Visual restoration therapy

Disappointing results from Nova Vision’s visual restoration therapy
J C Horton

We should refocus our search for new treatments in a more fruitful direction

Lesions posterior to the optic chiasm produce homonymous visual field loss—overlapping scotomas in the nasal field of one eye and the temporal field of the other eye. Patients retain normal acuity, but find their lives changed forever. A homonymous hemianopia, when complete, makes safe driving impossible and reading a chore. Although some patients experience partial, spontaneous improvement following the acute phase of an event, most remain handicapped by permanent field loss. No treatment was available before the recent advent of visual restoration therapy.

In a sensational series of reports, Sabel and colleagues (Kasten et al) have described partial recovery of homonymous visual field defects by intensive computer-based rehabilitation therapy.1–3 Their approach is remarkably simple. Patients practise perimetry at home for 6 months, using a software program loaded on their personal computer. A chin support is used for head stability and a monitor is placed 30 cm away. Stimuli are white, suprathreshold lights measuring 0.15˚ in diameter shown against a dark background. Protocols are tailored for each patient to present most stimuli near the border of the field defect (“transition” zone) to maximise potential therapeutic benefit. Sabel has founded a company (NovaVision) that offers visual rehabilitation therapy for about €5000.

The idea behind visual restoration therapy is that after stroke or traumatic brain injury, a region of salvageable vision exists between areas of the visual field served by normal and damaged brain tissue. Visual stimulation in this zone with more than 1000 trials a day is postulated to resuscitate its functional potential. After treatment, homonymous field defects have been reported to show a mean azimuth reduction of 4.9˚ (nine patients).1 Individual patients have shown up to 30˚ of field recovery. These are dramatic results for patients suffering from post-chiasmal visual field loss. A wildly optimistic commentary accompanying the findings in a scientific journal carried the title, “Those that were blind can now see.”4

A major problem with the data reported by Sabel et al was that the same software program used for visual restoration therapy was also used to show improvement in the visual fields. Obviously, the data would be more compelling if visual field improvement could be demonstrated with any standard clinical perimeter. When patients with post-chiasmatic lesions were tested before and after visual restoration therapy with the Tübinger automatic perimeter, no benefit of treatment could be detected.1

To resolve doubts regarding the efficacy of visual restoration therapy, Sabel teamed up with scientists in Tübingen, a centre renowned for leadership in the field of perimetry. In the resulting study, published in this issue of BJO (p 30), 17 patients with stable homonymous field defects were treated according to the visual restoration therapy protocol. Independent visual field testing was done before and after treatment at Tübingen to assess the outcome. A crucial innovation was that perimetry was performed using a scanning laser ophthalmoscope, which allows the examiner to control fixation assiduously by simultaneous visualisation of the retina, fixation cross, and stimulus. Under such conditions, invalid trials as a result of inadequate fixation (for example, saccades) can be disregarded. Unfortunately, the study found no significant improvement in visual field defects, although most patients had the subjective impression that they had benefited from visual restoration therapy. This discrepancy underscores a limitation of outcome satisfaction surveys: patients can be swayed by placebo effects.

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How can one reconcile Sabel's original findings with this latest study? Patients with homonymous field defects compensate by making frequent saccades towards their scotoma in an effort to maintain surveillance of blind regions in their visual fields.5–6 It is notoriously difficult to control fixation in such subjects. During visual restoration therapy, fixation is monitored by randomly changing the colour of a 0.75˚ fixation light from bright green to yellow whereupon the subject is required to respond within 500 ms by pressing a button. The problem with this technique is that the colour transition is so easy to detect that it does not require foveal vision. Patients soon learn that they can sneak 5˚ saccades into their blind hemifield, and still detect a change in the colour of the fixation monitoring light. Hence, the mean 5˚ improvement in the visual field defect.

Several aspects of the original report describing visual rehabilitation therapy should have raised doubts earlier. Firstly, no information was provided regarding false negative, false positive, and fixation loss rates for patients. Perimetric data purporting to show improvement in visual fields are difficult to interpret without these indices. Secondly, the proposed mechanism for partial field recovery in patients with complete hemianopia was flawed. In such subjects, the normal occipital lobe and the affected occipital lobe are physically separate—no fringe of injured but salvageable tissue exists that represents the border of the visual field defect. In this situation, why should visual rehabilitation therapy produce field recovery along the vertical meridian? Thirdly, visual rehabilitation therapy was reported to be effective for both monocular optic nerve diseases and homonymous, post-chiasmal lesions. It is difficult to conceive of a physiological mechanism that could explain improvement from the same treatments at different levels of the visual system. It is so unlikely, in fact, that an artefact such as poor fixation control should have been suspected immediately. Fourthly, why should an artificial stimulus applied for an hour a day be more effective than the incredibly rich repertoire of natural light patterns that stimulate the retina under normal, everyday circumstances?

If physical therapy is helpful in patients who are partially paralysed by a stroke, why shouldn’t visual rehabilitation therapy work too? The difference, of course, is that the former involves motor systems that can be retrained to compensate for deficits. Through therapy, patients learn how to use new motor strategies with still functional muscle groups to accomplish a physical act. In contrast, lesions of the retino-geniculo-cortical pathway produce a purely
sensory deficit. No credible evidence exists to suggest that the adult visual cortex can be revived after injury by training exercises or visual therapy. Patients with homonymous hemianopia can benefit from counselling to assist with safe travel, obstacle avoidance, and career planning. Regrettably, it still remains true that no therapeutic intervention, prosthesis, or prism can correct effectively the underlying visual field deficit.

This is not the first time that hopes for visual field recovery by rehabilitation training have been raised and dashed. Twenty years ago, Zihl and von Camon reported improvement in field deficits in patients with post-geniculate damage by visual training. The findings were later shown to be artefacts of poor fixation control. Sabel is due great credit for submitting visual rehabilitation therapy to independent scrutiny. What distinguishes medicine from “alternative therapies” is that it strives to be evidence based. Here, a proposed therapy has been retested scientifically and found to be ineffective. This information allows us to refocus our search for new treatments in a more fruitful direction.


Correspondence to: J C Horton, UCSF Department of Ophthalmology 10 Kirkham Street, K301 San Francisco, CA 94143, USA; horton@hsa.ucsf.edu

REFERENCES


Editorial

A work out for hemianopia

G T Plant

A rigorous study finds no evidence of improvement in homonymous visual field defects with training

At the recent meeting of the International Neuro-ophthalmology Society in Geneva one of the most interesting presentations was given by two collaborators who disagreed on the interpretation of their joint findings. The atmosphere was more than usually stimulating. The disputed results are published in this issue of Br J Ophthalmol (p 30).

It was Josef Zihl from Munich who in 1979, with von Cramon, put forward the idea that patients with homonymous hemianopia by encouraging the development of eye and head movement strategies as Zihl himself later reported. These may be tailored for specific tasks—for example, navigation (Christopher Kennard’s group at the Charing Cross Hospital in London) and reading (Richard Wise and Alex Leff at the Royal Free Hospital in London). These strategies do not claim to improve the bare perimetric results but may enable the patient to make better use of his or her residual vision.

Collaborations of this type require a degree of courage and trust and we should suitably applaud these researchers.


Correspondence to: G T Plant, The National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK; gordon@plant.globalnet.co.uk

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Temporal or inferotemporal disc margin. These crescents were usually bilateral and were most often located along the semilunar line, partially parallel to the peripapillary scleral ring (scleral lip). One must be aware not to confound the grey crescent with the alpha zone. The difference between both structures is that the alpha zone is located outside of the optic disc and may not be counted as neuroretinal rim, whereas the grey crescent is located inside of the optic disc and may partially or completely be regarded as neuroretinal rim. Not considering the differences between the alpha zone of parapapillary atrophy and the grey crescent will, therefore, markedly influence the morphological analysis of the optic disc.

The question arises what the histological equivalent of the grey crescent may be. Since the grey crescent is relatively dark, it may be associated with retinal pigment epithelium cells. These cells sitting on and forming Bruch’s membrane, may partially be located in the optic nerve head region if Bruch’s membrane extends internally to the peripapillary scleral ring. Future histological studies of the normal optic nerve head may be directed towards finding the clinical-histological correlate of the grey crescent, differentiating it histologically from the physiological alpha zone of parapapillary atrophy, and giving hints for the rate of the histological occurrence of the grey crescent in normal eyes. Optical coherence tomography may be an additional clinical method to analyse intravitreally the grey crescent and its spatial associations with the surrounding tissues.

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