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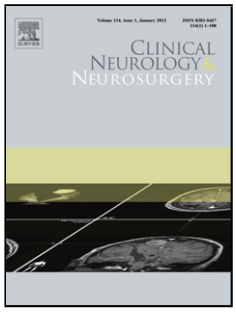

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Patients with chronic dizziness following traumatic head injury typically have multiple diagnoses involving combined peripheral and central vestibular dysfunction

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Highlights

- Commonest causes of dizziness following head injury are either BPPV or Vestibular migraine
- 30% of patients have combined peripheral and central vestibular dysfunction
- 80% of patients recover fully 2 years post injury.

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Abstract

Objective: We hypothesised that chronic vestibular symptoms (CVS) of imbalance and dizziness post-traumatic head injury (THI) may relate to: (i) the occurrence of multiple simultaneous vestibular diagnoses including both peripheral and central vestibular dysfunction in individual patients increasing the chance of missed diagnoses and suboptimal treatment; (ii) an impaired response to vestibular rehabilitation since the central mechanisms that mediate rehabilitation related brain plasticity may themselves be disrupted.

Methods: We report the results of a retrospective analysis of both the comprehensive clinical and vestibular laboratory testing of 20 consecutive THI patients with prominent and persisting vestibular symptoms still present at least 6 months post THI.

Results: Individual THI patients typically had multiple vestibular diagnoses and unique to this group of vestibular patients, often displayed both peripheral and central vestibular dysfunction. Despite expert neuro-otological management, at two years 20% of patients still had persisting vestibular symptoms.

Conclusion: In summary, chronic vestibular dysfunction in THI could relate to: (i) the presence of multiple vestibular diagnoses, increasing the risk of 'missed' vestibular diagnoses leading to persisting symptoms; (ii) the impact of brain trauma which may impair brain plasticity mediated repair mechanisms. Apart from alerting physicians to the potential for multiple vestibular diagnoses in THI, future work to identify the specific deficits in brain function mediating poor recovery from post-THI vestibular dysfunction could provide the rationale for developing new therapy for head injury patients whose vestibular symptoms are resistant to treatment.

Keywords; Vestibular dysfunction, Traumatic head injury, Chronic dizziness

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INRODUCTION:

Traumatic head injury (THI) is the commonest cause of disability in young adults[1] and chronic vestibular symptoms (CVS) of dizziness and imbalance are amongst the commonest causes for post-traumatic morbidity affecting up to half of patients at 5 years[2,3]. Vestibular symptoms are an independent predictor of failure to return to work with two-thirds of mild THI patients with vestibular symptoms not back at work at 6 months compared to one-quarter of THI patients without vestibular symptoms[3].

Despite its importance, the reasons for CVS following head trauma are unclear. One reason for the development of CVS in THI may be the failure to make a correct diagnosis, a necessary first step in formulating effective therapy. Another reason could be the disruption of central mechanisms that themselves mediate recovery from vestibular dysfunction, be they peripheral or central. Given these considerations, we routinely assess THI patients using a comprehensive clinical and laboratory battery (contrastingly, in non-THI patients we apply a more focussed approach). Our comprehensive testing approach meant that patients received the same comprehensive evaluation even if there was an initial obvious vestibular diagnosis. Such an approach is less likely to miss vestibular diagnoses when multiple, and improve the identification of the full gamut of vestibular deficit, particularly in cases with combined peripheral and central vestibular dysfunction.

METHODS:

Study population; We retrospectively studied from January 2011- November 2012 a consecutive cohort of twenty patients referred to a tertiary referral balance clinic with post-THI dizziness and/or imbalance still present for at least 6 months following their head injury (no exclusion criteria). At the time of initial assessment, none of the patients had received any treatment or intervention for their dizziness symptoms.

Standard protocol approvals, registrations, and patient consents; Patient data were obtained as standard of care for patients referred to a regional neuro-otology service and were reviewed under the waiver of consent category.

Clinical assessment and specialist neuro-otological testing in the Neuro-Otology clinic; All patients were clinically assessed by a Neuro-Otology consultant (BMS) and then underwent a comprehensive vestibular battery by an experienced vestibular scientist (QA) to assess both peripheral and central vestibular function including: (i) bi-thermal caloric irrigations (cold 30° C and warm water 44° C); (ii) rotational chair testing (i.e. velocity step rotations 90°/s); (iii) vestibular evoked myogenic potentials (VEMP); (iv) smooth pursuit at four different frequencies (0.1, 0.2, 0.3 and 0.4 Hz); (v) VOR suppression (0.25 Hz, 40°/sec); and (vi) optokinetic stimulation (OKS) (40°/s). Functional asymmetries were assessed using paired t-tests. All data was compared to 20 age and sex matched normal controls.

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All patients had initially been admitted to a regional Trauma Unit where admission Glasgow Coma scores (GCS) are routinely assessed. Additionally all patients had cognitive function assessed in a specialist traumatic brain injury clinic using the Addenbrookes cognitive examination (ACE-R).

RESULTS

Patient demographics and baseline clinical data; The mean age of the patient cohort was 44.7 years (SD= 13.6, range 19-69), comprising 12 males. The time since head injury ranged from 6-18 months. The average GCS at time of admission to the trauma centre was 10.2 (SD = 4.6). The mean ACE-R score in the patients was 78.4 (10.4) (Table 1).

Neuro-otology assessment

The clinical diagnoses in the 20 patients (Table 1) were found to be as follows; benign positional paroxysmal vertigo (BPPV) (N=8), vestibular migraine (n=4)[4], and both BPPV and vestibular migraine (n=8). Laboratory testing showed that only two out of 20 patients displayed overt peripheral vestibular abnormality, with significant asymmetries on caloric testing (canal paresis of 43% and 70% respectively), rotational responses and VEMP testing. The testing abnormality in these two patients confirmed the clinical examination (i.e. positive head impulse test) and was attributable to traumatic vestibular nerve transection. VEMP testing did not otherwise reveal any significant asymmetries in either right or left P13 or N23 components or amplitude ($p>0.3$; paired-samples t-test). Note, that in six patients we did not elicit VEMPs, however five of these patients were over 55 years of age and VEMPs are often absent in the healthy elderly[5] . No differences were observed in vestibular dysfunction when comparing patients with different head injury severity as assessed by the initial GCS score, possibly attributable to a lack of variance in the GCS (see table 1).

Central vestibular dysfunction as evidenced by impaired VOR suppression and significantly broken pursuit (i.e. compared to our normal controls; patients typically had gain below 0.40; range 0.1-0.4 mean gain =2.8, SD 1.7), was found in 30 % (6 out of 20) of our patient cohort, typically with both peripheral and central vestibular involvement (Viz. 2 vestibular migraine, 1 vestibular migraine + BPPV and 3 BPPV). The gain of OKN was normal for all patients (i.e. no difference to our control data; range 0.62-1.04, mean 0.90, SD=0.18). Hence, despite the fact that broken pursuit and impaired VOR suppression are poorly localising they do provide strong signs indicating central vestibular dysfunction. It was ensured that during testing all subjects were fully attentive and cooperated fully with the testing procedure.

Long-term outcome of vestibular symptoms;

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Upon following up our patient sample 24 months after the initial consultation, sixteen patients reported no further symptoms. Of the four patients with persisting vestibular symptoms at 2 years, one patient was diagnosed with vestibular migraine (and no other vestibular diagnoses), one with vestibular migraine, BPPV and central vestibular dysfunction and two patients were diagnosed with combined vestibular migraine and BPPV. Accordingly, the success rate of treating our patient sample was 80% at a 2 year follow up time point.

DISCUSSION

Our data are of importance for those involved in the management of head injury patients as it highlights that the two commonest causes of chronic post-head injury dizziness are BPPV and vestibular migraine. However, unfortunately we did not ascertain whether those patients that developed migraine post-THI, had a previous history of migraine and future studies should aim to examine this directly.

Notably both vestibular migraine and BPPV are treatable, i.e. with repositioning manoeuvres for BPPV[6] and with pharmacotherapy with drugs such as propranolol, topiramate or amitriptyline for vestibular migraine[7]. The two patients with peripheral vestibular loss (i.e. vestibular nerve transection) were referred to a vestibular physiotherapist for a customised programme of vestibular rehabilitation.

Furthermore, 40% of our sample had two or more vestibular diagnoses, so a thorough evaluation is required to detect all potential vestibular diagnoses. In addition, 30% of our patient cohort displayed central vestibular dysfunction, in particular, impaired VOR suppression and/or broken pursuit. Previous workers have aimed at classifying post-traumatic CVS with the aim of diagnostic simplification[8], an approach that is usually appropriate for most vestibular disorders. However although such diagnostic simplification could theoretically expedite clinical assessment and improve research efficiency, at least in head trauma patients, it does so at the cost of underplaying the occurrence of multiple simultaneous vestibular diagnoses.

An important take home message is that, to avoid missing all relevant vestibular diagnoses in THI, the clinician should not stop looking once the first vestibular diagnosis is made. Practically this means that, in contrast to other vestibular patient groups where a focussed clinical and laboratory approach is appropriate, in head injury we recommend a comprehensive clinical and laboratory ‘battery’ approach. That is, unlike other causes of dizziness, head injury results in typically multiple albeit treatable vestibular diagnoses, often combining peripheral and central vestibular dysfunction.

Given this complexity, it is likely that at least in part, chronic vestibular symptoms in head injury may result from a failure to detect all vestibular diagnoses in a given patient. The good news is that of patients referred to us with chronic dizziness from head injury, 80% showed a resolution of vestibular symptoms at two years. However, even with a vigorous attempt to define and treat known causes of head injury dizziness, 20% of patients in our cohort were still symptomatic at two years. We speculate that the unique combination of central and peripheral vestibular diagnoses in head injury may be important in the pathogenesis of chronic dizziness in this group. In particular, given that central vestibular plasticity is critically involved in vestibular adaptation[9,10],

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disruption of central vestibular pathways may theoretically render the recovery from any form of vestibular injury (peripheral or central) less liable to respond to standard vestibular therapy. Developing treatment for those patients with refractory post-THI dizziness and imbalance will require a better understanding of how central deficits may impede recovery from vestibular dysfunction in THI.

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Table

Table 1: Clinical Summary of Patient demographics, vestibular function tests, Glasgow coma scores and diagnosis.

Patient number & sex	Age	Bithermal caloric irrigation		Vemps (Vestibular-evoked myogenic responses)			Velocity step responses 0 to 90deg/s		Velocity step responses 90 to 0deg/s		VOR Sinusoidal Responses (0.25Hz)	Optokinetic nystagmus	Smooth pursuit gain (at stimulus frequencies shown)				VOR SUPPRESSION	Admission Glasgow Coma Score	Diagnose
Patient		Canal Paresis (%)	Directional Preponderance (%)	P13 (ms)	N23 (ms)	Amplitude (mV)	Gain	Time constant (s)	Gain	Time constant (s)	Gain	Gain	0.1Hz	0.2 Hz	0.3 Hz	0.4 Hz	N - normal	GCS Score 3-15	(see no below)
1F	19	9R	17L	17.5	26.05	0.2665	0.66	18.9	0.495	22.6	0.465	1.085	0.93	0.885	0.775	0.755	Impaired	9	1, 3
2M	58	-	-	-	-	-	0.7	14.95	0.65	15.75	0.675	1.02	0.425	0.365	0.265	0.2	Impaired	10	2, 3
3M	24	0	2R	17.4	25.35	0.211	0.76	21.6	0.7	18.85	0.775	1.02	0.775	0.72	0.825	0.675	N	10	1
4M	46	23L	29R	20.4	32.1	0.1695	0.7	17	0.7	16.5	0.8	1.045	0.85	0.9	0.925	0.95	N	15	1
5M	50	20R	31L	-	-	-	0.7	13	0.66	13.5	0.8	0.875	1.05	1.045	1.025	1.04	N	11	2
6F	57	2R	12L	14.35	21.45	0.207	0.675	16.5	0.74	17.5	0.695	1	1.045	0.85	0.675	0.65	N	12	1, 2
7F	41	2L	14L	16.15	23.7	0.147	0.575	14.45	0.625	14.75	0.715	0.89	0.7	0.575	0.6	0.565	Impaired	10	1, 2,
8M	29	43L	40R	-	-	-	0.54	7.8	0.56	10.45	0.605	0.875	0.945	0.86	0.88	0.85	N	10	1, 3
9F	27	8R	26L	13.95	25.95	0.2665	0.535	11.75	0.6	13.9	0.635	1.025	0.725	0.58	0.55	0.555	N	9	2
10M	54	7R	3L	15.6	29.95	0.2945	0.515	7.35	0.56	9.55	0.73	0.895	0.885	0.76	0.725	0.705	N	10	1, 2
11F	30	2R	0	14	28	0.235	0.765	19.7	0.81	20.85	0.85	1.02	0.375	0.15	0.1	0.05	Impaired	8	1, 2,
12F	36	7R	26L	14.7	23.35	0.1365	0.475	9.55	0.49	10.7	0.775	1.075	0.925	0.935	0.895	0.875	N	13	1, 2
13M	51	4L	11R	17.1	27.3	0.344	0.41	10	0.475	11.1	0.7	0.905	0.94	0.97	1.05	0.915	N	8	1, 2
14M	69	1R	7R	-	-	-	0.575	13.95	0.53	12.15	0.815	0.635	0.375	0.275	0.34	0.295	Impaired	8	1, 2,
15M	59	1R	0	-	-	-	0.47	7.85	0.415	6.4	0.45	0.675	0.775	0.575	0.425	0.4	N	8	1
16F	60	4R	7L	-	-	-	0.59	14.7	0.56	14.4	0.435	1.06	0.98	0.915	0.88	0.795	N	8	1, 2
17M	34	6R	14L	16.1	27.45	0.1615	0.6	8.85	0.59	11.2	0.61	0.985	0.95	0.965	0.985	1.02	N	10	1, 2
18M	48	0	0	13.1	24	0.2085	0.585	10.15	0.515	13	0.5	0.735	0.745	0.76	0.7	0.625	Impaired	10	1, 3
19F	55	5R	4L	-	-	-	0.675	6.35	0.565	7.4	0.65	0.885	0.875	0.805	0.75	0.76	N	9	2
20M	47	73R	14L	-	-	-	-	-	-	-	0.575	-	0.55	0.515	-	-	Impaired	13	1, 3,

****Diagnoses: 1.** BPPV; **2.** Vestibular migraine; **3.** Central vestibular dysfunction (impaired VOR suppression); **4.** Vestibular nerve transection.