts with phonation	7
4	F	57	SD adduction	Increased TA rest activity. Bursts with phonation	8
5	F	55	SD adduction	Increased TA rest activity. Bursts with phonation	8
6	F	38	SD adduction	Increased TA rest activity. Bursts with phonation	8
7	F	48	SD adduction	Increased TA rest activity. Bursts with phonation	10
8	F	73	SD adduction	Increased TA rest activity. Bursts with phonation	5
9	F	67	SD adduction	Increased TA rest activity. Bursts with phonation	7
10	F	59	SD adduction	Increased TA rest activity. Bursts with phonation	8
11	F	38	SD abduction	Increased CAP rest activity. Bursts with phonation	4
12	M	37	SD adduction	Increased TA rest activity. Bursts with phonation	8
13	M	54	SD adduction	Increased TA rest activity. Bursts with phonation	10
14	M	33	SD abduction	Increased CAP rest activity. Bursts with phonation	2
15	F	21	SD adduction	Increased TA rest activity with normal activity during phonation	-
16	F	32	SD adduction	Increased TA rest activity with normal activity during phonation	-
17	M	38	SD adduction	Increased TA rest activity with normal activity during phonation	-
18	M	58	SD adduction	Increased TA rest activity with normal activity during phonation	-
19	M	25	SD adduction	Increased TA rest activity with normal activity during phonation	-
20	M	65	Vocal tremor (P)	Rest: 5-6Hz rhythmic TA activity. Action: Normal	-
21	F	69	Vocal tremor (P)	Rest: 6Hz rhythmic TA activity. Action: Normal	-
22	F	70	Vocal tremor (ET)	Rest: Normal Action: 9-11 Hz rhythmic TA activity.	8
23	F	81	Vocal tremor (ET)	Rest: Normal Action: 8-9 Hz rhythmic TA activity.	4
24	F	86	Vocal tremor (ET)	Rest: Normal Action: 10Hz rhythmic TA activity.	6
25	F	27	VocalTic (GTS)	Subtle bursts of TA activity at rest or during phonation	8

SD, spasmodic dysphonia; TA, tireoaritenoideus muscle; CAP, cricoaritenoideus posterioris muscle; P, Parkinson; ET, essencial tremor; GTS, Gilles de la Tourette syndrome.

normal activity at rest and 4-12 Hz muscle rhythm tremor during sustained phonation[23]. The most common EMG pattern in ET is agonist-antagonist muscle co-activation[24]. In our patients there was a higher frequency of muscle activity but the other characteristics were similar. According to Brin et al.[1,14], treatment with botulinum toxin showed mild to significant improvement in all our patients.

In Parkinson's disease, vocal tremors are seen as a 4-7 Hz rest rhythmic muscle activity[25,26] similar to oral and jaw tremor[27], which can oscillate[25,26], and disappear with phonation[1]. Tremor can be unilateral in Parkinson's disease[1] with the most frequent EMG pattern being alternating agonist-antagonist muscle activation[24]. Our 2 PD patients showed similar characteristics. Botulinum toxin was not administered in our patients at the time of research, because speech therapy was suggested as the first treatment.

Tics are brief, purposeless, stereotyped and repetitive movements of muscle groups[1,28]. GTS, considered the most severe form of tic, may occur with vo-

cal tics[1,28]. No reports of LEMG that evaluated GTS were found. Our only GTS patient presented subtle bursts of TA activity at rest or during phonation.

In all 25 patients motor unit action potentials were of normal amplitude and duration.

REFERENCES

1. Brin MF, Fahn S, Blitzer A, Ramig L, Stewart C. Movement disorders of the larynx. In Blitzer A, Sasaki CT, Fahn S, Brin A, Harris KS (eds). Neurologic disorders of the larynx. New York: Thieme, 1992;248-278.
2. Hirano M, Nozoe I, Shin T, Maeyama T. Electromyographic findings in recurrent laryngeal nerve paralysis: a study of 130 cases. Pract Otol Kyoto 1974;67:231-242.
3. Kotby MN, Haugen LK. Clinical application of electromyography in vocal fold mobility disorders. Acta Otolaryngol 1970;70:428-437.
4. Kotby MN, Fadly E, Madkour O, et al. Electromyography and neurography in neurolaryngology. J Voice 1992;6:159-187.
5. Koufman JA, Postma GN, Whang CS, et al. Diagnostic laryngeal electromyography: the Wake Forest experience 1995-1999. Otolaryngol Head Neck Surg 2001;124:603-606.
6. Lovelace RE, Blitzer A, Ludlow CL. Clinical laryngeal electromyography. In Blitzer A, Sasaki CT, Fahn S, Brin A, Harris KS. (EDS) Neurologic disorders of the larynx. New York: Thieme 1992:66-81.
7. Munin MC, Murry T, Rosen CA. Laryngeal electromyography: diagnosis and prognostic applications. Otolaryngologic Clin N Am 2000; 4:759-771.
8. Yin SS, Qiu WW, Stucker FJ. Major patterns of laryngeal electromyography and their clinical application. Laryngoscope 1997;107:126-136.
9. Gupta SR, Bastian RW. Use of laryngeal electromyography in prediction of recovery after vocal cord paralysis. Muscle Nerve 1993;16:977-978.
10. Parnes SM, Satya-Murti S. Predictive value of laryngeal electromyography in patients with vocal cord paralysis of neurogenic origin. Laryngoscope 1985;95:1323-1326.
11. Sittel C, Stennert E, Thumfart WT, Dapunt U, Ecke HE. Prognostic value of laryngeal electromyography in vocal fold paralysis. Arch Otolaryngol Head Neck Surg 2001;127:155-160.
12. Blitzer A, Lovelace RE, Brin MF, Fahn S, Fink ME. Electromyographic findings in focal laryngeal dystonia (spastic dysphonia). Ann Otol Rhinol Laryngol 1985;94:591-594.
13. Blitzer A, Brin MF, Fahn S, Lovelace RE. Localized injection of botulinum toxin for the treatment of focal laryngeal dystonia (spastic dysphonia). Laryngoscope 1988;98:193-197.
14. Brin MF, Blitzer A, Stewart C, Fahn S. Treatment of spasmodic dysphonia (laryngeal dystonia) with local injections of botulinum toxin: review and technical aspects. In Blitzer A, Sasaki CT, Fahn S, Brin A, Harris KS (EDS). Neurologic disorders of the larynx. New York, Thieme, 1992;214-228.
15. Kotby MN. Percutaneous laryngeal electromyography: standardization of the technique. Folia Phoniatr 1975;27:116-127.
16. Brin MF, Jankovic J, Comella C, Blitzer A, Tsui J, Pulman SL. Treatment of dystonia using botulinum toxin. In Kurlan R (ED). Treatment of movement disorders. Philadelphia Lippincott, 1995;183-226.
17. Blitzer A, Brin MF, Fahn S, Lovelace RE. Clinical and laboratory characteristics of focal laryngeal dystonia: study of 110 cases. Laryngoscope 1988;98:636-640.
18. Aronson AE, Brown JR, Litin EM, Pearson JS. Spastic dysphonia: I. Voice, neurologic, and psychiatric aspects. J Speech Hear Disord 1968;33:203-218.
19. Aronson AE, Brown JR, Litin EM, Pearson JS. Spastic dysphonia II. Comparison with essential (voice) tremor and other neurologic and psychogenic dysphonias. J Speech Hear Disord 1968;33:219-231.
20. Brown JR, Simonson J. Organic voice tremor: a tremor of phonation. Neurology 1963;13:520-525.
21. Deuschl G, Raethjen J, Lindemann M, Krack P. The pathophysiology of tremor. Muscle Nerve 2001;24:716-735.
22. Ardran G, Kinsbourne M, Rushworth G. Dysphonia due to tremor. J Neurol Neurosurg Psychiatry 1966;29:219-223.
23. Koda J, Ludlow CL. An evaluation of laryngeal muscle activation in patients with voice tremor. Otolaryngol Head Neck Surg 1992;107:684-696.
24. Milanov I. Electromyographic differentiation of tremors. Clin Neurophysiol 2001;112:1626-1632.
25. Philippbar SA, Robin DA, Luschesi ES. Limb, jaw and vocal tremor in Parkinson's patients. In Yorkston K, Beukelman D (EDS). Recent advances in clinical dysarthria. San Diego: College Hill Press; 1991:226.
26. Ramig LA, Scherer RC, Titze IR, Ringel SP. Acoustic analysis of voices of patients with neurologic disease: rationale and preliminary data. Ann Otol Rhinol Laryngol 1988;97:164-172.
27. Hunker C, Abbs J. Physiological analysis of Parkinsonian tremors in the oral facial system. In Anonymous. The dysarthrias. San Diego: College Hill Press, 1984:69-100.
28. Jankovic J. Phenomenology and classification of tics. Neurol Clin 1997;15:267-276.