VISUAL DYSFUNCTION IS UNDERESTIMATED IN PATIENTS WITH ACQUIRED BRAIN INJURY

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Objectives: More than 50% of human cerebral activity is related to vision. Visual impairments are therefore common after acquired brain injury, although they are often overlooked. In order to evaluate the prevalence of visual deficits in our Out-patient Brain Injury Program, a structured screening questionnaire, the Visual Interview, was administered.

Methods: A total of 170 patients with acquired brain injury, mean age 47 years, who were enrolled in the programme during 2010–12, underwent the Visual Interview. The interview consists of 18 questions concerning visual impairment and was performed on admission. The different types of visual impairment were compared with regard to sex and diagnosis.

Results: Fifty-four percent of the patients reported visual changes, mainly reading difficulties, photophobia and disturbances of the visual field. Sixteen patients who did not experience visual changes also reported visual symptoms in 4–9 questions. Only slight differences were noted in the occurrence of visual symptoms when correlated with sex or diagnosis.

Conclusion: Visual impairments are common after acquired brain injury, but some patients do not define their problems as vision-related. A structured questionnaire, covering the most common visual symptoms, is helpful for the rehabilitation team to facilitate assessment of visual changes.

Key words: stroke; traumatic brain injury; visual disorder; diplopia; hemianopia; photophobia.

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The visual system is widely distributed in the brain. It is integrated in more than 50% of human cerebral activity and is fundamental for interpretation of, and interaction with, the environment (1, 2).

A pyramidal hierarchical model of visual perceptual function was presented by Warren in 1993 (3). In this model, visual cognition forms the top level, followed by, in descending order: visual memory, pattern recognition, scanning, attention and a base level holding acuity, visual fields, and ocular motor control. The model illustrates how higher visual skills evolve from integration and interaction with lower skills and how visual cognition depends on well-functioning lower levels of visual perception.

Base level disturbances, such as visual field defects (VFDs), visual acuity changes, diplopia, strabismus, photophobia and different types of binocular disorders, are common after acquired brain injury (ABI) (4, 5), and lead to chronic headache, fatigue, dizziness, reading problems, and difficulties navigating the environment (6, 7). Although a complete VFD or manifest diplopia seldom escapes notice, disturbances of ocular motor abilities and photophobia are likely to be overlooked. Examinations of convergence and accommodation are not customary in standard ophthalmological assessments. Ordinary short examinations are unable to reveal declining attention ability and fatigue. Thus, the true problems may remain hidden.

Several reports of prevalence and quality of visual deficits after ABI document visual dysfunctions in approximately 50–75% of patients (8–13). The occurrence of different visual symptoms differs between the studies, including reading disturbances, VFD, diplopia, ocular motor dysfunction and photophobia. Nevertheless, visual symptoms are often overlooked in neurorehabilitation. The observations of Sand et al. (14) are noteworthy, i.e. that 1 of 4 stroke patients with VFD, 6 months after onset of stroke, considered that their visual problems reduced quality of life and increased their disability.

Visual disturbances after ABI are common and lead to reduced quality of life. An important question is why they are so often overlooked in neurorehabilitation? A possible explanation is the difficulty for different professionals to co-operate. Vision disturbances are complex and many different professionals operate in the field. An ability to co-operate is needed for a high-quality assessment. Another explanation could be the patients’ difficulty describing their shortcomings. They experience decreased reading speed, fatigue and dizziness, but do not recognize these problems as expressions of visual deficits. A structured questionnaire admission would help the clinician to obtain informative answers.

In 1990, Kerkhoff et al (15), compiled an “Interview Questionnaire” in order to capture visual disorders after ABI. This interview was used by Wilhelmson 2003 (12). Jacobsson & Hamelius translated it from Norwegian to
Swedish in 2010 (16). During the last 5 years we have used this questionnaire, slightly modified, termed the Vision Interview (TVI), as an aid to discover visual deficits in our Out-patient Brain Injury Program.

The aim of the present study was to examine and analyse the occurrence of self-reported visual changes in a Swedish out-patient group with medium to severe ABI, based on TVI.

**MATERIAL AND METHODS**

A total of 196 patients were admitted to the Out-patient Brain Injury Rehabilitation Unit at the Department of Rehabilitation Medicine, Danderyd University Hospital Stockholm, Sweden, during the period 1 September 2010 to 30 June 2012. The unit offers team-based assessment of brain injury and rehabilitation of physical, cognitive and emotional deficits due to ABI. The patients (18–68 years of age) entered the programme approximately 3–12 months after the onset of injury/illness (Table I). The severity of injury is given in Table II according to the Glasgow Outcome Scale (GOSE) (17). The level of severity in the study group was GOSE 4–7 (Table II).

Twenty-six of the 196 admitted patients had to be excluded due to: severe aphasia (n = 1); interview missed at admission (n = 14); symptoms not primarily caused by ABI (n = 4); entered the rehabilitation programme directly based on an assessment elsewhere (n = 3); and interview interrupted or refused by patient (n = 4). The remaining 170 patients, mean age 47 years, were classified according to diagnoses, sex, and age, and included in the study (Table III).

Two diagnoses dominated: stroke and TBI (Table III). The number of patients with diagnoses of subarachnoid haemorrhage (SAH), infections in the brain, benign brain tumour, anoxia caused by heart disease and a group of occasional diagnoses (= Other) was small (Table III). Due to the difference in number between the diagnoses, analyses of differences between all diagnoses were not operational. The stroke group and TBI group were the only ones analysed statistically in relation to one another.

At admission each patient underwent TVI, which took approximately 10 min and was administered by the physician. TVI includes 18 questions dealing with visual changes after injury/illness (Table IV). It contains a primary question: “Have you noticed any type of vision changes?” (Q1), and 16 questions sorted according to disturbances of function (Q2, 8–17), disturbances of activity (Q4–Q7), and a final question: “Have you controlled your eyesight after you fell ill?” (Q18).

**RESULTS**

The TVI findings are summarized in Table IV. In addition, all positive answers are given both for the group of patients who did notice visual changes and for the group who did not (Fig. 1).

**Table I.** Time after onset of injury/illness at admission

| Time after onset of injury/illness | Patients, n (%) |
|-----------------------------------|-----------------|
| 1–3 months                        | 46 (27)         |
| 4–6 months                        | 64 (38)         |
| 6–12 months                       | 34 (20)         |
| 13–18 months                      | 13 (8)          |
| >18 months                        | 12 (7)          |

**Table II.** Degree of difficulty of injury/illness according to the Glasgow Outcome Scale (GOSE)

| GOSE | Patients, n (%) |
|------|----------------|
| 4    | 10 (6)         |
| 5    | 89 (53)        |
| 6    | 62 (37)        |
| 7    | 6 (4)          |

1 = dead, 2 = vegetative state, 3 = lower severe disability, 4 = upper severe disability, 5 = lower moderate disability, 6 = upper moderate disability, 7 = lower good recovery, 8 = upper good recovery.

**Table III.** Diagnoses by sex and age groups

| Age group, years | Sex | Stroke | Men | Women | TBI | Men | Women | SAH | Men | Women | Infection | Men | Women | Anoxia | Men | Women | Other | Other |
|------------------|-----|--------|-----|-------|-----|-----|-------|-----|-----|-------|-----------|-----|-------|--------|-----|-------|-------|-------|
| 18–35            |     | 77     | 27  | 50    | 9   | 35  | 33    | 10  | 14  | 10    | 9          | 9   | 6     | 2      | 2   |       |       |
| 36–55            |     | 37     | 15  | 22    | 13  | 14  | 10    | 9   | 6   | 6     | 9          | 9   | 6     | 2      | 2   |       |       |
| 55–68            |     | 15     | 6   | 6     | 9   | 6   | 6     |     |     |       | 10         |     |       | 2      |     |       |       |

*Other: different surgical interventions in the brain (n=6), post-radiation of tumour (1), epilepsy (1), multi-organ failure (1), NMDA encephalitis (1), sinus thrombosis (1), a. vertebral dissection (1), late effects of intracerebral haemorrhage (1); TBI: traumatic brain injury; SAH: subarachnoid haemorrhage.

**Table IV.** Answers from the Visual Interview (n = 170)

| Self-reported symptoms after injury/illness | Yes | n (%) |
|-------------------------------------------|-----|-------|
| 1 Have you noticed any type of vision changes? | 91  | (54)  |
| 2 Do you suffer from double vision?        | 33  | (19)  |
| 3 Do you have problems while reading?      | 90  | (53)  |
| 4 Do people and objects suddenly appear before you in an unexpected way? | 41  | (24)  |
| 5 Do you crash into people and objects when you are on the move? | 52  | (31)  |
| 6 Do you find it difficult to estimate depths or heights in a stairway? | 19  | (11)  |
| 7 Do you find it difficult to grasp a glass, a door handle or shake hands? | 33  | (19)  |
| 8 Do you find it difficult to recognize faces? | 19  | (11)  |
| 9 Do you interpret familiar faces in a way that differs from before? | 15  | (9)   |
| 10 Does light blind you more than before?  | 60  | (35)  |
| 11 Do you need stronger light now than before in order to obtain distinct vision? | 43  | (25)  |
| 12 Do you need stronger light now while reading? | 63  | (37)  |
| 13 Is your vision more blurred than before? | 60  | (35)  |
| 14 Have you experienced that colours have changed? | 3   | (2)   |
| 15 Have you experienced any sight phenomena? | 36  | (21)  |
| 16 Have you had any other unexpected sight experiences? | 23  | (14)  |
| 17 Are there areas of reduced sight in your visual field? | 46  | (27)  |
| 18 Have you eyesight been examined after you fell ill? | 83  | (49)  |

(by an ophthalmologist, visual therapist or optometrist)

**Statistical methods**

The subjectively experienced visual changes are presented as individual values as well as percentages. Non-parametric statistics were used to evaluate differences between groups: Significance levels of \( p < 0.05 \) were accepted. The statistical package SPSS, version 22, was used.

**Ethical considerations**

The study was approved by the regional ethics board of Stockholm, Sweden, (Reg. no. 2013/157-31/3), according to the principles of the Declaration of Helsinki 1978.
According to their answers to Q1, 91 patients (54%) had experienced visual changes after injury/illness and 79 (46%) did not notice any visual changes. It should be strongly emphasized that, in the latter group, the patients who reported no visual changes, 53 individuals gave positive answers to from 1 up to 9 questions, among those given in the interval Q2–17 (Fig. 2).

The combination of such disturbing visual symptoms as diplopia (Q2), photophobia (Q10), blurred vision (Q13), and VFD (Q17) with altered activity levels (Q4–7) and reading disturbances (Q3) can be summarized as follows.

**Diplopia.** Q2: 33 patients experienced diplopia. Of these, 18 did not report any of the difficulties described in the activity questions, 15 (45%) answered “yes” to at least one of the activity questions and 7 were positive to 3 or 4. Twenty-six patients (76%) reported reading difficulties and 20 (61%) had been examined by an ophthalmologist. Two of the latter patients had been referred to an orthoptist before they were admitted to our programme.

**Photophobia.** Q10: 60 patients reported experiencing photophobia. Of these, 22 did not report any of the difficulties described in the activity questions, 38 (63%) answered “yes” to at least 1 of the activity questions and 12 were positive to 3 or 4. Forty-six patients (77%) reported reading difficulties and 36 (60%) had been examined by an ophthalmologist.

**Blurred vision.** Q13: 60 patients experienced blurred vision. Of these 29 did not report any of the difficulties described in the activity questions, 31 (52%) answered “yes” to at least 1 of the activity questions and 12 were positive to 3 or 4. Forty-three (72%) had reading difficulties and 25 (42%) had been examined by an ophthalmologist.

**Visual field defect.** Q17: 46 patients reported a VFD. Of these 16 did not report any of the difficulties described in the activity questions, 30 (65%) answered “yes” to at least 1 of the activity questions and 9 were positive to 3 or 4. Thirty-five patients (76%) experienced reading difficulties and 33 (72%) had been examined by an ophthalmologist. Eleven patients who answered...
“yes” to Q17 also answered “yes” to either Q2, Q10 or Q13, 16 patients answered “yes” to 2 of the these questions and 4 answered “yes” to all 3 questions. Fifteen patients did not answer “yes” to any of these questions.

An examination by an ophthalmologist had been performed after the onset of illness (Q18) in 54 (59%) patients from the group that experienced visual changes and in 31(39%) from the group that denied visual changes.

Two symptoms differed between sex: women suffered more often from photophobia \( (p<0.002 \text{ Fisher’s Exact Test}) \), and experienced more sight phenomena, \( (p<0.001 \text{ Fisher’s Exact Test}) \).

Two symptoms differed between the Stroke and the TBI groups: TBI patients suffered more often from photophobia \( (p<0.004, \text{Pearson } \chi^2) \), and Stroke patients had fewer problems with sudden appearance of objects \( (p<0.012, \text{Pearson } \chi^2) \).

### DISCUSSION

In agreement with previous studies (8–13) approximately half of our ABI patients had noticed changes in their visual capabilities after injury/illness (“yes” in Q1). Diplopia, visual field defects, photophobia, other light-dependent changes and blurred vision were commonly reported disturbances and recognized as vision related. On the other hand, there were many patients in the study group who denied visual changes (“no” in Q1), but who reported the symptoms listed above (Figs 1 and 2). These patients did not recognize their experiences as vision related per se. This strongly supports the need for a screening method applied to all patients after ABI and that the question “Have you noticed any visual change?” is too blunt and should be followed by more detailed questions concerning different visual changes. This also indicates that the estimate that half of ABI patients experience visual changes is probably too low.

There were only a few differences in the frequencies of symptoms between women and men and between patients with diagnoses of stroke and those with TBI, underlining the fact that visual networks are widely distributed in the brain and easily damaged, independent of the type of injury.

The following comments can be made about the more frequently reported symptoms.

### Diplopia (Q2)

The difference between the number of patients with experienced diplopia who were examined by an ophthalmologist (61%; 20/33), and those referred to an orthoptist was notable in 2 patients of these 20. In Sweden only an ophthalmologist is allowed to refer a patient to an orthoptist. This highlights the need for better referrals and better communication with ophthalmologists and for the possibility for the rehabilitation team to refer patients to an orthoptist.

### Reading disturbances (Q3)

Approximately half of the patients reported reading disturbances. In combination with diplopia, photophobia, blurred vision or VFD, the frequency was even higher (72–79%). Reading is a basic requirement for work and social communication. Impaired reading ability may lead to reduced social interaction and unemployment. Reading disturbances can be caused by visual dysfunction, but also by higher cognitive and/or linguistic defects. A weakness of the TVI is the lack of discrimination between different aspects of reading dysfunction. This could explain why 23 patients experienced reading difficulties but no visual changes.

In a study with a cross-over design Schuett et al. (18) compared the therapeutic effects of compensatory oculomotor reading and visual-exploration training in 36 patients with homonymous visual field loss. The aim was to evaluate whether training of reading skills could increase visual exploration or vice versa. The study showed that the training after VFDs had to be highly specific for each type of exercise, and reading must therefore be practised by reading.

There are several rehabilitation methods dealing with reading problems in patients with VFD or binocular difficulties (19, 20). The high occurrence of reported reading disturbances, both in our study and others, is alarming. The high priority of reading skills in rehabilitation after ABI must be emphasized.

### Activity levels (Q4–7)

The positive answers to the 4 questions about visual difficulties correlated with movements, ranged from 11% to 31%. Of the patients who experienced diplopia, photophobia, blurred vision or VFD, 45–65% answered “yes” to at least one of these questions.

Vision is highly integrated in movement. This rather low frequency of positive answers probably shows that these questions do not cover the movement disturbances as carefully as they should.

### Photophobia (Q10)

More than one-third of the patients experienced photophobia, which is a painful reaction to light and interferes with daily life. Photophobia interferes with reading, watching television and makes work in front of a computer screen tiresome and demanding (21).
The results of this study confirm a connection between reading problems and photophobia.

Photophobia due to ABI is probably the effect of a cortical hypersensitivity. Noseda et al. (22) found that exacerbation of migraine headaches by light is driven by photic signals transmitted from the retina to posterior thalamic nuclei. These nuclei process nociceptive signals from the meninges. Noseda et al. (22) identified dura-sensitive neurones in the posterior thalamus, which activity was distinctly modulated by light. Posterior thalamic nuclei project to somatosensory, visual and associative parts of the cortex. These findings suggest a connection between photophobia and injury to the meninges. In the present study the patients in the TBI group suffered more from photophobia than did the stroke patients. An ischaemic stroke involves focal damage to the brain tissue and more seldom interferes with the meninges. TBI, on the other hand, as well as SAH and encephalitis, commonly involve the meninges. This may be an explanation for the development of photophobia in patients with TBI.

**Blurred vision (Q13)**

One-third of patients experienced blurred vision, which can be caused by defect acuity, unstable fixation, and dysfunction of accommodation and convergence. Optometry offers several methods for binocular therapy (23, 24). Ciuffreda et al. (25) showed complete or marked reduction in oculomotor-based symptoms, and improvement in related clinical signs due to optometric visual therapy for 40 patients with mild TBI. The effect of the treatment was stable during the 2–3-month follow-up.

**Visual field disorder (Q17)**

One-third of the patients reported VFD. There are several methods for visual field rehabilitation: restitution, compensation and substitution. A Cochrane review in 2011 (26) concluded that there was no grade 1 evidence for any form of rehabilitation of VFD. However, there were indications that compensatory treatment helped the patients to deal with their environment.

Rowe et al. examined 479 patients with VFD. In almost half of the patients the VFD coincided with other visual disturbances. Our study showed that 67% (31/46) of the patients experiencing VFD had also noticed diplopia, blurred vision or photophobia. In VFD rehabilitation one should be aware of the risk for this combination of symptoms and, if needed, direct the rehabilitation not only to compensation techniques.

Today, computer programs are used in many different brain rehabilitation settings, including home-based visual exploration training. In a review of evidence-based methods in cognitive rehabilitation Cicerone et al. (28) pointed out that computerized training of cognitive deficits without contact with a therapist is not recommended. It is possible that this conclusion is also relevant to computerized home-based visual exploration training. Feedback methodology is central in evidence-based neurorehabilitation. In future the problem of therapeutic guidance may be solved by interactive programmes. After all, the world is three-dimensional and eye movements have to be trained in reality.

**Ophthalmologist assessment (Q18)**

Half of the patients had had an ophthalmological assessment before entering the brain injury programme, particularly those with VFD (72%). Otherwise it is difficult to see a pattern in the referrals; at least it is difficult to connect patients’ symptoms with a referral. Sand et al. (29) showed in a Norwegian study only 9% of their patients with VFD were referred to perimetry, and 8% to vision rehabilitation. These results indicate a lack of connection between the eye specialists, neuro-rehabilitation and acute care units.

**Conclusion**

The aim of this study was to highlight the vision system and its vulnerability to injury after ABI, and to find a structured and easy method for scanning visual deficits within the speciality of neuro-rehabilitation. A weakness of the present study is that it is limited to self-reported symptoms of visual impairments. The true relationship between subjective reports, as collected by TVI, and objective findings are not known. A new study relating TVI to objective measurements of visual and binocular function is in progress.

Rowe et al. (30), after interviewing personnel working in different units for integrated care of vision/stroke patients, listed key elements in order to obtain high-quality stroke/vision services. Among these key elements were standardized screening/referral forms, an orthoptist as a core member of the team, formal support from stroke physician and a vision care pathway. A screening instrument, such as the TVI, could be the first step in this chain and a tool that is easy to use. The visual aspects of ABI are now frequently discussed in our team and integrated into rehabilitation plans. TVI has made it easier to provide specific information during referrals and thereby facilitates the necessary cooperation with the ophthalmologist. An important step was to integrate a person with visual-therapy competence into our rehabilitation team (30). As a
result, the rehabilitation assessments and vision-related rehabilitation have improved, although much work remains to be done to fulfill the goal of high-quality vision care for patients with ABI. In order to achieve this, a creative link is required between neurorehabilitation, optometry and ophthalmology.

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REFERENCES

1. Fellman DJ, Van Essen DC. Distributed hierarchal processing in the primate cortex. Cereb Cortex 1991; 1: 1–47.
2. Rizzolatti G, Sinigaglia C. Mirrors in the brain – how our minds share actions and emotions. Oxford: Oxford University Press; 2008.
3. Warren M. A Hierarchical model for evaluation and treatment of visual perceptual dysfunction in adult acquired brain injury, part 1. Am J Occup Ther 1993; 47: 42–54.
4. Rowe F, Brand D, Jackson C, Price A, Walker L, Harrison S, et al. Visual impairment following stroke: do stroke patients require vision assessment? Age Ageing 2009; 38: 188–193.
5. Greenland BD, Kapoor N, Singh AD. Visual impairment in the first year after traumatic brain injury. Brain Inj 2012; 26: 1338–1359.
6. Warren M. Pilot study on activities of daily limitations in adults with hemianopia. Am J Occup Ther 2009; 63: 626–633.
7. Wolter M, Preda S. Visual deficits following stroke: maximizing participation in rehabilitation. Stroke Rehabil 2006; 13: 12–21.
8. Steilmaack JA, Frith T, Van Koeveering D, Rinne S, Stelmaack TR. Visual function in patients followed at a Veterans Affairs polytrauma network site: an electronic medical record review. J Am Optom Assoc 2009; 80: 419–424.
9. Bulson R, Jun W, Hayes J. Visual symptomatology and referral patterns for Operation Iraqi Freedom and Operation Enduring Freedom veterans with traumatic brain injury. J Rehabil Res Dev 2012; 49: 1075–1082.
10. Brahman KD, Wildengur, H M, Kirby J, Ingalla S, Chang C-Y, Goodrich G. Visual impairment and dysfunction in combat-injured service members with traumatic brain injury. Optom Vis Sci 2009; 86: 817–824.
11. Goodrich G, Flyg HM, Kirby JE, Chang C-Y, Martinssen GL. Mechanisms of TBI and visual consequences in military and veteran populations. Optom Vis Sci 2013; 90: 105–112.
12. Wilhelmsen GB. [Seeing is not always enough.] Oslo: Unipub Forlag; 2003 (in Norwegian).
13. Siong KH, Woo GC, Chan D, Chan K, Li LS, Cheung HK, et.al. Prevalence of visual problems among stroke survivors in Hong Kong. Clin Exp Optom 2014; 97: 433–441.
14. Sand KM, Wilhelmsen G, Naess H, Midelfart A, Thomassen L, Hoff JM. Vision problems in ischaemic stroke patients: effects on life quality and disability. Eur J Neuro 2016; 23: Suppl 1, 1–7.
15. Kerckhoff G, Schaub J, Zihl J. Die Anamnese zerebral betingter Sehestörungen. Der Nervenzt 1990; 61: 711–718.
16. Jakobsson M, Hamelius L. [Mapping of self-reported visual disturbances and their effect on activity after acquired brain injury.] Examination essay. Karolinska Institutet, Department of Neuropsychology, Section Occupational Therapy; 2010 (in Swedish).
17. Teasdale GM, Pettigrew LE, Wilson JT, Murray G, Jenett B. Analyzing outcome of treatment of severe head injury: a review and update on advancing the use of the Glasgow Outcome Scale. J Neurotrauma 1998; 15: 587–597.
18. Schuett S, Heywood CA, Kentridge RW, Dauner R, Zihl J. Rehabilitation of reading and visual exploration in visual field disorders: transfer or specificity? Brain 2012; 135: 912–921.
19. Schuett S. The rehabilitation of hemianopic dyslexia. Nat Rev Neurol 2009; 5: 427–437.
20. Han Y, Ciuiffreda K, Kapoor N. Reading-related oculomotor testing and training protocols for acquired brain injury in humans. Brain Res Protoc 2004; 14: 1–12.
21. Stern CD. Photophobia, light, and colour in acquired brain injury. In: Suter PS, Harvey LH, editors. Vision rehabilitation: multidisciplinary care of the patient following brain injury. Boca Raton: CRC Press; 2011, p. 283–285.
22. Noseda R, Kainz V, Jakubowski M, Gooley J, Saper C, Digre K, et al. A neural mechanism for exacerbation of headache by light. Nat Neurosci 2010; 13: 239–245.
23. Ciuffreda K, Ludlam D. Conceptual model of optometric vision care in mild traumatic brain injury. J Behav Optom 2011; 22: 10–12.
24. Valent C. Rehabilitation of Persons with Visual Sequelae Resulting from Traumatic Brain Injury. Journal of Optometric Vision Development 2003; 34: 105–110.
25. Ciuffreda K, Rutner D, Kapoor N, Suchoff I, Craig S, Han M. Vision therapy for oculomotor dysfunction in acquired brain injury: a retrospective analysis. Optometry 2008; 79: 18–22.
26. Pollock A, Hazleton C, Henderson CA, Angilley J, Dhillon B, Langhorne P, et al. Intervention for visual field defects in patients with stroke. The Cochran Library 2011. Available from: http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD008388.pub2/full.
27. Rowe F, Wright D, Brand D, Jackson C, Harrison S, Maan T, et al. A prospective profile of visual field loss following stroke: prevalence, type, rehabilitation, and outcome. BioMed Research International 2013, Article ID 719096.
28. Cicerone K, Langenbahn D, Braden C, Malec J, Kalmar K, Fraas M, et al. Evidence-based cognitive rehabilitation: update review of the literature from 2003 through 2008. Arch Phys Med Rehabil 2011; 92: 519–530.
29. Sand K, Thomassen L, Nassa H, Radahl E, Hoff JM. Diagnosis and rehabilitation of visual field defects in stroke patients: a retrospective audit. Cerebrovasc Dis Extra 2012; 2: 17–23.
30. Rowe F, Walker M, Rockliffe J, Pollock A, Noonan C, Howard C, et al. Delivery of high quality stroke and vision care: experiences of UK services. Disabil Rehabil 2015; 2: 1–5.