

Visual impairment following stroke: do stroke patients require vision assessment?

FIONA ROWE¹, DARREN BRAND², CAROLE A. JACKSON², ALISON PRICE³, LINDA WALKER⁴,
SHIRLEY HARRISON⁵, CARLA ECCLESTON⁶, CLAIRE SCOTT⁷, NICOLA AKERMAN⁸,
CAROLINE DODRIDGE⁹, CLAIRE HOWARD¹⁰, TRACEY SHIPMAN¹¹, UNA SPERRING¹²,
SONIA MACDIARMID¹³, CICELY FREEMAN¹⁴

¹ Directorate of Orthoptics and Vision Science, University of Liverpool, Thompson Yates Building, Brownlow Hill, Liverpool L69 3GB, UK

² Department of Orthoptics, Royal United Hospitals NHS Trust, Combe Park, Bath BA1 3NG, UK

³ Department of Orthoptics, Sandwell and West Birmingham NHS Trust, City Hospital, Birmingham B18 7QH, UK

⁴ Department of Orthoptics, East Lancashire Hospitals NHS Trust, Royal Blackburn Hospital, Lancashire, BB2 3HH, UK

⁵ Department of Orthoptics, Bury PCT, Bury, Lancashire BL9 0EN, UK

⁶ Department of Orthoptics, Derby Hospitals NHS Foundation Trust, Derby City General Hospital, Derby, Derbyshire, DE22 3NE, UK

⁷ Department of Orthoptics, Ipswich Hospital NHS Trust, Ipswich, Suffolk IP4 5PD, UK

⁸ Department of Orthoptics, University Hospitals NHS Trust, Southampton, Hampshire SO16 6YD, UK

⁹ Department of Orthoptics, Oxford Radcliffe Hospitals NHS Trust, Headley Way Headington, Oxford OX3 9DU, UK

¹⁰ Department of Orthoptics, Salford Primary Care Trust, Pendleton Way, Salford M6 5FW, UK

¹¹ Department of Orthoptics, Sheffield Teaching Hospitals Foundation Trust, Glossop Road, Sheffield S10 2JF, UK

¹² Department of Orthoptics, Swindon and Marlborough NHS Trust, Marlborough Road, Swindon SN3 6BB, UK

¹³ Department of Orthoptics, Wrightington, Wigan and Leigh NHS Trust, Wigan Lane, Wigan WN1 2NN, UK

¹⁴ Department of Orthoptics, Worcestershire Acute Hospitals NHS Trust, Charles Hasting Way, Worcester WR5 1DD, UK

Address correspondence to: F. Rowe. Tel: (+44) 0151 7945732; Fax: (+44) 0151 7945781. Email: rowef@liverpool.ac.uk

Abstract

Background: the types of visual impairment following stroke are wide ranging and encompass low vision, eye movement and visual field abnormalities, and visual perceptual difficulties.

Objective: the purpose of this paper is to present a 1-year data set and identify the types of visual impairment occurring following stroke and their prevalence.

Methods: a multi-centre prospective observation study was undertaken in 14 acute trust hospitals. Stroke survivors with a suspected visual difficulty were recruited. Standardised screening/referral 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: three hundred and twenty-three patients were recruited with a mean age of 69 years [standard deviation (SD) 15]. Sixty-eight per cent had eye alignment/movement impairment, 49% had visual field impairment, 26.5% had low vision and 20.5% had perceptual difficulties.

Conclusions: of patients referred with a suspected visual difficulty, only 8% had normal vision status confirmed on examination. Ninety-two per cent had visual impairment of some form confirmed which is considerably higher than previous publications and probably relates to the prospective, standardised investigation offered by specialist orthoptists. However, under-ascertainment of visual problems cannot be ruled out.

Keywords: *visual impairment, stroke, ocular motility, visual field, visual perception, elderly*

Introduction

Every year, an estimated 110,000 people in the UK have a stroke [1]. The visual sequelae of stroke are many [2–9].

Low vision can relate to associated vascular pathology or to other co-existent ocular abnormalities [10]. Visual field loss commonly will involve a homonymous hemianopia or quadrantanopia [3, 4, 7, 12]. Ocular motility disorders can be

divided into cortical deficits of eye movements and brain stem defects. Visual perception difficulties are wide ranging [13]. The most commonly recognised is visual inattention/neglect [8, 9].

Visual problems such as complete hemianopia or large strabismus can be spotted easily. Subtle disorders such as impaired fast eye movements and gaze defects can be difficult to identify without correct evaluation of the visual system. A significant proportion of patients in stroke units have unrecognised visual problems resulting in little or no advice or management [2, 14]. The eye-care team can address many of these issues directly along with timely feedback of such information to other health professionals involved in the care of these patients [11].

The impact of visual impairment can be wide ranging. The impact on functional performance can include general mobility, ability to judge distances due to diplopia or impaired stereo vision, reading impairment due to cortical or ocular dysfunction and visual hallucinations. Impact to quality of life includes changes to independent living, ability to drive, loss of confidence and links to depression [16–20].

Although it is recognised that many patients will suffer visual disability as a result of stroke, data are lacking as to the prevalence of disability, extent and recovery. There are little data with respect to strabismus and ocular motility disorders following stroke [3–5, 21].

In May 2006, a prospective multi-centre trial commenced in the UK. The primary aim is to evaluate the effect of stroke on vision: specifically ocular perception, alignment and movement, how these impact on quality of life and the information that is required from visual assessment for the multidisciplinary team. The purpose of this paper is to highlight the extent of visual impairment occurring within this stroke population.

Methods and materials

The design is a prospective multi-centre observational case cohort trial with ethical approval. The vision in stroke (VIS) group consists of 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 inpatient wards, rehabilitation units, community services or outpatient clinics. Patients were given an information sheet and recruited after informed, written consent. The patients were excluded if they were unable to consent, unwilling to consent, if their diagnosis was that of transient ischaemic attack or if they were discharged without vision assessment.

Patients with a suspected visual difficulty are identified using a screening form. Subsequently this is used as the referral form to the orthoptic service. A standardised investigation sheet is used for the eye assessment consisting of identification of known pre-existent ocular pathology, symptoms and

signs, investigation of visual field, ocular motility and perceptual aspects. Visual fields are assessed by confrontation if the patient is seen on the ward or rehabilitation unit. When seen in clinic, quantitative measures of visual field are undertaken by Humphrey automated perimeter and Goldmann manual perimeter. Complete homonymous hemianopia was defined as loss of visual field to one side from central fixation and the vertical meridian of the field outwards. Partial homonymous hemianopia was defined as loss of visual field to one side that was incomplete with some residual vision on the affected side near the vertical meridian of the field.

Assessment of ocular alignment and motility consists of cover test, evaluation of saccadic, smooth pursuit and vergence eye movements, retinal correspondence (Bagolini glasses), fusional vergence (20D or fusional range), stereopsis (Frisby near test), prism cover test and lid and pupil function.

Perceptual deficits are recorded after questioning of the patient and/or carers and relatives. Inattention is assessed by means of a combination of assessments including line bisection, Albert’s test, cancellation tests, memory tests using verbal description and drawing.

Visual acuity was assessed at near and distance fixation with Snellen and logMAR acuity tests. Low visual acuity was considered in two categories. The first defined low visual acuity as less than best corrected 6/12 Snellens acuity or 0.3 logMAR in accordance with UK driving standards [22]. The second defined low visual acuity as <6/18 Snellens acuity or 0.5 logMAR and equal to or better than 3/60 Snellens acuity as per World Health Organisation (WHO) guidelines [23]. Additionally, WHO define low vision as a corresponding visual field loss to <20 degrees in the best corrected eye. WHO define blindness as a visual acuity of <3/60 Snellens acuity or a corresponding visual field loss to <10 degrees in the best corrected eye.

Stroke details are recorded from patient notes accounting for stroke laterality, type and area involved. Ocular treatment details are recorded along with outcome.

Results were inputted to the statistical package SPSS version 15.

Results

Data are presented from a 1-year data set: 1 May 2006 to 30 April 2007. Five hundred and one patients were referred for visual assessment. One hundred and seventy-eight patients were excluded (49% male, 51% female: Table 1). Patients were not excluded because of the type or severity of

Table 1. Reasons for exclusion

Inability to consent (cognitive impairment)	52%
Unavailable for orthoptic assessment	21%
Diagnosis other than stroke	10%
Unwilling to participate in study	10%
Death prior to orthoptic assessment	5%
Did not attend orthoptic appointment	2%

stroke. An attempt was made to establish the visual status of these patients. Twenty-seven per cent had a visual field defect documented, 20% had ocular motility disturbance, 4% had low vision and 2% had perceptual difficulties including inattention. Fifteen per cent had combinations of visual field, ocular motility, low vision and perceptual deficits.

Three hundred and twenty-three patients were recruited (59% male, 41% female). The mean age at the onset of stroke was 69 years (1–92; SD 15 years). The median duration from the onset of stroke to initial eye examination was 22 days (0–2543 days), the mean of 70 days being skewed by three outliers (patients referred a number of years after the stroke onset). One patient had a recurrent stroke, hence their referral for new eye assessment. The other two patients were simply not referred for eye assessment when they first had their stroke. They did not have recurrent strokes and were finally referred as outpatients.

Strokes were cortical in 88.6% and brain stem or cerebellar areas 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. Cortical areas included frontal, parietal, temporal and occipital lobes, external and internal capsule, intraventricular and periventricular areas, thalamus, basal ganglia and lacunar areas. Patients who had a cortical stroke had mostly visual field loss or ocular motility abnormalities. Patients who had lesions in brain stem or cerebellar areas were most likely to demonstrate ocular motility abnormalities than any other type of ocular deficit.

Eight per cent of patients were found to have normal visual status when examined. For the remaining 92%, the visual deficits documented in this study were grouped into

1. Low vision
2. Eye movement deficit
3. Visual field impairment
4. Perceptual deficit.

Low vision

Low visual acuity of $<6/12$ or 0.3 best corrected acuity was documented in 26.5% of patients at near vision and 25% at distance vision. Low visual acuity of $<6/18$ or 0.5 best corrected acuity was documented in 13% of patients at both near and distance vision. Seventy-two per cent required spectacles to obtain best corrected visual acuity. Ocular pathology was noted in 34.7% of patients including cataract, age-related macular degeneration, diabetic retinopathy, glaucoma, myopic degeneration, amblyopia, retinal dystrophy, optic nerve disease and anoxia. Of patients with low vision of $<6/12$, 31% had low vision attributable to associated ocular pathology and not as a result of their stroke.

Eye movement deficit

A total of 68.4% of patients had an alignment or ocular motility defect confirmed on examination (Figure 1). Twenty-two per cent had manifest strabismus in primary gaze. Twelve per cent of patients had nystagmus. Reduced convergence

at <10 cm was recorded in 32.7% of patients. Symptoms included diplopia, oscillopsia or reading difficulty. The type of ocular motility defect was evaluated according to the stroke site (Table 2). The site of lesion was cortical in 83%. Seventeen per cent had lesions of the cerebellum or brain stem.

Visual field impairment

Almost half of patients (49.5%) had visual field impairment with complete homonymous hemianopia in 29.4%. Other types of visual field impairment included partial homonymous hemianopia, inferior or superior quadrantanopia, scotomas, altitudinal defects, macular sparing hemianopia and chequerboard defects. Visual field loss was right sided in 19.8%, left sided in 27.2% and bilateral in 2.5%.

The site of stroke was cortical in 94% (Table 2). Six per cent had lesions of the cerebellum or brain stem.

Perceptual deficit

A total of 20.5% of patients reported or had documented perceptual deficits (Table 2). The largest group were patients with left-sided visual inattention/neglect (14%). A small number of patients had visual hallucinations (2.5%) and object agnosia (2.2%). Three patients had a difficulty with depth judgement and one patient had acquired colour detection difficulties.

Discussion

This study is not an epidemiology study. We have looked prospectively at the prevalence of visual impairment in stroke survivors referred specifically for a vision assessment where there is an obvious visual deficit or symptom or where health care staff suspect there may be a visual deficit based on patient responses/behaviour. This constitutes a limitation for this study. There is a further population of patients who are not referred because a visual impairment is not suspected. It is unknown how many of these have undetected visual impairment and this warrants further research.

We found 92% of patients to have documented visual impairment. Previous reported retrospective case reviews have reported visual impairment in 62–71% [3]. Our prevalence estimates are higher and probably relate to the prospective design of this trial and the use of standardised investigation forms administered by specialist orthoptists. We classed visual impairment as low vision, eye movement deficits, visual field impairment and perceptual difficulties. A total of 54.8% had a combination of two or more visual impairments.

Low vision accounted for 26.5% of visual impairment which is similar to a previous study on low vision in stroke [10]. This could be attributed to co-existent ocular diseases in approximately one-third of cases. Of the remaining cases, it is possible that some patients may have had pre-existent low vision without co-existent ocular disease but it was not possible to establish whether this was the case in the absence of past visual acuity measures. It is likely, however, that many

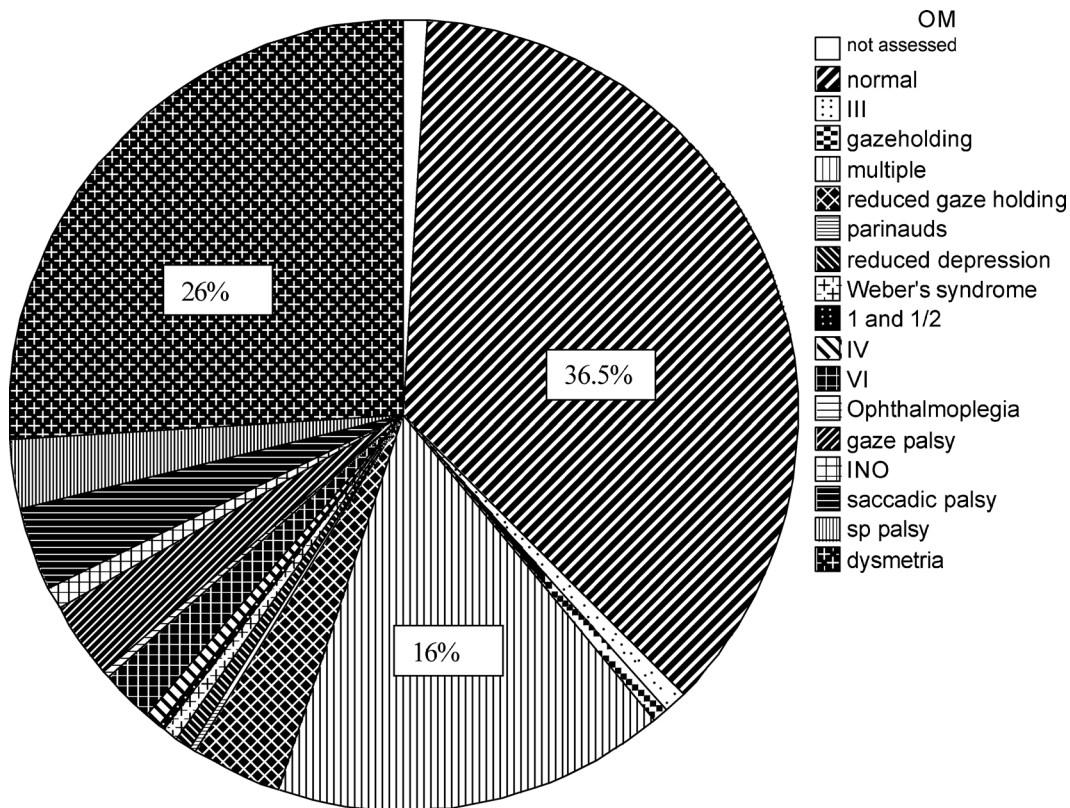


Figure 1. Ocular motility abnormalities. 36.5% of patients had normal ocular motility on evaluation. The largest individual category of impairment was saccadic dysmetria (26%); an inability to make accurate fast eye movement. Impaired and reduced gaze holding (5%), Saccadic palsy (3%), Gaze palsy (3%), Smooth pursuit palsy (2.5%), VI nerve palsy (2%), III nerve palsy (1%), INO (1%). 1/2% involved for each category of Parinaud’s dorsal midbrain syndrome, Reduced depression, Weber’s syndrome, one and half syndrome and IV nerve palsy. Of patients with multiple ocular motility impairments (16%), 12% had saccadic dysmetria in association with other motility deficits and 11% had gaze impairments in association with other motility deficits. III Third nerve palsy; IV Fourth nerve palsy; VI Sixth nerve palsy; 1 and 1/2 One and a half syndrome; INO Internuclear ophthalmoplegia; SP palsy Smooth pursuit palsy.

of these remaining cases had the recent onset of poor vision as this was often a new symptom for them. It is important that low vision is identified in older patients. In many cases it is treatable. Importantly, undiagnosed or untreated low vision is a risk factor for falls and is also linked with depression and reduced activity of daily living performance [15, 16, 24, 25]. Low vision can also impact on the rehabilitation of stroke [15].

Eye movement and alignment deficits accounted for 68.4% of visual impairment compared to reported figures of up to 52% [3, 5, 21, 26]. Nystagmus and manifest strabismus were documented and patients were aware of oscillopsia and diplopia. Impaired convergence which contributed to a reading difficulty was found in 33%. However, it was often other factors that led to reading difficulties such as problems tracking, inattention and visual field defects.

It is important to recognise that improvement can be noted with ocular motility treatment [6, 27]. Our patients were treated according to their symptoms. Those with diplopia were given Fresnel prisms to join the double images and where prisms were ineffective, occlusion—either total or sector—was prescribed. Exercises were advocated for con-

vergence weakness, and compensatory head postures were advised to aid gaze disorders. Additionally, advice was given in relation to scanning and tracking strategies and typoscopes to aid reading.

Visual field loss was reported as occurring in 20–57% [3, 4, 7, 12, 28] and was documented in 49% of our patients of which 2.8% had visual field impairment due to a previous stroke or pre-existent ocular pathology. Complete homonymous hemianopia was common and typically associated with lesions in the occipital and parietal lobe areas. There were many other types including quadrantanopia, checkerboard defects, macular sparing hemianopia and spared temporal crescent defects. Patients displayed varying adaptation to their visual field loss. Advice was provided on field awareness, head movements and scanning strategies. 5.2% had adaptation treatment with Peli prisms [29] which involve applying sector Fresnel prisms on the upper and lower sections of one spectacle lens. This has the effect of displacing images from the ‘blind’ visual field across into the seeing side. Patients may then make scanning head and eye movements to the blind side to view objects of interest on that side.

Table 2. Visual impairment and site of stroke

	Site of stroke	Percentage
<i>Eye movement deficit</i>		
Cranial nerve palsy	Posterior fossa: cerebellum, brain stem, thalamus, occipital lobe	9
Saccadic		
Palsy	Cortical: parietal lobe, occipital lobe, lacunar, internal capsule, intraventricular	9
Dysmetria	Cortical: frontal lobe, parietal lobe, occipital lobe, thalamus, lacunar, basal ganglia, periventricular	50.5
	Brain stem, cerebellar	6
Smooth pursuit palsy	Cortical: parietal lobe	3
	Cerebellar	1.5
Gaze palsy	Cortical: frontal lobe, occipital lobe, parietal lobe, lacunar, basal ganglia, periventricular	4
	Brain stem	17
<i>Visual field impairment</i>		
Homonymous hemianopia	Occipital lobe, parietal lobe	56
Quadrantanopic defects	Occipital lobe, parietal lobe	20
Altitudinal, homonymous scotomas	Occipital lobe	6
<i>Perceptual deficit</i>	Cortical: parietal lobe, occipital lobe, temporal lobe, internal capsule, periventricular	100

Treatment for visual field loss is not commonplace, and although advocates promote increased speed of adaptation, there is little objective alteration to the loss [29–32]. However, the increased speed of adaptation may benefit general rehabilitation and further research is required in this area. Visual field loss is associated with a higher risk of falls, and thus, it is important to diagnose visual field impairment for this group of already compromised patients [25].

Visual inattention/neglect constitutes the most commonly recognised perceptual difficulty for vision after stroke. Fourteen per cent of our patients were found to have neglect. Eight per cent of these patients had an associated visual field defect. A small percentage (6.5%) of patients were diagnosed with agnosia, visual hallucinations and depth perception difficulties, amongst others. There is a variable prevalence of neglect reported in the literature ranging from 8 to 82% of right hemispheric stroke [8, 9] and many patients show recovery [2, 33]. Our treatment largely consisted of advice on scanning strategies, use of compensatory head postures and general awareness. We did not use treatment with prisms or occlusion for these patients although there is a small literature on such management options [34–37]. In terms of visual hallucinations and perceptual deficits such as agnosia, alexia, depth interpretation and colour vision disturbance, we feel that these problems are underestimated as detection is largely reliant on clear questioning by health care staff or self-reporting by patients. Patients may not wish to report the presence of hallucinations for fear of their mental state being questioned. We found that recognition of the entity was a considerable relief to the patient.

Summary

Ninety-two per cent of those referred with a suspected visual difficulty were indeed diagnosed with a visual impairment. Thus, stroke patients with visual deficits do require visual evaluation. Visual impairment comprised 68% with eye movement deficits, 49% with visual field impairment,

26% with low vision and 20% with perceptual difficulties—either as a sole impairment or in combination. This is a considerable number but the prevalence of visual impairment in the overall stroke population remains uncertain. All patients were offered specific treatment options or targeted advice. Although patients may not regain normal visual status following a stroke, it is important that they receive full eye assessments to ascertain the full extent of their impairment and offer targeted treatment or advice as appropriate. It is also important that the outcome of eye assessment is relayed to the wider team of health care professionals. Vision is required for many general rehabilitation techniques and being accurately informed of visual impairment will target alternative therapy options for stroke patients to enhance outcome. Orthoptists can also have an important positive effect on patients concerns, explaining why they are experiencing vision problems. Some fear further deterioration is inevitable leading to blindness and they can be reassured that this is not the case.

Key points

- Ninety-two per cent of stroke survivors with a suspected visual difficulty have visual impairment.
- Visual impairment impacts on activities of daily living performance.
- Targeted treatment or advice can be offered.

Conflicts of interest

The authors have no conflicts of interest.

Supplementary data

Supplementary data are available at *Age and Ageing* online.

References

1. National Audit Office. Reducing Brain Damage: Faster Access to Better Stroke Care. London: NAO, 2005.
2. Jones SA, Shinton RA. Improving outcome in stroke patients with visual problems. *Age Ageing* 2006; 35: 560–5.
3. MacIntosh C. Stroke re-visited: visual problems following stroke and their effect on rehabilitation. *Br Orthopt J* 2003; 60: 10–14.
4. Freeman CF, Rudge NB. Cerebrovascular accident and the orthoptist. *Br Orthopt J* 1988; 45: 8–18.
5. Fowler MS, Wade DT, Richardson AJ, Stein JE. Squints and diplopia seen after brain damage. *J Neurol* 1996; 243: 86–90.
6. Kapoor N, Ciuffreda KJ, Han Y. Oculomotor rehabilitation in acquired brain injury: a case series. *Arch Phys Med Rehabil* 2004; 85: 1667–78.
7. Gilhotra JS, Mitchell P, Healey PR, Cumming RG, Currie J. Homonymous visual field defects and stroke in an older population. *Stroke* 2002; 33: 2417–20.
8. Stone SP, Halligan PW, Greenwood RJ. The incidence of neglect phenomena and related disorders in patients with an acute right or left hemisphere stroke. *Age Ageing* 1993; 22: 46–52.
9. Sunderland A, Wade DT, Langton Hewer R. The natural history of visual neglect after stroke. Indications from two methods of assessment. *Int Disabil Stud* 1987; 9: 55–9.
10. Lotery AJ, Wiggam MI, Jackson AJ *et al*. Correctable visual impairment in stroke rehabilitation patients. *Age Ageing* 2000; 29: 221–222.
11. Freeman CF. Collaborative working on a stroke-rehabilitation ward. *Parallel Vis (Br Ir Orthopt Soc)* 2003; 56: 3.
12. Falke P, Abela BM, Krakau CE *et al*. High frequency of asymptomatic visual field defects in subjects with transient ischaemic attacks or minor strokes. *J Intern Med* 1991; 229: 521–5.
13. Dutton GN. Cognitive vision, its disorders and differential diagnosis in adults and children: knowing where and what things are. *Eye* 2003; 17: 289–304.
14. Pollock L. Managing patients with visual symptoms of cerebrovascular disease. *Eye News* 2000; 7: 23–26.
15. Johansen A, White S, Waraisch P. Screening for visual impairment after stroke: validation of the Cardiff Acuity Test. *Arch Gerontol Geriatr* 2003; 36: 289–93.
16. Ramrattan RS, Wolfs RC, Panda-Jonas S *et al*. Prevalence and causes of visual field loss in the elderly and associations with impairment in daily functioning: the Rotterdam Study. *Arch Ophthalmol* 2001; 119: 1788–94.
17. Granger CV, Cotter AC, Hamilton BB, Fiedler RC. Functional assessment scales: a study of persons after stroke. *Arch Phys Med Rehabil* 1993; 74: 133–8.
18. Nelles G, Esser J, Eckstein A, Tiede A, Gerhard H, Diener HC. Compensatory visual field training for patients with hemianopia after stroke. *Neurosci Lett* 2001; 306: 189–92.
19. Tsai SY, Cheng CY, Hsu WM, Su TP, Liu JH, Chou P. Association between visual impairment and depression in the elderly. *J Formos Med Assoc* 2003; 102: 86–90.
20. West CG, Gildengorin G, Haegerstrom-Portney G, Schneck ME, Lott L, Brabyn JA. Is vision function related to physical functional ability in older adults? *J Am Geriatr Soc* 2002; 50: 136–45.
21. DeLuca M, Spinelli D, Zoccolotti P. Eye movement patterns in reading as a function of visual field defects and contrast sensitivity loss. *Cortex* 1996; 32: 491–502.
22. Drivers Medical Group, Driver and Vehicle Licensing Agency. For Medical Practitioners. At a Glance: Guide to the Current Medical Standards of Fitness to Drive. Swansea: Drivers Medical Group, DVLA, 2007.
23. World Health Organisation. <http://www.who.int/blindness/causes/priority/en/index5.html>.
24. Corriveau H, Herbert R, Raiche M, Prince F. Evaluation of postural stability in the elderly with stroke. *Arch Phys Med Rehabil* 2004; 85: 1095–101.
25. Marx MS, Werner P, Cohen-Mansfield J, Feldman R. The relationship between low vision and performances of activities of daily living in nursing home residents. *J Am Geriatr Soc* 1992; 40: 1018–20.
26. Clisby C. Visual assessment of patients with cerebrovascular accident on the elderly care wards. *Br Orthopt J* 1995; 52: 38–40.
27. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME. Vision therapy for oculomotor dysfunction in acquired brain injury: a retrospective analysis. *Optometry* 2008; 79: 18–22.
28. Maulaz AB, Bezerra DC, Bogousslavsky J. Posterior cerebral artery infarction from middle cerebral artery infarction. *Arch Neurol* 2005; 62: 938–941.
29. Peli E. Field expansion for homonymous hemianopia by optically induced peripheral exotropia. *Optom Vis Sci* 2000; 77: 453–64.
30. Reinhard J, Schreiber A, Schiefer U *et al*. Does visual restitution training change absolute homonymous visual field defects? A fundus controlled study. *Br J Ophthalmol* 2005; 89: 30–5.
31. Pambakian AL, Mannan SK, Hodgson TL, Kennard C. Saccadic visual search training: a treatment for patients with homonymous hemianopia. *J Neurol Neurosurg Psychiatry* 2004; 75: 1443–8.
32. Poggel DA, Kasten E, Sabel BA. Attentional cueing improves vision restoration therapy in patients with visual field defects. *Neurology* 2004; 63: 2069–76.
33. Stone SP, Patel P, Greenwood RJ, Halligan PW. Measuring visual neglect in acute stroke and predicting its recovery: the visual neglect recovery index. *J Neurol Neurosurg Psychiatry* 1992; 55: 431–6.
34. Rossetti Y, Rode G, Pisella L *et al*. Prism adaptation to a rightward optical deviation rehabilitates left visuospatial neglect. *Nature*. 1998; 395: 166–169.
35. Frassinetti F, Angeli V, Meneghello F, Avanzi S, Ladavas E. Long lasting amelioration of visuospatial neglect by prism adaptation. *Brain* 2002; 125: 608–623.
36. Rode G, Tossetti Y, Boisson D. Prism adaptation improves representational neglect. *Neuropsychologica* 2001; 39: 1250–1254.
37. Rousseaux M, Bernati T, Saj A, Kozlowski O. Ineffectiveness of prism adaptation on spatial neglect signs. *Stroke* 2006; 37: 542–543.

Received 18 February 2008; accepted in revised form 26 August 2008