VESTIBOLOGY

Vestibular and stabilometric findings in whiplash injury and minor head trauma

Reperti vestibolari e stabilometrici nel trauma distorsivo del rachide cervicale e nel trauma cranico minore

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SUMMARY

Vertigo and postural instability following whiplash and/or minor head injuries is very frequent. According to some authors, post-whiplash vertigo cannot be caused by real injury to vestibular structures; other authors maintain that vestibular damage is possible even in the case of isolated whiplash, with vascular or post-traumatic involvement. Furthermore, many of the balance disorders reported after trauma can be justified by post-traumatic modification to the cervical proprioceptive input, with consequent damage to the vestibular spinal reflex. The aim of this study was to evaluate the vestibular condition and postural status in a group of patients (Group A, n = 90) affected with balance disorders following whiplash, and in a second group (Group B, n = 20) with balance disorders after minor head injury associated with whiplash. Both groups were submitted to videonystagmography (VNG) and stabilometric investigation (open eyes – OE, closed eyes - CE, closed eyes with head retroflexed - CER) within 15 days of their injuries and repeated within 10 days after conclusion of cervical physiotherapy treatment. The VNG tests revealed vestibulopathy in 19% of cases in Group A (11% peripheral, 5% central, 3% in an undefined site) and in 60% of subjects in Group B (50% peripheral, 10% central). At the follow-up examination, all cases of non-compensated labyrinth deficit showed signs of compensation, while there were two cases (2%) in Group A and one case (5%) in Group B of PPV. As far as the altered posturographic recordings are concerned, while there was no specific pattern in the two groups, they were clearly pathologic, especially during CER. Both in OE and in CE there was an increase in the surface values and in those pertaining to shifting of the gravity centre on the sagittal plane, which was even more evident during CER. In Group A, the pre-post-physiotherapy comparison of CER results showed that there was a statistically significant improvement in the majority of the parameters after treatment. Moreover, in Group B there was frequent lateral shifting of the centre of gravity that was probably linked with the high percentage of labyrinth deficits. The comparison between the first and second stabilometric examinations was statistically significant only in those parameters referring to gravity centre shifting on the frontal plane, which was probably due to the progressive improvement in the associated vestibulopathy rather than to the physiotherapy treatment performed for the cervical damage. Hence, our study confirms that only in a minority of cases can whiplash cause central or peripheral vestibulopathy, and that this is more probable after minor head injury associated with whiplash. In addition, our data confirm that static stabilometry is fundamental for assessing postural deficits following a cervical proprioceptive disorder. In these cases, in fact, analysis of the different parameters and the indices referring to cervical interference not only permits evaluation of altered postural performance, but also detects and quantifies destabilisation activity within the cervical proprioceptive component.

KEY WORDS: Whiplash injury • Minor head trauma • Vestibular pathology • Stabilometry • Vestibular-spinal reflex

RIASSUNTO

La presenza di vertigine ed instabilità posturale in caso di trauma distorsivo del rachide cervicale e/o trauma cranico minore, è un evento molto frequente. Secondo alcuni autori il danno vestibolare è possibile anche in caso di trauma distorsivo isolato del rachide cervicale, sulla base di fenomeni vascolari. Molti disturbi dell'equilibrio post-traumatici inoltre, possono trovare giustificazione nell'alterazione dell'input propriocettivo cervicale con conseguente danno a carico del riflesso vestibolo-spinale. Lo scopo di questo studio è stato quello di valutare dal punto di vista vestibolare e posturale, un gruppo di pazienti (Gruppo A; n = 90) affetto da disturbi dell'equilibrio post-trauma distorsivo del rachide cervicale ed un secondo gruppo (Gruppo B; n = 20) affetto da trauma cranico minore associato a trauma distorsivo del rachide. In entrambi i gruppi sono state effettuate videonistagmografia e stabilometria statica (occhi aperti - OA, occhi chiusi - OC e occhi chiusi con testa retroflessa – OCR) entro 15 gg dal trauma e ripetute entro 10 gg dal termine del trattamento fisioterapico cervicale. La VNG ha dimostrato la presenza di una vestibolopatia nel 19% dei casi nel Gruppo A (11% periferica, 5% centrale, 3% sede non definibile) e nel 60% dei casi nel Gruppo B (50% periferica, 10% centrale). Al controllo tutti i casi di deficit labirintico non compensato andavano incontro a compenso mentre persistevano ancora 2 casi nel Gruppo A ed 1 caso nel Gruppo B di VPP. I tracciati posturografici, seppur francamente patologici soprattutto in OCR, non presentano un pattern specifico nei due gruppi. Sia in OA sia in OC, si osserva un incremento dei valori di superficie e dei valori relativi allo spostamento del centro di pressione sul piano sagittale, reperti che appaiono ancora più evidenti in OCR. Nel Gruppo A il confronto pre- e post-fisioterapia, mette in evidenza nel test OCR, un miglioramento statisticamente significativo della maggior parte dei parametri dopo trattamento. Nel Gruppo B, si osserva inoltre un frequente spostamento del centro di pressione lateralmente probabilmente legato all'elevata percentuale di deficit labirintici. Il confronto tra il primo ed il secondo esame stabilometrico, risulta statisticamente significativo solo per i parametri relativi allo spostamento del centro di pressione sul piano frontale correlabile probabilmente con la progressiva risoluzione della vestibolopatia associata. Il nostro studio quindi conferma la possibilità che un trauma distorsivo del rachide cervicale possa determinare, in una minoranza dei casi, una vestibolopatia centrale o periferica, vestibolopatia più probabile dopo trauma cranico minore associato a trauma distorsivo del rachide. Inoltre, i nostri dati confermano quanto già descritto in letteratura cioè che la stabilometria statica rappresenta il momento fondamentale per la valutazione di un disturbo posturale conseguente ad una patologia propriocettiva cervicale. In questi casi, lo studio dei diversi parametri e degli indici di interferenza cervicale permettono infatti non solo di valutare l'alterazione della performance posturale ma anche di evidenziare e quantificare l'attività destabilizzante della componente propriocettiva cervicale.

PAROLE CHIAVE: Trauma distorsivo del rachide cervicale • Trauma cranico minore • Vestibolopatia • Stabilometria • Riflesso vestibolospinale

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Introduction

The clinical situation that arises after an episode of whiplash comprises a variety of associated symptoms of different degrees. The mechanical event responsible for the clinical aspects is the flexor-extension of the spine without direct trauma 1 that often occurs in what is commonly known as whiplash². Due to the dynamics of road accidents, this trauma can also be associated with head injuries that are generally without concussion. The symptoms arising from whiplash (whether associated with minor head injury or not) can include headache, cervicalgia, stiffness of the muscles in the cervical area, paraesthesia in the upper limbs ³⁻⁸, neuropsychiatric dysfunction ^{5 9-13} and temporo-mandibular articulation disorders 14-17. In this array of symptoms, those associated with involvement of audio-vestibular structures should not be neglected, some of which are particularly frequent, while others are either rare or even exceptional.

Some patients complain of hypoacousia, a sensation of fullness in their ears and tinnitus 681318-22, while many refer a swaying sensation, postural instability 23-25 and/or distinct objective vertigo 6-8 13 20 26-30. While objective vertigo and postural instability are very frequent in subjects who have suffered a whiplash injury, these symptoms are almost always referred if there has been minor head injury at the same time as the whiplash injury. Some authors maintain that if there are signs of pathological nystagmus associated with asymmetry in the labyrinth function, it might have been present in these patients before the trauma; on the other hand, other authors consider that vestibular damage is possible only after a minor head injury, but that it can be present even in isolated whiplash injury 13 31 32. In these cases, natural vascular events (ischaemic, post-haemorrhagic, etc.) might explain the vestibular damage, whether peripheral or central 20 24, or there could be direct or indirect post-traumatic phenomena in the central nervous system (brain, cerebellar or labyrinth concussion, brain-stem stretching, etc.) 13 30. However, all authors agree that vestibular damage following whiplash injury is possible if the course of the vertebral arteries was already abnormal beforehand 33. Considering the relative reports in the literature and the different interpretations of post-traumatic vestibulopathy that have emerged ¹⁰, it is

fairly obvious that the cause/effect relationship between trauma and vestibular damage is still to be demonstrated. Furthermore, according to some authors, post-traumatic modification in the cervical proprioceptive input might be behind the imbalance frequently complained of, and objectively and clinically recognised after an indirect whiplash injury, even when there is no sign of pathological nystagmus ^{3 7 13 23-25 34-40}. Hence, considering the ongoing debate and the often discordant data reported in the literature, we evaluated the vestibular and postural conditions in two groups of patients with different clinical characteristics: subjects in one group had suffered a whiplash injury, while those in the other had whiplash injury associated with minor head injury. We examined all patients to see whether post-traumatic vestibulopathy was present, and looked for post-traumatic postural modifications by studying stabilometric parameters and, after physiotherapy, we performed complete vestibular and postural examination.

Materials and methods

We studied two groups of subjects: the first (Group A) included 90 subjects (age range: 17-73 years; mean age 40.8 ± 12.6; 59 females and 31 males) suffering from whiplash injury (Grades 1 and 2 of the Whiplash Associated Disorders classification – WAD) ⁸ (Table I); the second group (Group B) comprised 20 subjects (age range: 23-79 years; mean age: 42.8 ± 17.1; 10 females and 10 males) with whiplash injury (Grades 1 and 2 of the WAD) associated with minor head injury (14-15 in the Glasgow Coma Scale – GCS) ⁴¹. All the subjects (n = 110) in the study complained of a balance disorder (postural instability, uncertain gait, vertigo, etc.) that had arisen after trauma.

Within 15 days of the trauma, all the subjects (Group A and Group B) were submitted to the following tests: liminal tone audiometry, impedenzometry, investigation for spontaneous nystagmus (positional and positioning, with or without Frenzel glasses), and the Head Shaking Test (HST). In addition, all subjects underwent a vestibular bithermal caloric balance test according to Fitzgerald-Hallpike and videonystagmographic investigation with the Ulmer-Synapsys® System. In our study, in accordance

Table I. Whiplash-Associated Disorders (WAD) represent a range of injuries to the neck caused by or related to a sudden distortion of the neck associated with extension. The *Quebec Task Force* has divided WAD into four grades ⁸.

Grade 0	Neck pain, stiffness, or any physical signs are noticed
Grade 1	Neck complaints of pain, stiffness or tenderness only, but no physical signs are noted by the examining physician
Grade 2	Neck complaints and the examining physician finds decreased range of motion and point tenderness in the neck
Grade 3	Neck complaints plus neurological signs such as decreased deep tendon reflexes, weakness and sensory deficits
Grade 4	Neck complaints and fracture or dislocation, or injury to the spinal cord

with the literature, vestibular paresis is defined as > 25% asymmetry between left and right-sided responses, and a directional preponderance as > 30% asymmetry between left and right-beating nystagmus ⁴².

In addition to the above tests for the presence of vestibulopathy and damage to the vestibular-oculomotor reflex (VOR), the vestibular-spinal reflex (VSR) in all

patients was investigated by means of static stabilometry using the S.Ve.P. Amplifon System®, recording the posturographic parameters standardised by the system; the parameters considered are listed in Table II. The stabilometric test (performed within 15 days of trauma, like the vestibular test) was carried out with open eyes (OE), closed eyes (CE) and closed eyes with head retroflexed (CER). To evaluate the role of cervical proprioceptive afferences, the cervical interference index corresponding to the surface (SCI) and to the length of the oscillations (LCI) was calculated from the percentage ratio between the S and L values when the eyes were closed and the head retro-flexed and the same parameter when the eyes were closed but the head was erect, considering a value less than 120 as normal 43.

All tests (audio-impedenzometry, vestibular investigation and static stabilometry) were repeated within 10 days after conclusion of physiotherapy treatment (two months after trauma). The subjects manifesting pathologic videonystagmography (VNG) at the follow-up examination were submitted to a further vestibular test with VNG recording 6 months after trauma. In addition, within 3 days of the trauma all patients underwent X-rays of the cervical spine

Table II. Parameters, explanation, graphic representation of the posturographic test and the normal values supplied by the instrument.

Parameter	Explanation		OE (Normal Value)	CE (Normal Value)	CER (Normal Value)
X min, max, mean, SD (mm)	Measurement of centre of gravity shift on the frontal plane (right-left) and the relative standard deviation (SD))	Xmin Xmax Xmean	From -19.1 to 5.9 From -5.4 to 20.2 From -11.9 to 12.5	From -22.3 to 5.9 From -8.1 to 24.6 From -11.3 to 12.7	From -23.8 to 6.2 From -6.2 to 25.4 From -12.6 to 13.9
Y min, max, mean, SD (mm)	Measurement of centre of gravity shift on the sagittal plane (back/forth) and the relative standard deviation (SD)	Ymin Ymax Ymean	From -74.0 to -13.2 From -55.0 to 2.6 From -64.0 to -6.4	From -76.2 to -15.2 From -51.2 to 2.8 From -62.4 to -6.8	From -53.8 to -32.6 From -48.1 to -9.5 From -59.6 to -2.8
S: surface of the ellipse with 90% (mm²)	Surface of the ellipse containing 90% of the sampled points; expresses postural system precision	S	From 0 to 280.0	From 0 to 426.0	From 0 to 560.2
L: total length of the recording (mm)	Length of the connecting subsequent positions of the centre of gravity.	L	From 148.8 to 531.2	From 120.3 to 832.7	From 113.6 to 940.9
V and SD: mean velocity and SD (mm/sec)	Velocity of the shift from the centre of gravity and the relative standard deviation (SD)	SD	From 1.4 to 7.4	From 1.0 to 11.5	From 1.4 to 12.6
LFS: length in function of S	Value expressing the energy spent in relation to the precision of the postural system	-	-	-	-
RI: Romberg Index	Quotient between the previous 6 values measured with eyes closed and the corresponding values with eyes open	-	-	-	-
Stabilogram (mm)	Graphic representation of the shifts from the centre of gravity on the two axes in relation to time	-	-	-	-
Statokinesigram (mm)	Graphic representation of the projection of the postural oscillations on the support polygon.	-	-	-	-
FFT: Fast Fourier Transform (Hz)*	Transformation of the oscillation signal on the two axes $(X-Y)$ in the frequency dominion	-	-	-	-

The FFT demonstrates the spectre of oscillations, where the amplitude is proportional to the degree of energy in that particular frequency. The oscillation in the pressure centre, detected by means of Stabilometry, can be considered an f(t) function that is non-periodic but one that is limited to the t time and which, consequently, can be an analysed by Fourier's integral. The oscillations on the two axes are evaluated separately and the highest frequency found is attributed 100 while the others are expressed in percentages.

in two projections, orthopaedic examination and, thereafter, encephalic NMR, cervical NMR and a neurological examination.

To eliminate any cases of simulation, stabilometry (before treatment and after rehabilitation therapy) was performed twice within the same session in OE, CE and CER (retest) 25. Moreover, to exclude exaggerated or simulated postural behaviour, subjects who had inter-correlation stabilometric recordings that appeared frankly sinusoidal and/or those who had over 2000 mm surface values in OE were excluded from the study (12 subjects) 25 44. None of the subjects in Group A (n = 90) or Group B (n = 20) showed stabilometric recordings of sinusoidal inter-correlation in all three tests (OE, CE, CER) either before or after physiotherapy. Furthermore, no patient had surface values greater than 2000 mm in the OE test. Subjects with a positive anamnesis of otologic and/or neurootologic pathology, such as a previous acute labyrinth deficit, Ménière's disease, previous positional paroxysmal vertigo (PPV), previously diagnosed migraine-associated vertigo, sudden deafness, acute and/or chronic middle ear otitis, otosclerosis, or previous otologic surgery, were excluded from the study, as were subjects with central nervous system disease.

Whiplash physioterapy

The exercise programme was carried out under the supervision of a physiotherapist and comprised specific exercises to improve the movement and control of the neck and shoulder girdles. This list of exercises included: re-education of cranio-cervical flexion movement; training the holding capacity of the neck flexors; retraining eccentric control of the cranio-cervical flexors in upright postures; re-education of cranio-cervical extension movement; training of the holding capacity of the neck extensors; reeducation of the neutral spine in the sitting position, including lumbar-pelvic, thoracic and cervical neutral spine; retraining scapular orientation in neutral posture; training endurance capacity of the scapular stabilizers; retraining dynamic scapular control with arm movement and load, facilitated with rotation, using self-resisted isometric rotation in either supine or in a correct upright sitting posture; head lift in supine position, preceded by cranio-cervical flexion and followed by cervical flexion to lift the head slightly from the supporting surface; graded reduction in pillow height to a flat surface. The exercises were of a low load nature and were designed to be pain-free. Furthermore, each subject was treated by electrical nerve stimulation (TENS, transcutaneous electrical nerve stimulator) for mild chronic musculoskeletal pain.

Statistical analysis

A Student's t-test was used to compare the mean values obtained in the static stabilometric test in OE, CE and CER before and after physiotherapy. A χ^2 test was also carried

out to compare the pre- and post-rehabilitation percentages of the pathological patients in each group in OE, CE and CER, and to compare the percentage of patients in the two groups with positive VNG. Lastly, stepwise regression multivariate analysis was performed – using both backward and forward methods – considering a dependant variable the result of the vestibular examination (current presence or absence of vestibular disease), to demonstrate if (and which) independent variables could be considered predictive of the vestibular examination returning to negative. Statistical analyses were carried out with Stat View 2.0 software.

Results

Videonystagmography

The results of the videonystagmographic examinations in Group A and Group B – in baseline conditions, at the first control and six months after trauma – are reported in Table III. When comparing the percentages of patients in the two groups who were considered pathological in the VNG examination, there was a statistically significant difference at the first examination (Group A 19% vs. Group B 60%; p = 0.0002), at the first follow-up visit two months after the trauma (16% vs. 55%, respectively; p < 0.001) and six months after the trauma (10% vs. 50%; p < 0.01).

Posturographic examination

The percentage of patients who were pathological in the static stabilometric test, both in baseline conditions and at the follow-up visit, are shown in Tables IV and V.

As far as the indices of cervical interference are concerned and which were calculated in both groups at the first stabilometric examination, the following was obtained. In Group A, there was pathological SCI (n.v. < 120) in 59 cases (59/90; 65.6%) and pathological LCI (n.v. < 120) in 44 (44/90; 48.8%); 9 of the 17 subjects with pathological VNG manifested pathological SCI, and 7 pathological LCI. In Group B, we found 11 cases with pathological SCI (11/20; 55%) and 12 with pathological LCI (12/20; 60%); in the 12 cases with pathological VNG, 4 manifested pathological SCI, and 7 showed pathological LCI (Table VI).

With regards to statistical analysis of the posturographic tests, comparison between those obtained before and after physiotherapy rehabilitation of the cervical whiplash gave the following results in Group A:

Comparison between pre- and post-OE: comparing the mean values (mean \pm SD), there was a statistically significant value for Ymax, while only SD VEL was statistically significant when the percentages of pathological patients were compared.

Comparison between pre- and post-CE: comparison of the mean values showed statistically significant difference for SDY, S and FFTY.

Table III. Results of videonystagmographic examination in the two groups studied in baseline conditions, at the first control and six months after the trauma.

		-		-								
		80	Basal			First	First control			Second	Second control	
	VNG neg	Peripheral vestibulopathy	Central vestibulopathy	Indefinable site	VNG neg	Peripheral vestibulopathy	Central vestibulopathy	Indefinable site	VNG neg	Peripheral vestibulopathy	Central vestibulopathy	Indefinable site
Group A	73/90 (81%)	10/90 (11%)	4/90 (5%)	3/90 (3%)	76/90 (84%)	9/90 (10%)	4/90 (5%)	1/90 (1%)	81/90 (90%)	7/90 (%8)	1/90 (1%)	1/90 (1%)
		6/90 (6%) Non- compensated labyrinth deficit 1/90 (2%) Compensated labyrinth deficit 3/90 (3%) PPV	3/90 (3%) Down- beat Ny 1/90 (2%) Up- beat Ny	HSN and BCT within normal range		0/90 (0%) Non- Compensated labyrinth deficit 7/90 (8%) Compensated labyrinth deficit 2/90 (2%) PPV	3/90 (3%) Down- beat Ny 1/90 (2%) Up- beat Ny	HSN and BCT within normal range		0/90 (0%) Non- compensated labyrinth deficit 7/90 (8%) Compensated labyrinth deficit	1/90 (1%) Down-beat Ny	HSN and BCT within normal range
Group B	8/20 (40%)	10/20 (50%)	2/20 (10%)	ı	9/20 (45%)	9/20 (45%)	2/20 (10%)	1	10/20 (50%)	9/20 (45%)	1/20 (5%)	1
		4/20 (20%) Non- compensated labyrinth deficit 4/20 (20%) Compensated labyrinth deficit 2/20 (10%) PPV	1/20 (5%) Down-beat Ny 1/20 (5%) Up- Beat Ny			0/20 (0%) Non- compensated labyrinth deficit 8/20 (40%) Compensated labyrinth deficit 1/20 (5%) PPV	1/20 (5%) Down-beat Ny 1/20 (5%) Up- beat Ny			0/20 (0%) Non- compensated labyrinth deficit 8/20 (40%) Compensated labyrinth deficit	1/20 (5%) Down-beat Ny	

BCT: Bithermal Caloric Test (vestibular caloric balance test according to Fitzgerald- Hallpike); Non-compensated labyrinth deficit: Labyrinthine preponderance > 25% and Directional Preponderance (\$ 30%) 85 85, Down- and Up-beat Ny: observable with and without fixation persistent and purely vertical 42.

Table IV. Percentage of patients with pathologic static stabilometry in the three tests performed within 15 days of the trauma (baseline) and after rehabilitation therapy (control).

		Baseline		Control		
	0E	CE	CER	0E	CE	CER
Group A	73.3%	82.2%	92.2%	60%	78.9%	83.3%
Group B	95%	85%	95%	70%	85%	90%

Comparison between pre- and post-CER: comparison of the mean values yielded statistically significant results for Xmin, SDX, Ymin, SDY, L, S, VEL and SD VEL. Moreover, comparison of the percentage of pathological patients showed a statistically significant difference for Xmin, Ymax, L, S, and SD VEL.

Comparison between pre- and post-rehabilitation therapy in subjects with minor head injuries associated with indirect trauma in the cervical spine (Group B) gave the following results:

Comparison between pre- and post-OE: comparison of mean values showed a statistically significant value for FFTX.

Comparison between pre- and post-CE: when the means

were compared, there was a statistically significant difference for SDY.

Comparison between pre- and post-CER: the only significant parameter was Xmin when comparing the percentage of pathological patients before and after physiotherapy. Multivariate analysis of the data gave the following results. For Group A, we used the result of the vestibular examination as the dependent variable (vestibular pathology present/vestibular examination negative) and Xmed, SDX, Ymed, SDY, L, S, VEL and SD VEL as independent variables in entire series of tests (OE, CE, CER). When multivariate analysis was performed in backward mode, it demonstrated that all the above-mentioned independent variables could be jointly considered predictive of return to negative by

Table Va. Means \pm SD of the stabilometric parameters (and the relative percentage of pathological patients for whom the instrument supplies standardised values). The Table also shows the statistically significant differences resulting from comparison of the pre- and post-rehabilitation data in Group A and Group B separately.

				Group A				
	Xmin	Xmax	Xmed	SDX	Ymin	Ymax	Ymed	SDY
Pre-OE	22/90 (24.44%) -13.72 ± 8.07	14/90 (15.56%) 7.16 ± 9.70	11/90 (12.22%) -3.22 ± 7.49	0.41 ± 0.17	16/90 (17.78%) -57.19 ± 18.62	9/90 (10%) -33.16 ± 18.16*	17/90 (18.89%) -45.70 ± 17.73	0.50 ± 0.24
Post-0E	16/90 (17.78%) -13.05 ± 8.34	8/90 (8.89%) 7.01 ± 7.60	6/90 (6.67%) -2.92 ± 5.59	0.40 ± 0.20	18/90 (20%) -61.39 ± 16.72	14/90 (15.56%) -38.81 ± 16.65*	13/90 (14.44%) -50.42 ± 15.21	0.45 ± 0.21
Pre-CE	30/90 (33.33%) -19.42 ± 11.48	11/90 (12.22%) 13.53 ± 12.23	15/90 (16.67%) -3.07 ± 8.96	0.64 ± 0.29	28/90 (31.11%) -65.61 ± 21.77	16/90 (17.78%) -27.95 ± 20.45	19/90 (21.11%) -47.30 ± 16.44	0.77 ± 0.38 §
Post-CE	25/90 (27.78%) -16.76 ± 12.64	13/90 (14.44%) 11.95 ± 11.46	10/90 (11.11%) -2.85 ± 7.39	0.57 ± 0.29	23/90 (25.56%) -64.33 ± 17.34	9/90 (10%) -32.31 ± 14.18	18/90 (20%) -47.08 ± 16.95	0.64 ± 0.24 §
Pre-CER	35/90 (38.89%)§ -22.89 ± 13.08§	19/90 (21.11%) 17.69 ± 15.59	11/90 (12.22%) -2.20 ± 8.45	0.76 ± 0.35 §	73/90 (81.11%) -71.49 ± 20.73*	34/90 (37.78%) [§] -23.23 ± 21.74	26/90 (28.89%) -46.55 ± 18.01	0.92 ± 0.38 §
Post-CER	19/90 (21.11%) [§] -17.93 ± 10.13 [§]	12/90 (13.33%) 13.88 ± 11.24	6/90 (6.67%) -2.12 ± 7.18	0.60 ± 0.28 §	64/90 (71.11%) -64.90 ± 18.68*	17/90 (18.89%)§ -27.15 ± 16	20/90 (22.22%) -46.36 ± 15.91	0.75 ± 0.33 §
				Group B				
Pre-OE	8/20 (40%) -15.18 ± 9.60	8/20 (40%) 4.13 ± 11.67	5/20 (25%) -5.14 ± 9.24	0.39 ± 0.17	8/20 (40%) -62.63 ± 24.85	6/20 (30%) -37.82 ± 22.58	7/20 (35%) -44.33 ± 25.60	0.56 ± 0.25
Post-0E	3/20 (15%) -14.89 ± 5.85	3/20 (15%) 3.12 ± 8.06	3/20 (15%) -6.04 ± 6.95	0.34 ± 0.11	5/20 (25%) -60.78 ± 22.24	3/20 (15%) -39.06 ± 21.38	5/20 (25%) -47.35 ± 22.88	0.43 ± 0.20
Pre-CE	9/20 (45%) -20.77 ± 10.63	7/20 (35%) 13.64 ± 16.18	7/20 (35%) -4.01 ± 8.72	0.65 ± 0.30	9/20 (45%) -66.64 ± 26.48	6/20 (30%) -27.47 ± 24.04	7/20 (35%) -45.48 ± 23.65	$0.74 \pm 0.35^{*}$
Post-CE	6/20 (30%) -22.44 ± 11.19	2/20 (10%) 6.53 ± 11.16	7/20 (35%) -6.40 ± 9.14	0.52 ± 0.26	6/20 (30%) -64.55 ± 25.26	5/20 (25%) -34.59 ± 21.04	5/20 (25%) -48.58 ± 22.08	$0.54 \pm 0.25^{*}$
Pre-CER	12/20 (60%) [§] -23.31 ± 12.79	8/20 (40%) 18.36 ± 17.48	4/20 (20%) -1.01 ± 10.83	0.74 ± 0.23	17/20 (85%) -67.52 ± 28.19	10/20 (50%) -23.60 ± 28.07	6/20 (30%) -46.41 ± 27.12	0.95 ± 0.44
Post-CER	4/20 (20%)§ -21.65 ± 10.20	6/20 (30%) 15.25 ± 11.86	2/20 (10%) -3.43 ± 7.75	0.67 ± 0.26	14/20 (70%) -69.90 ± 21.03	7/20 (35%) -28.81 ± 23.48	5/20 (25%) -49.13 ± 21.20	0.85 ± 0.40

[§] p < 0.01; # p = 0.01; * p = 0.03.

Table Vb. Means \pm SD of the stabilometric parameters (and the relative percentage of pathological patients for whom the instrument supplies standardised values). The Table also shows the statistically significant differences resulting from comparison of the pre- and post- rehabilitation data in Group A and Group B separately.

			Group A			
	L	S	FFTX	FFTY	VEL	SD VEL
Pre-OE	7/90 (7.78%) 308.56 ± 113.16	39/90 (43.33%) 325.82 ± 255.76	0.13 ± 0.13	0.08 ± 0.06	12.18 ± 4.44	51/90 (56.67%) [*] 8.52 ± 4.16
Post-0E	9/90 (10%) 303.81 ± 134.13	31/90 (34.44%) 286.18 ± 234.34	0.12 ± 0.11	0.09 ± 0.07	11.91 ± 5.28	36/90 (40%) [*] 8.34 ± 4.81
Pre-CE	12/90 (13.33%) 557.10 ± 226.60	58/90 (64.44%) 814.65 ± 803.03#	0.16 ± 0.16	$0.11 \pm 0.10^{\circ}$	21.93 ± 8.92	57/90 (63.33%) 15.29 ± 6.98
Post-CE	9/90 (10%) 508.64 ± 228.22	45/90 (50%) 580.66 ± 474.57#	0.17 ± 0.13	$0.15 \pm 0.14^{*}$	20.02 ± 9	51/90 (56.67%) 13.75 ± 6.77
Pre-CER	20/90 (22.22%) [#] 714.46 ± 322.47 [#]	65/90 (72.22%)§ 1148.57 ± 998.63§	0.14 ± 0.12	0.14 ± 0.11	28.13 ± 12.70#	71/90 (78.89%) [#] 21.21 ± 12.41 [§]
Post-CER	9/90 (10%)# 599.87 ± 294.10#	42/90 (46.67%)§ 740.89 ± 628.71§	0.13 ± 0.10	0.13 ± 0.15	23.45 ± 11.69#	56/90 (62.22%) [#] 16.10 ± 8.18 [§]
			Group B			
Pre-OE	2/20 (10%) 306.57 ± 127	9/20 (45%) 323.20 ± 253.25	0.17 ± 0.11§	0.09 ± 0.08	13.68 ± 7.66	11/20 (55%) 9.10 ± 5.39
Post-0E	1/20 (5%) 277.15 ± 94.07	5/20 (25%) 237.87 ± 134.35	$0.10 \pm 0.05^{\S}$	0.08 ± 0.05	10.94 ± 3.76	8/20 (40%) 7.16 ± 2.76
Pre-CE	2/20 (10%) 542.06 ± 246.54	16/20 (80%) 883.46 ± 632.07	0.16 ± 0.12	0.15 ± 0.14	22.90 ± 10.15	14/20 (70%) 15.73 ± 8.05
Post-CE	0/20 (0%) 454.64 ± 188.33	11/20 (55%) 579.32 ± 326.85	0.11 ± 0.07	0.12 ± 0.09	18.32 ± 7.09	10/20 (50%) 12.07 ± 5.51
Pre-CER	2/20 (10%) 646.88 ± 248.72	16/20 (80%) 1101.87 ± 613.88	0.12 ± 0.07	0.12 ± 0.13	26.35 ± 9.23	16/20 (80%) 19.43 ± 8.61
Post-CER	1/20 (5%) 623.64 ± 228.62	16/20 (80%) 987.49 ± 531.23	0.11 ± 0.08	0.18 ± 0.29	24.81 ± 9.09	13/20 (65%) 17.14 ± 6.90

[§] p < 0.01; #p = 0.01; *p = 0.03.

Table VI. Patients with pathological/normal indices of cervical interference in all subjects of Group A (n = 90) and Group B (n = 20) and in subjects with pathological VNG in Group A (n = 17) and Group B (n = 12).

	Pathological SCI (≥ 120)	Normal SCI (< 120)	Pathological LCI (≥ 120)	Normal LCI (< 120)
Group A (total patients) (n = 90)	59/90 (65.6%)	31/90 (34.4%)	44/90 (48.8%)	46/90 (51.2%)
Group A (with pathological VNG) ($n = 17$)	9/17 (52.9%)	8/17 (47.1%)	7/17 (41.2%)	10/17 (58.8%)
Group B (total patients) (n = 20)	11/20 (55%)	9/20 (45%)	12/20 (60%)	8/20 (40%)
Group B (with pathological VNG) ($n = 12$)	4/12 (33.3%)	8/12 (66.7%)	7/12 (58.3%)	5/12 (41.7%)

vestibular examination (p < 0.001). Furthermore, statistical analysis demonstrated that if Xmed and VEL were excluded from the independent variables, the significance of the test does not change (p < 0.001). Next, when multivariate analysis was performed in *forward* mode, the two parameters that were similarly predictive of the vestibular test returning to negative were SDX and Ymed (p < 0.01). For multivariate analysis in Group B, considering the three tests together, we again used the presence/absence of modifications in the vestibular investigation as the dependent variable, and Xmed, Ymed, L and VEL as independent variables (the other independent variables were not accepted by the

software used). The *backward* mode demonstrated that all four of the independent variables stated above were predictive of resolution of the vestibular situation (p = 0.002). Because of the limited number of patients and due to the results of the individual parameters, the software was unable to calculate the significance of the parameters in the *forward* mode.

Other tests

Brain NMR was negative for focal lesions in all subjects and neurological examination was also negative, thus confirming a diagnosis of minor head injury (grades 14 and 15 of the GCS). Orthopaedic examinations confirmed the status of cervical whiplash (grades 1 and 2 in the WAD classification), while they excluded more serious musculoskeletal pathologies (previous fractures, scoliosis, etc.) in all subjects. X-rays of the cervical spine were negative for signs of fracture and demonstrated a physiological cervical lordosis deficiency in 73 cases in Group A (73/90; 81.1%) and in 15 of those in Group B (15/20; 75%). Cervical NMR confirmed the absence of discal hernias. Audio-impedenzometric tests in all subjects were compatible with normal hearing or bilateral and symmetric presbyacousia, and none had asymmetric neurosensorial hypoacousia. There were no signs of gaps in transmission or tubaric dysfunction.

Discussion

Vertigo and postural instability are very frequently present among the symptoms of cervical whiplash injuries 6 18 19 22. Furthermore, dizziness and vertigo is even more frequently referred by subjects who suffered minor head injuries as well as cervical whiplash 40. Not all authors agree that cervical whiplash and neuro-otological injury are related from a physiopathological point of view. In fact, some authors maintain that when central or peripheral vestibular damage is diagnosed after an episode of whiplash it can be attributed to pre-existing condition 33 or to misinterpretation of the neuro-otological examination ¹⁰. In reality, there are certain exceptional cases with cochlear involvement (sensorineural hypoacousia and tinnitus) following cervical whiplash injuries that confirm this interpretation. The only neuro-otological evidence that has been maintained to be directly attributable to cervical whiplash and/ or minor head injury is the onset of positional paroxysmal vertigo (PPV) due to otoconia detachment, where the cause/effect of the trauma appears evident 45-49. Patients frequently refer neuro-otological disorders, and occasionally the pathological nystagmus and vestibular damage detected can find explanation only in some traumatic aetiology (young subjects with negative general and neurootological anamnesis, with no history of trauma and negative on neuro-radiological examination). In addition, it has been pointed out recently that the permanence of symptoms and oto-vestibular objectivity in cases of whiplash is not directly correlated to the severity of the trauma 31 32. Certain authors link neuro-otological damage following whiplash injury with pre-existing anatomical anomalies in the vertebral arteries 33. MacNab (2002) attributes tinnitus, hypoacousia and pathological nystgamus following an indirect injury in the cervical spine to a spasm in vertebral arteries ²⁰, while other studies report anecdotal cases of cerebral infarction revealed with NMR in the areas fed by branches of the vertebral-basilar arteries after whiplash injury ⁵⁰. Nevertheless, the majority of neuro-otological injuries seen after an episode of whiplash are probably

attributable to ischaemic or haemorrhagic events in the labyrinth membrane (peripheral vestibulopathy, tinnitus, sensorineural hypoacousia) or to concussion and/or brainstem stretching (central vestibulopathy, tinnitus) 13 30. In fact, violent distortion of the cervical spine can cause concussion-like damage in the labyrinth because the abrupt acceleration-deceleration that occurs when there is flexion-hyperextension of the spine can produce mechanical shaking of the internal ear. Furthermore, other reports in the literature maintain that labyrinth damage in cases of whiplash injury can be caused by vascular mechanisms as well, due to the sudden compression in the vertebral artery brought on by the violent trauma and the consequent abrupt reduction in blood flow in the internal ear ²⁴. Naturally, damage in the vestibular structures is more likely (from a physiopathological point of view) in the case of minor head injury, since ischaemic or haemorrhagic events in the labyrinth membrane, as well as concussion and/or brain-stem stretching, can be caused directly by the trauma $^{13\ 30\ 51}$.

Our data confirm that vestibular damage can be caused by cervical whiplash and that it is more likely when the whiplash is associated with minor head injury (19% vs. 60%; p = 0.0002). Analysis of videonystagmographic investigations performed in both of our groups within 15 days of the trauma shows that it is possible to find both peripheral (non-compensated/compensated labyrinth deficit and PPV) and central (vertical nystagmus) vestibulopathy. It is important to note that in the analysis of the vestibular data referring to the first control examination, the non-compensated peripheral deficits detected in the basal VNGs all tended towards compensation, both in the cases of cervical whiplash and in those with minor head injuries. Lastly, considering the VNG examinations, the data collected in our study confirm reports in the literature; indeed, PPV is possible in cervical whiplash trauma as well, even though it is more frequent in cases of minor head injury (3% vs. 10%) and is more difficult to resolve compared to the idiopathic forms. In fact, in spite of the freeing manoeuvres performed on all patients during the first VNG examinations, there were still two cases (2/90; 2.2%) of paroxysmal vertigo among those with isolated whiplash and one case (1/20; 5%) of PPV among those with whiplash associated with minor head injury. In our study, all cases of PPV (Group A and Group B) involved the posterior semicircular canal without vestibular paresis. Semont manoeuvres were performed and no transformations in PPV of the horizontal semicircular canal occurred.

In our opinion, differences in the vestibular examination from the first, to the second and to the third videonystagmographic investigation (effective compensation of the peripheral deficits, HST returning to negative, complete resolution of certain cases of PPV, disappearance of many episodes of vertical nystagmus), and the fact that all the subjects were negative by brain NMRs with contrast medium, confirm the causal relationship between trauma (whiplash injury and/or minor head injury) and vestibular findings. Moreover, the relatively young mean age of the subjects with pathological vestibular findings in the first VNG (Group A: 40.2 ± 7.9 SD years; Group B: 41.7 ± 19.4 SD years) and the negative anamnesis for cardiovascular disease and recent infections in all subjects, allows us to exclude – with fair approximation – other pathogenic factors which might (in theory) cause the vestibular aspects found after trauma (vestibulopathy of a viral nature, microvascular vestibulopathy, etc.)

Armato and Ferri (2002) published a study on 569 patients with whiplash, with or without associated head injuries ⁴⁰. Of these, 21% had either normal vestibular functioning or only static stabilometric alterations that were compatible with a modified proprioception of the cervical spine according to the criteria proposed by Brandt (1981), Norré (1987) and Guidetti (1988, 1996) ³⁴⁻³⁶⁻³⁹; clinical and instrumental neuro-otological examinations were normal with no vestibular deficits or signs of central oculomotor involvement. The remaining 79% of the subjects manifested some alteration in the vestibular system and, in over two-thirds of these, posturographic investigation demonstrated modifications in the cervical proprioceptive system.

Cervical whiplash injury can also be responsible for important balance disorders, even without causing direct damage to vestibular structures. In fact, since a whiplash injury implies stretching of the muscles and intense irritation of the complex cervical proprioceptive system, the outcome is altered function both of the neuromuscular spindles in the cervical area and of the osteo-articulartendon sensors in the entire spine. Cervical musculature, which is very rich in spindles, is one of the structures that keeps the head erect; in particular, the nape muscles contain a large quantity of spindles, equalled only by the extrinsic ocular muscles, vocal chords and the interosseous muscles in the hand – all structures that require particularly intricate motor regulation ^{52 53}. The correlation between cervical proprioceptive afferences and the vestibular system is now well known. Among other effects, the nape proprioceptive afferences activate neurons in the dorsal-caudal part of Dieters ipsilateral vestibular nucleus, both directly and indirectly, and interfere in the activity of group X, the inferior olive and the vermian cortex of the anterior cerebellar lobe 54, interacting strongly on posture control. The vestibular system controls cervical muscles through vestibular-spinal pathways, in particular through the medial ones, which prevalently project contralaterally. Hence, it appears evident that a cervical whiplash injury can cause postural disorders that do not depend on the presence of pathological nystagmus that can worsen the postural disorders of subjects with associated vestibulopathy. In effect, cervical proprioceptive afferences can be a valid alternative to the vestibular reflexes at low stimulation frequencies and can be fundamental in compensation mechanisms involved in peripheral vestibular damage 55-⁵⁹. Our data confirm what has been already described in the literature 60-64; namely that static stabilometry is a fundamental tool for assessing postural disorders following cervical proprioceptive disorders and whiplash injury in particular. In these cases, analysis of various parameters and indices of cervical interference (SCI and LCI) can supply not only an evaluation of the altered postural performance, but can also reveal and quantify the destabilising activity of the cervical proprioceptive component 60. In fact, static stabilometry performed on subjects in Group A before physiotherapy (subjects with cervical whiplash) gave pathological results, especially when the head was retroflexed; after therapy, in spite of the fact that stabilometry tended towards normal in many subjects, performance was nevertheless poorer when the head was bent back during the test compared to when it was erect. Stabilometry performed before therapy in Group B (subjects with cervical whiplash injury associated with minor head injury) gave highly pathological results even in the eyesopen and eyes-closed conditions, which demonstrates that retroflexion of the head within 15 days of the trauma in these cases was less important for revealing postural imbalance; on the other hand, in spite of the fact that stabilometric examination was returning to normal in many patients, postural performance was nevertheless worse in the retroflexed condition than when the head was kept erect during the test, thus demonstrating that the destabilising activity of the cervical component was not related to the persistent vestibulopathy. To further demonstrate the role that spinal proprioception plays in postural modification before treatment, we calculated the cervical interference indices (SCI and LCI), which ere altered in both groups but with no statistically significant differences. Furthermore, approximately half of the subjects in both groups manifesting a vestibular disorder (pathologic VNG) had pathologic SCI and LCI, which confirms that static stabilometry can reveal and quantify the destabilising activity of the cervical proprioceptive component even when pathological nystagmus is present.

As far as alterations in posturographic recordings are concerned, we observed no specific pattern in OE and CE conditions in Group A. In the stabilogram, wide and arrhythmic oscillations on both planes were seen prevalently in the CE position, though they were more evident on the sagittal plane. The statokinesigram revealed an increase in the surface values in both OE and in CE. These data appear even more altered when heads were retroflexed (CER); even in this case, a clear increase in the surface values and in those corresponding to the shift from the centre of gravity on the sagittal plane prevailed compared with the guideline reference values. What is extremely interesting, and which confirms that the cervical proprioceptive

system is involved in cases of whiplash, is that when we compared the pre- and post-physiotherapy data for Group A statistically significant improvement was noted in the majority of parameters referring to the CER condition after therapy, considering both the mean values and the percentage of pathological patients. Likewise, there were no specific stabilometric patterns in Group B, in spite of the large number of patients with at least one pathologic stabilometric value. Even in this case, the statokinesigram revealed a clear increase in surface values, especially in the CE position, and frequent lateral shifting from the centre of gravity. Furthermore, there was no significant change in the overall characteristics of the stabilometric test in the CER position in Group B patients, even though the length and surface parameters were considerably pathologic for this position, which was noted in the pathological values of the LCI and SCI parameters as well. These posturographic data are also in agreement with the results of vestibular examination, which was pathological in a high percentage (60%) of cases in Group B. The stabilometric test evidently gives clearly pathologic results in patients who have suffered minor head injuries, since it is influenced both by the vestibulopathy present and by the altered vestibulo-spinal reflex following the cervical whiplash trauma. Confirmation of this possibility is found by comparing the first and the second stabilometric examination in Group B subjects, which was statistically significant only for parameters referring to the shift from the centre of gravity on the frontal plane. This improvement might be attributable to the progressive resolution of the associated vestibulopathy rather than to the physiotherapy. In fact, the non-compensated labyrinth deficits that were present in 20% of cases during the first examination appeared almost completely compensated in the second examination, and the number of PPV cases showed a 50% decrease from the first to the second test.

Multivariate analysis was performed to demonstrate whether one of the parameters might predict resolution of the vestibular condition. In this regard, the statistical analysis demonstrated that all the parameters taken into account (Xmed, SDX, Ymed, SDY, L, S, VEL, SD VEL for Group A and Xmed, Ymed, L, VEL for Group B) could be considered jointly predictive of the vestibular test returning to negative. In other words, in the case of whiplash and in minor head injuries no single stabilometric parameter can be considered for presuming the resolution of vestibulopathy. By increasing the number of cases, this type of analysis might be able to supply more significant data.

When a post-traumatic patient is examined, one must always keep in mind that exaggeration or simulation for claiming compensation for damages is possible, particularly during the static stabilometric test. In this study (and in agreement with the data in the literature), we retested all subjects, excluding 12 (11 with isolated cervical whiplash and one with a minor head injury associated with whip-

lash) in whom we found no definite signs of vestibular-oculomotor (VOR) involvement and whose inter-correlation stabilometric recordings were clearly sinusoidal ^{25 44}.

Conclusions

From what has been described above, it appears evident that the evaluation of neuro-otological data following cervical whiplash and/or minor head injuries is quite problematic. This study confirms that both minor head injuries and whiplash (the latter in a lower percentage of cases) can cause both peripheral and central vestibular damage that is occasionally transient. Videonystagmography can reveal not only pathological nystagmus, but can also follow the evolution of the post-trauma neuro-otological damage over time. Moreover, the study also confirms what has already been reported; namely, that considerable postural instability can occur after an episode of cervical whiplash even if there is no pathological nystagmus, and that this is prevalently attributable to a modification in the cervical proprioceptive input and, consequently, in the vestibular spinal reflex. Static stabilometry not only contributes to the diagnosis of the vestibular spinal reflex damage, but also reveals and quantifies the destabilising activity of the cervical proprioception component both in subjects with cervical whiplash and in those with minor head injuries associated with indirect spinal trauma. Furthermore, stabilometry can also show improvements in postural performance achieved through functional rehabilitation of cervical neuro-muscular-skeletal structures. Nevertheless, while these data demonstrate that it is important to perform clinical and instrumental neuro-otological examination in subjects who complain of persistent postural imbalance after they have suffered cervical whiplash and/or minor head injuries, it must be stressed that further evaluation and confirmation of data from larger study groups and for longer periods of follow-up are necessary.

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