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Triceps Acoustically Evoked Myogenic Potentials in Patients with Spinal Cord Lesions

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Abstract

Objective (Background): Vestibular evoked myogenic potentials (VEMPs) are used to evaluate function of the vestibular system. Sound evoked triceps myogenic potentials (SETMPs) have not been studied extensively and differ anatomically from sternocleidomastoid (SCM) VEMPs because the efferent pathway for SETMPs traverses the spinal cord. The aim of this study was to examine whether SETMPs in people with spinal cord lesions have reduced amplitude or longer latency compared to responses in a group of asymptomatic controls without spinal cord lesions.

Methods: We tested a group of 15 subjects with lesions in the cervical spinal cord and a group of 11 normal (asymptomatic) subjects. All subjects had normal hearing and normal vestibular function as determined by pure tone audiometry and normal SCM VEMPs respectively. All subjects were tested for presence of responses on SCM and SETMPs using methodologies previously established.

Results: Nine of the subjects with spinal cord lesions had bilaterally absent SETMP response and the other five only had a response on one side. The unilateral responses were lower in amplitude than the responses in the control subjects. The control subjects all exhibited a SETMP response.

Conclusions: We found the SETMP response absent bilaterally in 67% and unilaterally in 33% of subjects with cervical cord lesions. This suggests that SETMPs are sensitive to cervical cord injury.

Keywords: Neck pain; Spinal cord lesion; Vestibular evoked myogenic potential; Vestibular system

Introduction

Myogenic potentials induced by sound were first reported in 1964 [1,2] and subsequent work showed them to be predominantly of vestibular origin [3]. It is generally accepted that sound stimulates the saccule, inducing a vestibulo-spinal response and changes in EMG activity. The most widely used auditory vestibular evoked myogenic potential (VEMP) is the sternocleidomastoid (SCM) VEMP. The receptor for VEMPs is believed to be the saccule [4-10]. From there the pathway projects through the inferior division of the vestibular nerve [9,11-15] to the lateral vestibular nucleus [8,9,16, 13,14] to the nucleus of the spinal accessory nerve that in turn innervates the SCM [17]. The resulting VEMP response from the SCM is a large response, occurring at short latency (about 13 msec) with a characteristic multiphasic waveform.

We previously reported that the same acoustic stimulus and general methodology as used for a VEMP in the SCM can elicit a similar response from the tonically activated triceps in control subjects [18]. We call this the sound evoked triceps myogenic potential (SETMP). In brief, this is a robust response of about 90 μv amplitude that occurs at about 35 msec, roughly 12 msec after the SCM VEMP latency. The SETMP is only obtained when triceps activation is relevant to balance [18].

Assuming that the SETMP is also generated by the saccule, the SETMP differs from the SCM VEMP in that it traverses the cervical spinal cord. Therefore, SETMPs are expected to have reduced amplitude or delayed latency as a result of spinal cord lesions. The aim of this study was to examine whether SETMPs in people with spinal cord lesions have reduced amplitude or delayed latency compared to responses in a group of asymptomatic controls without spinal cord lesions

Methods

Subjects: In this study, subjects with spinal cord lesions were compared to controls (asymptomatic subjects without spinal cord lesions). Subjects in the spinal cord lesion group were drawn from patients attending an otoneurology clinic for evaluation and treatment between 2007 and 2010. People suffering from dizziness not attributable to any identifiable pathology and who had well defined cervical spinal cord lesions (as diagnosed by cervical MRI) and a normal SCM VEMP were informed of the study and invited to participate.

Control subjects were recruited by advertisement in the otoneurology clinic and the Department of Physical Therapy and Human Movement Sciences at Northwestern University in 2010. They were included in the study if they had normal hearing as assessed by pure tone audiometry with a value of less than 25 dBHL, a normal SCM VEMP, and no history of cervical spine disease, surgery, or dizziness.

Written informed consent was obtained from all subjects using a consent form approved by the Northwestern University Institutional Review Board.

Testing procedure: All subjects were initially tested for hearing with pure tone audiometry and presence of SCM VEMP. SCM VEMPS were recorded using well established methodology that has been employed in research on SCM VEMPs [12]. If subjects had normal hearing as determined by pure tone audiometry and a normal SCM VEMP response they were included in the study and they were tested for SETMP response.

SETMPs were recorded using the methodology described elsewhere [18]. Surface electromyography (EMG) electrodes were applied to the middle third of the Triceps muscle and just distal to the elbow (over the proximal ulna) bilaterally; the elbow is used as a reference and the forehead as a ground. Subjects stood with the head facing towards the wall and the body leaning forwards, supporting themselves with both arms stretched out forwards and the elbows bent at 160° [18]. The distance between the subject and the wall was adjusted until the pressure transducer measured 10 lbs. of force. Our protocol used 500 consecutive rarefaction tone burst stimuli at 500 Hz and 95 dB nHL presented monaurally via ER3A insert earphones, with the stimulus repeating at 200 ms intervals. The time epoch for each stimulus was 90 msec. Unrectified surface EMG potentials from 500 consecutive stimuli were recorded using the Bio-Logic Navigator Pro evoked potential software (Bio-Logic Systems Corp, Mundelein, Illinois) and were averaged for each trial. The response was amplified 1000-fold and band-pass filtered between 10 Hz and 1500 Hz. Two response waveforms were obtained. Absolute latencies and amplitudes from the first positive and negative waves, p1 and n1, were measured and recorded. From those values we calculated inter-peak latencies and inter-peak amplitudes.

Data Analysis: Descriptive statistics were used characterize both groups of subjects and one way ANOVA was used to compare means between the spinal cord pathology group and the control group to determine similarity between the groups at baseline. One-way ANOVA was used for between group comparisons for SETMP responses (SPSS Version 19, 2010 SPSS Inc).

Results: Fifteen subjects with spinal cord lesions and 11 control subjects were recruited for this study; their characteristics are detailed in Table 1. There was no significant difference between the spinal cord lesion group and the control group in the mean right/left amplitude of the SCM VEMP (p=0.098).

Subject Group	Gender, number male (percent male)	Age, mean (standard deviation) in years	Pure tone average, mean (standard deviation) dBHL	SCM VEMP amplitude, mean (standard deviation) in μV
Spinal cord lesion subjects (n=15)	6 (40%)	56 (11)	19.8 (7.7)	92.25 (77.98)
Normal control subjects (n=11)	4 (36%)	43 (11)	10.1(5.1)	143 (60)

Table 1: Ages, pure tone averages, and SCM VEMP amplitudes in subjects with cervical spinal cord lesions and controls

In the spinal cord lesion group 9 of 14 (67%) subjects had an absent SETMP response bilaterally and 5 (33%) had a unilateral response. A typical response for spinal cord lesion subjects with a normal SCM VEMP and absent SETMP bilaterally is illustrated in Figure 1. All control subjects exhibited a normal SETMP response on each side (elicited by a contralateral monaural acoustic stimulus).

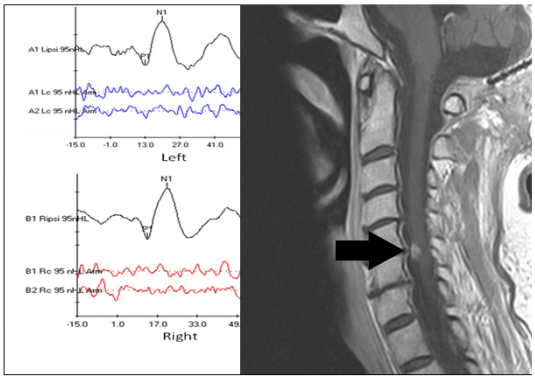

Figure 1: Cervical spine lesion preserves sternocleidomastoid VEMP but eliminates SETMP: typical response of spinal cord lesion subject. In the left panel, the upper tracings in each graph (A) are normal SCM VEMPs, while the lower tracings (B) demonstrate absent SETMPs. These results occurred in a subject with a C5/C6 lesion indicated by the arrow on the sagittal MRI in the right panel.

The characteristics of the spinal cord lesions in the 5 subjects with a unilateral SETMP response were examined for any unifying features (Table 2). In all 5 cases the lesions were extrinsic to the cord. Four subjects had lesions low in the cervical cord (C5/6 or C6/7) and the other had a mass at the cranio-cervical junction. One subject had a traumatic incident prior to the onset of their symptoms. Two subjects had undergone spinal surgery (fusion) that improved their symptoms and might explain the presence of a unilateral SETMP.

Age	Gender	MRI findings	Site of cord lesion	Severity	SETMP response	Mechanism or underlying cause
73	Female	5 x 8 mm mass in right antero-lateral spinal cord C5/6	Intrinsic	Severe	Absent bilaterally	Lung cancer metastatic to brain and spinal cord
58	Female	Severe cord compression at C5/6 due to a large disc herniation, with cord myelomalacia present at C6	Intrinsic and mixed intrinsic	Severe	Absent bilaterally	Cord compression
55	Male	Area of increased signal with right peripheral spinal cord at the level of C2/3 without evidence of enhancement.	Intrinsic		Absent bilaterally	Intrinsic spinal cord lesion

40	Female	Multiple oval areas of abnormal signal intensity throughout the cervical cord compatible with multiple sclerosis. Bulging discs C4/5 and C5/6.	Intrinsic and mixed intrinsic	Severe	Absent bilaterally	Multiple sclerosis plaques
59	Female	Mild cervical degenerative disc disease, minimal protrusion centrally at C3/4 without stenosis.	Extrinsic	Minimal	Absent bilaterally	Whiplash
44	Male	C4/5 small to moderate broad based bulging disc osteophyte complex, small midline focal disc protrusion resulting in indentation and deformity of the cord along the midline, mild spinal stenosis. This patient had had a fusion with flattening of the cord at C5/6.	Extrinsic	Moderate	Absent bilaterally	Motor vehicle accident
79	Female	Severe spinal stenosis at C3/4, C4/5 moderate to severe spinal canal stenosis, and moderate to severe foraminal stenosis, C4/5, C5/6 and C6/7.	Extrinsic	Severe	Absent bilaterally	Fall
48	Female	Minimal to mild central canal stenosis C3/4, C5/6 and C6/7.	Extrinsic	Mild	Absent bilaterally	Dizziness beginning one day following intubation for parathyroid surgery
57	Male	Mild bulging disks indent the ventral thecal sac at C4/5, C5/6, and C6/7.	Extrinsic	Moderate	Absent bilaterally	Right-sided myelitis affecting the C6 and C7 cervical level
42	Male	C5/6 mild central canal stenosis, degenerative changes and posterior osteophyte complex. Initially more severe; had subsequent cervical surgery.	Extrinsic	Severe	Unilateral response	Electrical injury to hand
58	Male	Spinal stenosis at the C6-C7 interface; posterior fusion; cord flattening.	Extrinsic	Moderate	Unilateral response	Onset following syncopal episode
57	Male	Mild central spinal stenosis C6/7 due to right paracentral protrusion and disc osteophyte complex. Chronic cord contouring, compression of right ventral cord.	Extrinsic	Mild	Unilateral response	
69	Female	MRI scan of the neck showed some degenerative changes at C5/6 with some indenting of the cord.	Extrinsic	Mild	Unilateral response	Symptoms began with an upper respiratory tract infection
60	Male	Dorsal left posterolateral intradural extramedullary mass at the craniocervical junction with a differential diagnosis of meningioma versus schwannoma.	Extrinsic	Mild	Unilateral response	Mass at cranio-cervical junction

Table 2: Age, gender, imaging findings, lesion characteristics and etiologies, and presence/absence of SETMP responses in subjects with spinal cord lesions.

The characteristics of the SCM VEMP and SETMP responses for both groups are shown in Table 2. The latencies for both the spinal cord lesion (with SETMP response) and control groups for SETMPs are similar. However, the mean inter-amplitude of the SETMP response in the spinal cord lesion group was significantly less than in the control group for the triceps response contralateral to the ear which received the auditory stimulus (p=0.002, p=0.004, right and left triceps response respectively).

	SCM VEMP (mean response from right and left stimulus)		**SETMPs on the right (contralateral to monaural acoustic stimulus applied to the left ear)**		**SETMPs on the left (contralateral to acoustic stimulus applied to the right ear)**	
	Spinal cord lesion	Spinal cord lesion	Spinal cord lesion	Control	Spinal cord lesion	Control
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
p1 latency (msec)	14.71 (1.54)	14.8 (0.92)	36.58 (2.3), n=3	37.53 (2.2)	34.98 (10.15), n=2	38.1 (1.6)
n1 latency (msec)	22.12 (1.83)	22.54 (1.28)	42.38 (2.0), n=3	44.80 (2.79)	40.34 (11.85), n=2	36.41 (18.2)

p1-n1 inter-latency (msec)	7.40 (1.01)	7.42 (0.88)	5.79 (0.25), n=3	7.27 (3.04)	5.36 (0.35), n=2	5.25 (3.4)
p1-n1 inter-amplitude (μV)	87.97 (81.75)	135.64 (71.46)	12.46 (25.53)	97.04 (102.26)	3.7 (9.96)	61.67 (63.9)

Table 3: SCM VEMPs (ipsilateral to monaural acoustic stimulation), and SETMPs (contralateral to monaural acoustic stimulation). The SCM VEMP is the mean (±SD) of the right and left response ipsilateral to the acoustic stimulus. The SETMP responses are the mean (±SD) of the right and left response contralateral to the ear that received the acoustic stimulus for the spinal cord lesion or control subjects. Since 9 out of 14 spinal cord lesion subjects did not have a SETMP response, the latencies for spinal cord lesion subjects are the mean (±SD) of those with a response, however, the inter-amplitudes are the mean (±SD) of all spinal cord lesion subjects.

Discussion

The results of this study show that SETMP amplitudes were diminished or absent in subjects with cervical spinal cord lesions. As there was no difference in SCM VEMPs between the people with spinal cord lesions and controls it would suggest that all subjects had normal saccule function. Subjects with spinal cord lesions were found to have an absent or diminished SETMP response, and we propose it is likely that the pathway for the response has been compromised in the spinal cord. To our knowledge, no previous studies have investigated the SETMP in subjects with spinal cord lesions. The results for the SETMPs of the normal subjects in our study are consistent with those described previously [18] and show similar values for latency and inter-amplitudes.

SCM VEMPs have been shown to diminish with age [19]. Preliminary examination of the SETMPs of our control subjects suggests that SETMP likely also diminishes with age. It is possible that the difference in age between the groups accounts for the fact the SETMP responses in the 5 spinal cord lesion subjects with a unilateral response were lower than the average SETMP responses in the control subjects; however, age alone is unlikely to account for the absence of SETMPS in 9 spinal cord lesion subjects.

It is not clear why some of the subjects in the spinal cord lesion group had a SETMP response on one side. There are no unifying features among these subjects except that they all had extrinsic cord lesions. Whether the lesion is intrinsic or extrinsic to the cord is unlikely to account for these subjects' having a SETMP response as there were 5 other subjects with extrinsic lesions that did not have a SETMP response.

Possible pathway of SETMPs: In SETMPs the afferent part of the reflex is probably similar to that of the SCM VEMP, but the efferent part of the reflex must differ as the response is bilateral [18] unlike the SCM VEMP response which is ipsilateral to the acoustic stimulus [3].

The projections from the saccule are thought to terminate primarily in the lateral vestibular nucleus [9,10,11,14,15]. Animal studies suggest there are no commissural connections between the lateral vestibular nuclei [20] and that projections from the lateral vestibular nuclei travel primarily through the lateral vestibulospinal tract [16], which does not decussate. However, as data from primates suggest that the saccule also projects to the medial vestibular nucleus [21,22], and projections from that nucleus are bilateral, it may be that in humans the saccular projection to the medial vestibular nucleus is relatively robust, and this may be the pathway that mediates the SETMP. Further research could examine the SETMP response in the presence of various spinal cord pathologies.

Conclusion

In this study the SETMP response was abnormal in all subjects with cervical spinal cord lesions with responses absent bilaterally in 67% and unilaterally in 33%. These data suggest that SETMPs are sensitive to cervical cord injury.

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References

1. Akin FW, Murnane OD (2001) Vestibular evoked myogenic potentials: preliminary report. J Am Acad Audiol 12: 445-452.
2. Akin FW, Murnane OD, Panus PC, Caruthers SK, Wilkinson AE, et al. (2004) The influence of voluntary tonic EMG level on the vestibular-evoked myogenic potential. J Rehabil Res Dev 41: 473-480.
3. Akin FW, Murnane OD, Proffitt TM (2003) The effects of click and tone-burst stimulus parameters on the vestibular evoked myogenic potential (VEMP). J Am Acad Audiol 14: 500-509.
4. Ali AS, Rowen KA, Iles JF (2003) Vestibular actions on back and lower limb muscles during postural tasks in man. J Physiol 546: 615-624.
5. Chen CN, Young YH (2006) Differentiating the cause of acute sensorineural hearing loss between Ménière's disease and sudden deafness. Acta Otolaryngol 126: 25-31.
6. Clarke AH, Schönfeld U, Helling K (2003) Unilateral examination of utricle and saccule function. J Vestib Res 13: 215-225.
7. Colebatch JG (2001) Vestibular evoked potentials. Curr Opin Neurol 14: 21-26.
8. Colebatch JG, Halmagyi GM, Skuse NF (1994) Myogenic potentials generated by a click-evoked vestibulocollic reflex. J Neurol Neurosurg Psychiatry 57: 190-197.
9. Iwasaki S, Takai Y, Ito K, Murofushi T (2005) Abnormal vestibular evoked myogenic potentials in the presence of normal caloric responses. Otol Neurotol 26: 1196-1199.
10. Karino S, Ito K, Ochiai A, Murofushi T (2005) Independent effects of simultaneous inputs from the saccule and lateral semicircular canal. Evaluation using VEMPs. Clin Neurophysiol 116: 1707-1715.
11. Rosengren SM, Colebatch JG (2006) Vestibular evoked potentials (VsEPs) in patients with severe to profound bilateral hearing loss. Clin Neurophysiol 117: 1145-1153.
12. Basta D, Todt I, Eisenschenk A, Ernst A (2005) Vestibular evoked myogenic potentials induced by intraoperative electrical stimulation of the human inferior vestibular nerve. Hear Res 204: 111-114.
13. Halmagyi GM, Aw ST, Karlberg M, Curthoys IS, Todd MJ (2002) Inferior vestibular neuritis. Ann N Y Acad Sci 956: 306-313.
14. Murofushi T, Halmagyi GM, Yavor RA, Colebatch JG (1996) Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis. An indicator of inferior vestibular nerve involvement? Arch Otolaryngol Head Neck Surg 122: 845-848.

15. Tsutsumi T, Tsunoda A, Noguchi Y, Komatsuzaki A (2000) Prediction of the nerves of origin of vestibular schwannomas with vestibular evoked myogenic potentials. Am J Otol 21: 712-715.
16. Didier A, Cazals Y, Aurousseau C (1987) Brainstem connections of the anterior and posterior parts of the saccule in the guinea pig. Acta Otolaryngol 104: 385-391.
17. Akkuzu G, Akkuzu B, Ozluoglu LN (2006) Vestibular evoked myogenic potentials in benign paroxysmal positional vertigo and Meniere's disease. Eur Arch Otorhinolaryngol 263: 510-517.
18. Cherchi M, Bellinaso NP, Card K, Covington A, Krumpe A, et al. (2009) Sound evoked triceps myogenic potentials. Otol Neurotol 30: 545-550.
19. Basta D, Todt I, Ernst A (2005) Normative data for P1/N1-latencies of vestibular evoked myogenic potentials induced by air- or bone-conducted tone bursts. Clin Neurophysiol 116: 2216-2219.
20. Carleton SC, Carpenter MB (1983) Afferent and efferent connections of the medial, inferior and lateral vestibular nuclei in the cat and monkey. Brain Res 278: 29-51.
21. Bickford RG, Jacobson JL, Cody DT (1964) Nature of average evoked potentials to sound and other stimuli in man. Ann N Y Acad Sci 112: 204-223.
22. Britton TC, Day BL, Brown P, Rothwell JC, Thompson PD, et al. (1993) Postural electromyographic responses in the arm and leg following galvanic vestibular stimulation in man. Exp Brain Res 94: 143-151.