ORIGINAL ARTICLE

Vestibular function after spinal cord injury: preliminary results

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Study design: The excitability of the vestibular apparatus and pathways was studied in 10 patients with acute spinal cord injury (ASCI) and in 7 patients with chronic spinal cord injury (CHSCI).

Objectives: Proprioceptive input that may be lost after spinal cord injury (SCI) is needed for integration with vestibular signals to produce proper postural control and perception of movement. This study examined whether there is a change in vestibular excitability after the disruption of the afferent proprioceptive input due to the spinal cord lesion. This study was carried out at Spine Injury Centre and Institute of Rehabilitation, Belgrade, Serbia.

Methods: A total of 10 patients with ASCI and 7 with CHSCI were compared to a group of 50 age-matched healthy subjects. The excitability of the vestibular pathways was studied by means of a modified caloric test in which the duration of postcaloric nystagmus was measured.

Results: ASCI patients with cervical lesions showed significantly reduced response durations ($P < 0.05$). There was no difference in control values for ASCI and for CHSCI patients with thoracic or lumbal lesions. There was no difference in control values for cervical CHSCI patients.

Conclusions: These results suggest that the vestibular response to caloric stimulation is supported by spinally mediated sensory input and that the loss of such input is compensated for over time. Further, these results show that the caloric test may be a useful tool for assessing the degree to which SCI disrupts multisensory integration in the vestibular system and tracking the process of reintegration.

Spinal Cord (2009) 47, 401–404; doi:10.1038/sc.2008.163; published online 27 January 2009

Keywords: vestibular excitability; ASCI patients; CHSCI patients; hypotonic vestibular response; vestibular–proprioceptive interactions

Introduction

The vestibuloocular as well as the vestibulospinal pathways have been extensively studied in animals and humans. However, studies of vestibular function in spinal cord injury (SCI) patients are rare. It is of common knowledge that during head and body movements the vestibuloocular reflex rotates the eyes in opposite direction of head movement to maintain gaze and clear vision.¹ It is also of common knowledge that the vestibulospinal reflexes control erect stance under static and dynamic conditions.² The descending vestibular signals are integrated with ascending proprioceptive signals for proper postural control and self-motion perception.^{3–5} Studies on human and animal show that a broad spectrum of proprioceptive signals originating from cutaneous, joint and muscle receptors reaches all levels of the vestibular pathways.^{6,7} These effects have been studied especially in the lateral vestibular nucleus.^{8,9}

The vestibulospinal reflexes can be tested only in standing position,^{10,11} whereas caloric stimulation of the vestibular apparatus can be performed on patient's bed. After the caloric stimulation of the vestibular apparatus the so-called postcaloric nystagmus occurs, and the duration of this nystagmus is a measure of the excitability of a part of the vestibular apparatus (the lateral semicircular canal). The neural pathways that mediate the vestibuloocular reflex are also involved in postcaloric nystagmus generation. Therefore, we have chosen to apply the caloric test as a measure of vestibular excitability, that is, of the functioning of the vestibuloocular reflex. The aim of this study was to establish whether there is some change in vestibular excitability after disruption of the afferent sensory input caused by SCI.

We have applied the caloric vestibular test in a group of acute spinal cord injury (ASCI) patients, as well as in a group of chronic spinal cord injury (CHSCI) patients.

Materials and methods

Subjects

The American Spinal Injury Association (ASIA) Impairment Scale (AIS) was used to categorize all SCI patients in this

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Received 7 February 2008; revised 19 November 2008; accepted 22 November 2008; published online 27 January 2009

study.¹² Two groups of motor-complete SCI patients, AIS categories A or B, were studied (Table 1). A total of 10 patients, 9 men and 1 woman, ranging in age from 18 to 60 years (mean value 36.5) were examined between 3 and 14 days after injury. They made up the first group, ASCI (Table 1). The causes of SCI were traffic accidents (seven), diving (two) and fall (one patient).

Seven CHSCI patients, all male 17–48 years (mean value 27.2), were examined (Table 1). The causes of SCI were traffic accidents (three patients) and war trauma (four patients). None of ASCI or CHSCI patients complained of vertigo, dizziness, nausea or sensation of image slipping during head movement. None of them had spontaneous or gaze-evoked nystagmus. Hearing was tested with tuning forks (500 and 1000 Hz) and found normal in all patients. Further, ASCI patients were not receiving medications that could be expected to reduce vestibular system excitability.¹³

In addition, 50 control subjects between 18 and 60 years of age (mean value 32.0) with normal hearing and without a history of neurological or vestibular diseases were tested.

Caloric stimulation

All control, ASCI and CHSCI subjects were tested lying in supine position without head elevation. This position was chosen because it is the usual position required for persons with an acute lesion of the cervical spine. Each ear was irrigated with 10 cm³ of water of 18 °C temperature (for 5 s), and with 100 cm³ of water of the same temperature (for 20 s).¹⁴ Nystagmus, which occurred after caloric stimulation, was observed by Frenzel's spectacles, standard magnifying glasses used in clinical practice. The duration of any nystagmus induced by caloric stimulation was measured using a stopwatch.

Data analysis

Control group mean values and standard deviation were calculated for 10 and 100 cm³ stimulation data sets for each

ear. Nystagmus duration obtained from ASCI and CHSCI subjects was then compared to the control values using the Mann–Whitney nonparametric test.

This work was approved by the ethical committee of Clinical Centre of Serbia and informed consent was obtained from all the subjects.

Results

Healthy subjects

In healthy subjects, the mean duration of the vestibular nystagmus after caloric stimulation with 10 cm³ cold water was 45.7 s for the left and 47.1 s for the right ear. The mean duration of the vestibular nystagmus after caloric stimulation with 100 cm³ cold water was 96.1 s for the left and 94.0 s for the right ear (Table 2). A statistically significant difference did not exist between the nystagmus duration after the stimulation of the right and the left ear ($P > 0.05$), neither for 10 nor for 100 cm³ stimulation. Other statistical parameters are shown in Table 2.

Acute spinal cord injury patients

In Table 3 the duration of postcaloric nystagmus is shown in ASCI patients. Note that five of the ASCI patients with

Table 2 Duration of the postcaloric nystagmus (in second) in 50 healthy subjects

Cold water (cm ³) Ear	10		100	
	Left	Right	Left	Right
Duration range (s)	23–95	28–97	55–141	61–144
Mean value	45.7	47.1	96.1	94.0
Standard deviation	± 16.06	± 15.50	± 15.50	± 19.79

Mann–Whitney nonparametric test: cold water 10 cm³ L:R $P > 0.05$; cold water 100 cm³ L:R $P > 0.05$.

Table 3 Duration of the postcaloric nystagmus in ASCI and CHSCI patients

Subject	AIS grade of lesion	10 cm ³		100 cm ³	
		Left	Right	Left	Right
1	C6 AIS-B	18	16	45	40
2	C5 AIS-A	17	16	30	32
3	C7 AIS-B	0	5	15	22
4	C7 AIS-A	0	0	25	48
5	C6 AIS-B	0	10	20	30
6	C6 AIS-B	42	0	68	20
7	L1 AIS-A	52	50	73	76
8	Th10 AIS-B	40	45	100	103
9	Th12 AIS-B	45	48	93	88
10	Th10 AIS-A	38	40	109	105
11	C3 AIS-B	72	70	112	114
12	C3 AIS-A	45	41	90	88
13	C6 AIS-B	49	52	115	120
14	C6 AIS-A	57	61	97	95
15	Th3 AIS-A	43	40	125	127
16	Th5 AIS-A	56	59	89	86
17	Th6 AIS-B	68	70	87	95

Abbreviation: AIS, American Spinal Injury Association (ASIA) Impairment Scale.

Table 1 Characteristics of ASCI and CHSCI patients

No	Sex	Age (years)	Time since injury	Level of motor lesion	AIS grade of lesion
1	M	35	3 days	C6	AIS-B
2	M	52	7 days	C5	AIS-A
3	M	25	4 days	C7	AIS-B
4	M	18	3 days	C7	AIS-A
5	M	21	7 days	C6	AIS-B
6	M	21	4 days	C6	AIS-B
7	F	30	10 days	L1	AIS-A
8	M	60	3 days	Th10	AIS-B
9	M	53	14 days	Th12	AIS-B
10	M	50	6 days	Th10	AIS-A
11	M	48	7 years	C3	AIS-B
12	M	19	6 months	C3	AIS-A
13	M	20	1 year	C6	AIS-B
14	M	17	8 months	C6	AIS-A
15	M	36	8 years	Th3	AIS-A
16	M	19	7 months	Th6	AIS-B
17	M	32	8 years	Th5	AIS-A

Abbreviations: AIS, American Spinal Injury Association (ASIA) Impairment Scale; F, female; M, male.

cervical lesion had a shorter (hypotonic) vestibular response on both sides; one had a shorter vestibular response on one side.

In three acute thoracic spine injured patients and in one acute lumbar injured patient, the duration of the postcaloric nystagmus after the stimulation with 10 and 100 cm³ of cold water was within normal limits. The Mann–Whitney non-parametric test showed no difference between the ASCI patients with thoracic and lumbar lesion and healthy subjects ($P > 0.05$ for all combinations).

Chronic spinal cord injury patients

In Table 3 the duration of the postcaloric nystagmus is shown for four patients with CHSCI of the cervical spine as well as for four patients with CHSCI of thoracic spine. Two with cervical lesion of AIS-A and two with cervical lesion of AIS-B had the duration of the postcaloric nystagmus within normal limits. Similarly, three patients with thoracic spine lesion (two AIS-A and one AIS-B) had the duration of the postcaloric nystagmus within normal limits.

The Mann–Whitney nonparametric test showed no statistical difference ($P > 0.05$ for all combinations) in nystagmus duration between healthy and CHSCI as well as between CHSCI patients with high and low lesion. On contrary, nystagmus duration was statistically different between CHSCI and ASCI patients with cervical lesion ($P < 0.05$) for both tests and for both ears.

Discussion

The question at the beginning of this study was whether there is a change in vestibular excitability after disruption of afferent proprioceptive input in SCI patients. If there is some change, is it present immediately after the injury or develops as the time passes. The results presented here show that in patients with cervical ASCI the duration of nystagmus elicited by caloric stimulation is significantly shorter compared to patients with CHSCI and healthy non-injured subjects. The ASCI patients with cervical lesion had injuries between C2 and C7, they were AIS-A (two) and AIS-B (four). We did not find any correlation between the level of cervical lesion or AIS grade of lesion and the duration of the postcaloric vestibular response.

All patients with acute thoracic or lumbar lesion and all patients with chronic cervical or thoracic lesion had normal durations of the postcaloric nystagmus bilaterally. Our results cannot be easily explained, but possible reasons for this shortening of the vestibular response in ASCI patients can be discussed.

In another pathological condition where the cervical spine is involved, the whiplash injury, patients often suffer unilateral peripheral vestibular damage. However, those patients complain of vertigo, dizziness, slipping of images with head movements and nausea.¹⁵ None of ASCI patients complained of those symptoms. Although examined within 14 days after the injury, none of them had a spontaneous or fixational nystagmus. Therefore, it is unlikely that the observed reduction of nystagmus response in this study is

due to peripheral vestibular damage. The most likely explanation for reduced vestibular excitability in cervical ASCI is disruption of the afferent proprioceptive input after SCI. As mentioned earlier, vestibular information contributes to spatial perception of head and body position in space and mediates reflexes that stabilize posture. The control of posture requires an integration of proprioceptive, visual and vestibular signals.³

Neurophysiological studies in animals show that neck proprioceptive input interacts with the vestibular system on many levels from vestibular sensory epithelium¹⁶ to the vestibular cortex.⁷ Gdowski and McCrea⁹ recorded the eye- and non-eye-movement-related units at the level of the vestibular nuclei of squirrel monkey. During whole-body rotation in the head-free condition, the vestibulocollic reflex diminished the velocity of the head rotation, and the head was moving slower than the body. In this manner the neck proprioceptive input was strongly stimulated. At the same time, the activity of the eye movement units within the vestibular nuclei was better correlated with the head velocity than with the trunk velocity. The activity of the eye movement units was thus influenced by neck proprioceptive input at the level of the vestibular nuclei. This experiment proves the influence of the neck proprioception on vestibular pathways within vestibular nuclei. In human, the connection between neck proprioception and eye movements is proven to exist as a reflex. It is called the cervicocollic reflex. It is usually examined in the head fixed-body passively rotated condition.¹⁷ We do not have applicable information about the influence of proprioceptive input on vestibular pathways after SCI. All the information we have about this influence is from different experimental situations that are not identical to cervical spine lesion.

There is one more reflex we have to consider in human: the vestibul sympathetic reflex. This reflex mediates muscle sympathetic nerve activity, which is likely to be lost after SCI. However this reflex is mediated by the otolith organs¹⁸ that were not examined during this study.

In CHSCI patients with cervical lesion the vestibular response was of normal duration bilaterally. Was it recovery or reorganization that explains the results in CHSCI patients with cervical lesions? Pathological studies demonstrated some anatomical continuity across the lesion in 50% of pre-mortem clinically complete SCI patients.¹⁹ In clinically complete SCI, by means of neurophysiological methods, residual brain influence on spinal cord motor function below the lesion²⁰ as well as retained sensory communication across the lesion²¹ was demonstrated. Two of our CHSCI patients with cervical lesion were AIS-A and two were AIS-B. In AIS-B group some recovery of the proprioceptive input could have happened, but the possibility was not high that in both of AIS-A patients some remnants of proprioceptive fibers were preserved, which contributed to the establishing of normal vestibular response in the after-injury period. Thus, the hypotonic/atonic vestibular response observed in ASCI patients may reflect a lack of whole-body proprioceptive input due to cervical lesion or absence of neck proprioceptive input. The normal vestibular response in CHSCI patients may reflect the consequence of

reorganization of projections to the eye movement pathways after proprioceptive deafferentation. The possibility to reorganize this activity is known within the vestibular system and was mostly studied after unilateral labyrinthectomy.²² It is likely that in cervical CHSCI patients, an integration of the changed proprioceptive, unchanged visual and vestibular signals as well as (probably changed) internal signals resulted in establishment of a normal caloric vestibular response, that is, normal vestibuloocular reflex.

The shortest time after injury period among the CHSCI patients with cervical lesion was 6 months (no. 12). This patient had an AIS-A, C3 lesion. The time needed for normalization of vestibular response cannot be inferred from our results, but it is not longer than 6 months. A longitudinal study is needed to determine whether the caloric vestibular test could be a measure of after-injury sensory reorganization. In addition, the clinical implications of the obtained results warrant further investigations.

Acknowledgements

This work was inspired by professor Milan R Dimitrijević's work with spinal cord injury patients. We thank engineer Ljubomir Damjanovic, biologist Zoran Gacic and neurophysiologist Barry McKay for reviewing the paper as well as for technical help.

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