Brain injury can result from a number of causes which in turn can lead to neurological dysfunction including dysfunctions in the visual system; acquired brain injury (ABI) is an umbrella term for patients who have such conditions. A greater proportion of patients who have ABI are seen in optometric practice than one might imagine. Indeed, anyone who has had a stroke, a penetrating or closed brain injury, brain surgery, those with congenital arteriovenous malformations, and those with congenital neurodevelopmental conditions such as cerebral palsy (CP), Down’s syndrome (DS), Parkinson’s disease, and Rett’s syndrome, are included in this group. This first article of a series provides an overview of neuro-optometric rehabilitation in terms of how ABI affects patients and its relevance to the eye examination.

Learning objectives
Be able to communicate effectively with other appropriate people involved with a patient’s care (Group 1, 1.2.5)
Be able to understand the special examination needs of patients with learning and other disabilities (Group 7, 7.1.6)

About the author
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Epidemiology

Patients with ABI tend to broadly fall into two age groups. Older patients are those who have usually experienced stroke or have had a brain injury following an accident. In the UK, it is estimated that 110,000 people suffer a stroke per year. This equates to 104 cases per 100,000 of the population, rising to 109 cases per 100,000 of the population, for patients aged 80 years and over.

Younger patients include military personnel who have experienced injuries in combat, paediatric patients with underlying congenital conditions, and patients from secondary school age upwards who have experienced a sport-related head injury. A study in the US estimated that as many as 8.9% of all high school athletes have sustained one or more head injuries so potentially have mild ABI. It is interesting to note that girls are reported to have a higher rate of concussion than boys in similar sports, perhaps because female athletes have weaker neck muscles and a smaller head mass than their male counterparts. However, male athletes are more likely to be reluctant to report any head injury because they want to continue playing. For this reason, the incidence of head injury in boys playing sport may be underestimated.

As regards popular recreational sports, head injuries during snowboarding were a frequently reported injury (19.1%). In a Norwegian study of male footballers, head injuries formed 6% of acute injuries, the most common cause being the elbow/arm/hand of one player coming into contact with the head of another player, particularly when players go for headers.

Penetrating or closed brain injuries are classified as traumatic brain injuries (TBI). TBI can occur due to blast injuries in the case of military personnel and due to head injuries in those playing sport, those involved in car accidents or industrial accidents, those who have been attacked with sharp instruments or those who have received a blow to the head. The highest incidence of TBI is in the 15-24 year age range with a greater incidence in the male population.

Many of these patients have subtle visual problems which can be diagnosed and remediated by optometrists in primary care practice. Often such visual problems have been overlooked upon the patient’s admission into hospital in light of other more pressing medical concerns such as broken limbs, blood loss, skull fracture and impaired breathing. Such patients may not have had a thorough visual assessment so visual anomalies have not been detected before discharge.

Mechanism of TBI

The mechanics of TBI follow that of coup contrecoup injury. Here there are two types of injury. The first injury is the coup or ‘acceleration’ injury, whereby an object in motion strikes the head, or when the head in motion strikes a stationary hard object. This coup injury can result in fracturing of the skull, subdural and epidural haematomas and contusion of the underlying brain tissue. The second injury is the contrecoup or ‘deceleration’ injury, which occurs after the coup injury whereby the brain is forced to move in the opposite direction to which it started; it decelerates as the head suddenly stops, causing impact on the skull (Figure 1). In such cases, the acceleration and deceleration forces cause the brain to move rapidly back and forth within the skull, resulting in injuries at the point of impact and at the area opposite the point of impact. In the case of an impact to the front of the head, this would mean that the occipital and frontal lobes would be damaged.

The rapid pressure changes induced by the swift movement of the head and subsequent swift movement of the brain within the skull causes compression of brain tissue, as well as shearing and tearing injuries due to rotational forces acting on the neurones. While linear acceleration is believed to produce superficial brain damage (such as to grey matter) resulting in contusions and haematomas in the epidural or subdural layers, it is the rotational forces that are believed to cause deeper cerebral disruption leading to diffuse axonal injuries (DAI). Brain tissue has a consistency that is close to that of a blancmange, and it is this soft tissue that is subject to shearing forces. Additional damage to tissue can occur as the brain slides along the rough inner surfaces of the skull, particularly in the temporal and frontal regions of the brain and around the brain stem.

The cranial nerves involved in vision can also be affected and a study of post-TBI ophthalmoplegia found that 39% of patients had 4th nerve (trochlear) palsies, 33% had 3rd nerve (oculomotor) palsies, 14% had 6th nerve (abducens) palsies, 10% had combined palsies and 4% had restrictive ophthalmopley. However, some of the other neurones subjected to these shearing, tearing and compression forces in TBI are communicating fibres, so damage to these will affect the ability of different areas of the brain to communicate with each other. This can potentially affect all areas of brain function including the workings of the visual system.

Clinical implications of ABI in the eye examination

As the communicating nerve fibres do not work as effectively, communication between different areas of the brain is slower. This can often mean that the patient takes longer to: respond to questions, learn new skills, take in new information and solve new problems.

Most people with ABI notice cognitive impairments that can last for three months...

Figure 1 Coup (blue)-contrecoup (red) injury. See text for details
post-injury and in 25-30% of cases, these cognitive impairments can last for over a year. Such problems with executive functioning are signs of frontal lobe injury and will have a significant impact on re-entry into the workplace. For similar reasons, patients with ABI also often show ‘impaired simultaneous processing’ resulting in slow processing of information including speech (and corresponding auditory) and visual. Consequently, in the context of an eye examination, such patients can find it extremely challenging to attend to visual information, as well as to listen to practitioners talk or give instructions and advice at the same time. Naturally, this will affect subjective testing performed during the eye examination (for example taking visual acuities, cover testing, ocular motility testing, measurement of accommodation, measurement of convergence, subjective examination, colour vision assessment, assessment of stereopsis) as well as the patient’s ability to cooperate during ancillary testing such as visual field assessment. In particular with the latter, the patient’s reaction time is slower and so any estimation of their visual field will be smaller than their actual visual field. For these reasons, it is important that the optometrist and practice staff are aware of the cognitive issues which affect the patient.

Difficult communication
Patients with ABI tend to have self-awareness deficits and consequently are not necessarily aware of any problems with their vision and it is only by asking highly specific questions that they become aware that they have a visual problem. This is often because they have become so accustomed to their present state of living that they are not able to remember what life was like prior to their injury. They are not able to remember what normality was like.

Practitioners might find that during the course of history and symptoms and the examination itself (for example cross-cyl. examination and sphere refinement), patients with ABI will agree with whatever the practitioner says, as they want to keep any conversation brief in an attempt to get through the examination as quickly as possible. Indeed, such patients will calculate that they will get tired, especially as answering lots of questions can be challenging, and this takes up more energy than normal.

Studies have shown that patients with learning disabilities such as DS tend to have transient memory of language so tend to agree with the last thing you have said. As such, considering the example of cross-cyl. examination, if practitioners only present ‘option 1 or option 2’, they might find that the patient consistently selects option 2 every time. Practitioners should therefore consider changing their presentation technique where appropriate, as well as taking into consideration the results of objective tests, such as retinoscopy, in order to prescribe the right refractive correction. In the author’s experience however, relying on autorefraction results with such patients does not necessarily give an accurate refraction. If you normally rely on an autorefractor in practice, you would be better advised to compare your autorefraction result with that of your near and distance retinoscopy findings before prescribing.

Otherwise you may encounter issues with non-tolerance with the refractive correction prescribed.

The key point to remember is that many of these patients with ABI have all of the above cognitive issues and in recognition of this, it is recommended that such patients are treated with patience and sensitivity.

Visual issues in ABI
Of people with ABI, 70-80% will have visual issues and broadly speaking these concern ocular health, refraction, binocular vision and visual fields. As the brain is effectively ‘shaken’ in ABI, the neurology of the entire brain, as well as the integrity of the eye, can be affected.

Dry eye
Dry eye is a very common symptom in ABI. Upon careful questioning, patients will often admit to experiencing redness, soreness and grittiness of the eyes as well as a burning and/or watery sensation. Disruption of the tear film can lead to intermittent blurry vision...
and ghosting of images too. These changes typically arise because the brain is working more slowly, leading to a reduced blink rate and reduced corneal sensitivity, which increases exposure of the ocular surface and contributing to dry eye symptoms. Associated with such changes, there is an increased risk of corneal abrasion, blepharitis, chalazion and hordeolum in TBI, while subconjunctival haemorrhage and ptosis are more prevalent in CVA.23

**Photophobia**

Pupils are commonly enlarged in ABI and because of slower brain function are also sluggish in reaction. Pupils may also be unequal in size (anisocoria). This can lead to symptoms of photophobia and blurred vision, which can in turn exacerbate dry eye symptoms. Provision of highly specific tints can be helpful in this respect.24

**Ocular trauma**

Trauma to the eye can result in a permanently enlarged pupil and/or traumatic cataract and/or hyphema (bleeding in the anterior chamber). A blow to the eye can also lead to retinal haemorrhage, retinal swelling (commotio retinae), and retinal detachment. Any blurry vision associated with retinal swelling or retinal detachment may be permanent.

**Papilloedema**

Optic nerve swelling or papilloedema can occur if intracranial pressure is raised following a brain injury (Figure 2). The optic nerves become inflamed as intracranial pressure is transmitted and this swelling can result in optic atrophy and optic neuritis with reduced vision, reduced contrast sensitivity, reduced colour vision, glare sensitivity and visual field loss.

**Binocular vision anomalies in ABI**

ABI commonly affects the binocular vision system. Most of the current research into this is being conducted by professor Ken Ciuffreda and his research team at the optometry department at the State University of New York (SUNY). They hypothesise that in ABI the nerve fibres become stretched, attenuated and bruised. Even though the nerve fibres appear intact on CT and MRI scans, the nerve fibres do not function as well as before.25

The neurology that serves blink rate, accommodation, convergence, fusion and eye movements may still work, though at only a fraction of the efficiency that it did before. As an analogy, the effect is akin to having a computer where the hardware is intact but the software is not working effectively.

**Accommodative anomalies**

Patients with ABI might present with accommodative insufficiency, accommodative fatigue, accommodative lag and accommodative infacility. Optometric intervention includes provision of appropriate lenses as well as accommodation training where appropriate.26

**Fusional anomalies**

In terms of convergence and fusional abilities, a patient with ABI might present with decompensated heterophoria, convergence insufficiency,27 reduced fusional ranges,
reduced stereopsis, fixation disparity and binocular instability. Optometric intervention includes provision of an appropriate spectacle correction including the use of ground prism or Fresnel prisms and training of convergence and divergence where appropriate.

Oculomotor control
Impaired control of extraocular muscles can lead to painful eye movements and if the nerves controlling the extraocular muscles are affected this can lead to muscle paresis and diplopia. Optometric intervention includes provision of sectorial patching and use of Fresnel prisms, as appropriate, in the affected position of gaze.

Visual field anomalies
Damage to the visual pathway can lead to visual field defects. One study found that one in seven cases of patients with severe TBI presented with bilateral visual field defects.28 Field loss usually correlates well with the position of the injury in relation to the visual pathway. This can include unilateral optic nerve lesions, paracentral scotomas, congruous and incongruous homonymous hemianopias and quadrantanopias, as well as altitudinal defects.29 It is beyond the scope of this article to describe every type of defect, but an effective summary of the types of field defects caused according to location of the lesion is shown in Figure 3. Where appropriate, patients can have prismatic devices fitted to their spectacles in order to help compensate for their visual field loss, as well as being given counselling as to how they can alter their behaviour in order to compensate for their field loss.

Activities for daily living (ADLs)
The visual sequelae of ABI is likely to affect many activities of daily living (ADLs), in particular driving, reading, the ability to watch the television, using a computer and the ability to walk. The ability to judge the distance of different objects in their environment might also be affected so that people bump into things, knock things over or when pouring tea miss the cup. Their ability to pay attention to, and remember, detail or recall of events can also all be affected.

All of these factors will affect the patient’s quality of life as well as their ability to care for themselves. These visual sequelae will therefore need to be considered as this can slow down the speed of their rehabilitation.29

Model of neuro-optometric rehabilitative care
One of the main principles of this model of optometric neuro-rehabilitation is that any care focuses on the patient. There is a multi-disciplinary approach to the rehabilitation of patients30 and therefore practitioners must ensure the highest levels of communication to other health professionals when dealing with the patient.

In primary care optometry, it is best practice to write to the patient’s GP summarising your findings. Advise the GP to pass on your findings to other health professionals dealing with the patient, in particular any other eye care practitioners, physiotherapists and occupational therapists. The work done by physiotherapists, occupational therapists, carers or support workers becomes easier with appropriate optometric intervention. Appropriate optometric intervention will allow the patient to attend to ADLs for longer than before and will allow them to become more capable. This in turn will speed up their rehabilitation and, in some cases, hopefully will allow a return to work. Where it is not possible for the patient to return to work, optometric intervention often at least improves their quality of life by facilitating ADLs, so that they are able to accomplish more than they could before.

Conclusion
This article has described the effects of ABI on the visual system and the key considerations for how this can affect patients in terms of performance in an eye examination as well as their daily life. The next article in this series will describe in detail the optometric approach to examining patients with ABI, in order to identify any dysfunctions in the visual system. The final article in this series will put this all together to discuss how patients with ABI can be managed, to improve visual function and quality of life.

MORE INFORMATION
References Visit www.optometry.co.uk/clinical, click on the article title and then on ‘references’ to download.

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