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The spectrum of nystagmus following cerebro-vascular accident

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Abstract

Aim: **To report the features of nystagmus documented following a confirmed diagnosis of cerebro-vascular accident (stroke).**

Methods: **A multi-centre prospective observational study was undertaken in 14 acute Trust hospitals. Stroke survivors with suspected visual difficulty were recruited. Standardised screening/referral forms and investigation forms were employed to document data on visual impairment, specifically assessment of visual acuity, ocular pathology, eye alignment and movement, visual perception (including inattention) and visual field defects.**

Results: **Of 323 patients, 38 were found to have nystagmus following cortical, brain stem or cerebellar stroke. Twenty were male and 18 female, with a mean age of 65 years. Acquired nystagmus accounted for 84% of the types documented. Four patients had oscillopsia and 3 had vertigo. Associated ocular motility deficits were found in 84% of patients and treatment was largely aimed at alleviating diplopia but also reading difficulties or blurred vision. Improvement was noted in 42%.**

Conclusion: **Twelve per cent of stroke survivors with suspected visual difficulties had nystagmus documented. Most had associated ocular motility defects. Symptoms relating to the nystagmus of oscillopsia and vertigo were reported in 18%. Improvement of ocular motility was recorded in 42%.**

Key words: Cerebro-vascular accident, Nystagmus, Ocular motility, Oscillopsia, Stroke

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Introduction

Cerebro-vascular accident (stroke) is the third biggest killer in the UK, affecting 130 000 individuals each year.[1] Many of those who survive a stroke are left with physical and/or cognitive disability, which requires significant rehabilitation.

Visual impairment has received little attention in the general medical community and the impairments recognised after stroke typically only include visual field loss (homonymous hemianopia) or visual inattention/neglect. Ocular motility disorders have been recognised as a sequela to stroke with a prevalence of 20–57%[2–4] and encompass many types including strabismus, gaze palsy, cranial nerve palsy and skew deviation. Nystagmus is mentioned rarely.

In a current prospective trial of visual impairment following stroke, the prevalence estimate of nystagmus was found to be 12% from a 1-year data set of results. The purpose of this paper is to report this subgroup of patients in terms of stroke demographics, type of nystagmus, symptoms and recovery.

Methods

The design of this study is a prospective multi-centre observational case cohort trial. Multi-centre ethics approval has been granted for this study with involvement of the R&D unit at each participating centre. The Vision In Stroke (VIS) group consists of the local investigators responsible for assessing stroke patients and collecting patient data. The data are collated centrally at the University of Liverpool. The study is being undertaken in accordance with the Tenets of Helsinki.

The target population was stroke patients suspected of having a visual difficulty. Referrals could be made from in-patient wards or rehabilitation units, community services or out-patient clinics. Patients were given an information sheet available in both detailed and brief format. They were recruited after informed, written consent was obtained. Patients were excluded from the study if they were unable to consent due to cognitive impairment, unwilling to consent, if their diagnosis was that of transient ischaemic attack as opposed to stroke, if they died prior to their vision assessment or were discharged prior to vision assessment.

The trial consists of the use of a screening form by which patients with suspected visual difficulty are identified. Subsequently this screening form is used as the referral form to the orthoptic service. A standardised investigation sheet is used for the eye assessment.

Table 1. Area of stroke

Nystagmus description	Area of brain/artery involved by stroke[a]
Abducting	• Brain stem (3 cases) • Lacunar
Congenital	• Partial anterior circulation infarct
Convergent retraction	• Thalamus (2 cases)
End point	• Cerebellum • Occipital lobe • Multiple areas (5 cases)
Gaze-evoked	• Brain stem • Cerebral peduncle • Lacunar • Occipital lobe (2 cases) • Parietal lobe • Periventricular • Thalamus (2 cases) • Middle cerebral artery • Partial anterior circulation infarct • Posterior cerebral artery
Latent	• Multiple area
Multivector	• Cerebellum • Multiple area (2 cases)
Pendular	• Cerebellum
Rotary	• Multiple area • Parietal lobe
Upbeat	• Brain stem • Cerebellum • Lacunar • Multiple area • Middle cerebral artery

[a]One case only affected unless otherwise stated.

Table 2. Ocular motility deficits associated with acquired nystagmus

Nystagmus description	Associated ocular motility findings[a]
Abducting	• Internuclear ophthalmoplegia (3 cases) • One and a half syndrome
Convergent retraction	• Dorsal midbrain syndrome with VI nerve palsy • Vertical gaze palsy with VI nerve palsy
End point	• IV nerve palsy with hypometric saccades • Weber's syndrome • Saccadic palsy
Gaze-evoked	• Dorsal midbrain syndrome • Smooth pursuit palsy • Gaze palsy (3 cases) • Reduced elevation with horizontal gaze palsy • Gaze palsy with hypometric saccades (3 cases) • Reduced elevation with hypometric saccades (3 cases)
Multivector	• III nerve palsy • Reduced elevation with VI nerve palsy • Reduced elevation with VI nerve palsy, horizontal gaze palsy and hypometric saccades
Pendular	• Smooth pursuit palsy
Rotary	• III nerve palsy • Hypometric saccades
Upbeat	• Gaze palsy with VI nerve palsy • Gaze palsy with hypometric saccades • Reduced elevation with smooth pursuit palsy • Reduced elevation with hypometric saccades • Vertical and horizontal gaze palsy

[a]One case only affected unless otherwise stated.

The eye assessment consists of identification of known pre-existent ocular pathology, symptoms and signs, investigation of the visual field, ocular motility and perceptual aspects. Stroke details are recorded from patient notes, accounting for stroke laterality, type and area involved. Ocular treatment details are also recorded along with outcome.

Results were inputted to the statistical package SPSS version 15. Non-parametric assessment of results was undertaken for non-uniform distributed data including visual acuity, ocular alignment and motility, and the visual perception results.

Results

General demographics

Data are presented from the first year of data collection: 1 May 2006 to 30 April 2007. One hundred and seventy-eight patients were excluded (49% male, 51% female). The most common reason for exclusion (52%) was an inability to consent because of cognitive difficulties and thus an inability to understand the patient information and the purpose of the study. Three hundred and twenty-three patients were recruited (59% male, 41% female). The mean age at onset of stroke was 69 years (range 1–92 years: SD 15 years; median 72 years). The median duration from onset of stroke to initial eye examination was 22 days (range 0–2543 days), the mean of 70 days being skewed by three outliers (patients referred a number of years after the onset of their stroke).

Strokes were cortical in 88.6% and brain stem or cerebellar in 11.4%. The lesion was right-sided in 48%, left-sided in 40% and bilateral in 12%. Infarcts accounted for 79.5%, the remainder being haemorrhagic in type.

Thirty-eight patients (12%) were found to have nystagmus on first visual assessment. There were 20 men and 18 women, with a mean age of 65 years: SD 13.46 (range 29–86 years). These patients were seen at a median of 19 days after the onset of stroke.

Stroke demographics for nystagmus patients

The stroke type was an infarct in 84% and haemorrhage in 16%, right-sided in 37%, left-sided in 42% and bilateral in 21%.

The areas of brain or artery involved by the stroke are outlined in Table 1. The cerebellum was affected in 5 patients, brain stem in 5 patients, thalamus in 4 patients, lacuna in 3 patients, occipital lobe in 3 patients, parietal lobe in 2 patients and periventricular area in 1 patient. The middle cerebral artery was involved in 2 patients, partial anterior circulation in 2 patients and posterior cerebral artery in 1 patient. The remaining 10 patients had multiple cortical areas involved by their stroke.

Type of nystagmus

Congenital or longstanding nystagmus included 1 case of idiopathic congenital nystagmus and 1 case of latent nystagmus. End point nystagmus was documented in 7 patients, 4 with entirely normal ocular rotations and 3 with associated ocular motility deficits including Weber's syndrome, saccadic palsy and fourth nerve palsy with saccadic hypometria.

Cases of acquired nystagmus included upbeat, pendular, rotary, abducting, convergence retraction, gaze-evoked and multivector nystagmus. All cases of acquired nystagmus were associated with ocular motility deficits as outlined in Table 2.

Table 3. Outcome for nystagmus patients

Nystagmus description	Follow-up appointments			Visual status (within 2 months)	
	Discharge	Review	Referral	Improvement	Static
Abducting		3	1	3	
Congenital	1				1
Convergent retraction		2		1	
End point	3	3	1		2
Gaze-evoked	1	8	3	5	3
Latent		1		1	
Multivector		3		2	1
Pendular		1		1	
Rotary	1	1		1	1
Upbeat	2	3		2	1
Total	8 (21%)	25 (66%)	5 (13%)	16	9

Symptoms

Four patients had the primary symptom of oscillopsia. Three patients reported vertigo with balance problems. Other symptoms included diplopia, blurred vision, visual field loss, reading difficulty and difficulties with spatial awareness.

Treatment and recovery

Treatment options included occlusion, prisms, refraction, typoscope, orthoptic exercises, magnifiers and advice. The advice incorporated information provision on scanning strategies, appropriate lighting, reading strategies, visual field or inattention awareness and use of a compensatory head posture.

Eight patients were discharged from orthoptic care, 25 were offered review appointments and 5 were referred for ophthalmic assessment. Patients were reviewed at varying time intervals from 1 week to 2 months following their first appointment (median 2 weeks). Sixteen patients showed improvement on review but did not attain normal ocular motility. Nine patients had no change in their visual status. Table 3 shows the outcome of these patients.

Discussion

Nystagmus is reported in the stroke population in various case reports and case series. However, there has been no estimate of the prevalence of nystagmus due to stroke. Following a 1-year prospective review of stroke patients, nystagmus was recorded in 12% of patients. This figure cannot be taken as a prevalence for nystagmus occurring in stroke survivors, however, as these patients were those recruited with suspected visual difficulty. This excludes those patients who could not be recruited despite being referred with suspected visual difficulty, and also patients not referred as visual difficulty was not suspected.

There is a vast array of types of nystagmus caused by strokes in cortical and subcortical areas of the brain. The areas affected by pathology that typically produce nystagmus include the cerebellum, thalamus, brain stem and posterior fossa areas. Specifically, nystagmus has been documented following 'top of the basilar syndrome', lesions of the brain stem and basal ganglia, vestibular nerve irritation or cerebellar damage.[3,5–11] Less commonly nystagmus is reported after cortical stroke and these are notably posterior cortical lesions.[12,13] A number of patients in this study also had strokes affecting the brain stem and cerebellum. However, over 50% of patients had strokes affecting cortical areas, many of which involved multiple areas. Thus the lack of localising ability for the presence of nystagmus is evident for these latter cases.

The types of nystagmus reported following stroke are many. Upper brain stem strokes typically result in convergence retraction nystagmus,[5,10] rebound nystagmus,[8] pendular nystagmus[14] and abducting nystagmus.[15] Rebound nystagmus has also been documented following cerebellar stroke.[11] Gaze-evoked nystagmus is seen in recovering gaze palsies.[3,6] Cerebellar damage may give rise to horizontal jerk nystagmus,[4] as may infarcts in the parietal lobe.[12] Infarcts of the hippocampus and basal ganglia have resulted in multivector nystagmus (horizontal and torsional).[7] A number of articles have reported the clinical signs following medial medullary infarction, with many documenting nystagmus ranging from ipsilesional, gaze-evoked, upbeat and hemiseesaw.[9,16–18] The acquired types of nystagmus documented in our patients were similar to those reported in the literature, with many related to stroke lesions in the brain stem, cerebellum, lacunar and thalamic areas and posterior cortical areas.

Oscillopsia is a difficult symptom to treat. Fortunately only 4 patients had this distressing symptom in this group of patients. In some patients there is also the symptom of vertigo.[3,7,8,12,18] Indeed, Huang and Young[8] suggested that patients presenting with rebound nystagmus and the symptom of vertigo should be considered as a potential candidates for progression to basilar artery stroke. Three patients in this study had the symptoms of vertigo and balance difficulty. These related to the presence of upbeat nystagmus in 1 case and 2 patients with gaze-evoked nystagmus.

None of the patients were treated specifically for their symptom of oscillopsia in that they were not offered drug therapies that are known to be beneficial to many cases of acquired nystagmus.[19] This no doubt relates to the priority of treating the cause of stroke first and dealing with associated ocular symptoms and signs at a later stage. The treatment options that could be offered largely related to alleviating reading difficulties, blurred vision and diplopia due to the associated ocular motility deficits, namely prisms, occlusion, refraction, magnifiers and typoscope use. Advice was also provided to patients to aid reading or improve awareness of their visual status.

Twenty-five patients were offered orthoptic review

appointments. Of these, 16 showed improvement and 8 had static visual status on follow-up. None of the nystagmus cases showed a complete resolution of their nystagmus and/or associated ocular motility although nystagmus types such as abducting or gaze-evoked nystagmus showed considerable improvement. Of the patients reviewed, none showed any further deterioration of the ocular motility. The time period over which improvement was documented ranged from 1 week to 2 months with a median of 2 weeks. This time frame is notable as it potentially could be utilised as a prognostic factor for recovery of nystagmus. Patients who have not shown improvement in their nystagmus by 2 months after onset of stroke are those most likely to have persistent nystagmus.

Interestingly only 5 patients (13%) had their nystagmus noted by the referring staff on the stroke ward/rehabilitation unit. These cases included rotary nystagmus, congenital nystagmus and 3 cases of gaze-evoked nystagmus. This relates in part to some types only becoming apparent on ocular rotation away from primary gaze, such as cases of internuclear ophthalmoplegia and convergence retraction nystagmus, but would also relate to the frequency of nystagmus.

Conclusions

Nystagmus is not uncommon following stroke and was documented in 12% of patients recruited to this study. It should be recognised that this is likely to under-represent the true prevalence of nystagmus in the overall stroke population. Acquired nystagmus accounted for the majority of nystagmus types documented (84%) and symptoms of oscillopsia and vertigo were reported in 18% of our patients. However, treatment was targeted not at the nystagmus but at alleviating coexistent diplopia, reading difficulties and blurred vision. Forty-two per cent showed improvement in their ocular motility on orthoptic review.

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