Loss of balance can result from numerous causes but the most common are pathologies associated with the peripheral vestibular organs of the inner ear. For example, over half of patients who have had a serious fall show signs of vestibular organ disorder. This is especially the case in the elderly, where approximately 25% of those aged over 65, and an astonishing 85% of those over 80, have dramatically reduced vestibular organ function [1,2]. Swelling these already significant numbers are adults under 65 with an injury to the balance organs [3]. The resulting costs are substantial; injuries alone in the USA are estimated to account for a staggering loss to the economy of $130 billion per year.

The two balance organs, one in each inner ear, sense head rotations and send signals almost directly to the eye muscles. Without this reflex eye movement, known as the vestibulo-ocular reflex (VOR), stable vision becomes impossible during head movement. A majority of those that have suffered a loss of vestibular function through ageing or injury, benefit from vestibular rehabilitation, a series of exercises designed to promote a relearning of balance control and vision stabilisation during head movements. However, 1 in 50 of these patients will have complete bilateral loss. No rehabilitative therapies can improve blurred vision during head movements in these patients because all existing therapies rely on the vestibular system receiving a signal from at least one partially functioning balance organ. Therefore development of a vestibular prosthesis is considered to be an important goal of vestibular research because it provides the only real hope of recovery for patients with complete vestibular organ loss [4]. However, once patients begin to receive vestibular prosthesis implants it is likely that vestibular rehabilitation will also be needed to assist their functional recovery.

Recent literature, including double-blind placebo controlled trials, has supported the utility of vestibular rehabilitation in a variety of conditions associated with dizziness. Most rehabilitation therapies are administered by physicians or physical therapists, but there are also some do-at-home exercises that have been tested by time, e.g., Cawthorne Cooksey exercises. Nevertheless, except for a few situations, these interventions are not powerful [5]. Another observation is that identical vestibular lesions that cause devastating loss of mobility and quality of life for most patients can result in no more than a minor inconvenience for others [6]. The reasons for this are unknown but the difference suggests that some patients compensate better than others. Visual stability can improve after a partial vestibular injury if the remaining vestibular system improves its response. The mechanisms behind this type of compensation are not well understood. However, it has been shown that the vestibular system is highly plastic and can adapt its response under normal conditions and post-lesion [7,8]. Visual stability can also improve when non-vestibular vision stabilizing oculomotor systems, e.g., smooth pursuit augments the reduced vestibular response [8,9]. Taken together these observations suggest that vestibular compensation outcomes would be much improved if the underlying mechanisms of vestibular adaptation and compensation were better understood.

One particular line of research is examining the factors that improve VOR adaptation. Retinal slip induces a VOR response change when the head and visual-target velocities are incongruent. A number of human VOR studies have demonstrated a robust capacity for adaptation of the normal VOR by coupling head motion with target motion to elicit retinal slip as a velocity error signal [10,11]. Classic studies of the VOR have shown that the VOR response (or gain; gain = eye velocity / head velocity) can be increased (5 days of wearing x2 magnifying lenses resulted in the VOR gain [tested in the dark] increasing by 53% when attending to an unseen earth-fixed surround [12]) decreased (short-term (2 hour) visual fixation on a mirror-reversed image of the surroundings resulted in an adaptive decrease of the VOR gain [tested in the dark]) and reversed (long-term 5 x 15 minutes adaptation sessions per day on days 1, 2, 6, 7 and 27 [12]). In the reversal study, the adaptation was retained overnight and added to with subsequent sessions, the VOR response was reversed by day 27, and de-adaptation occurred over a similar time period to adaptation, i.e., 2-3 weeks. The outcomes from these experiments, however, were not considered useful in a rehabilitation context because the adaptation was most evident when testing the VOR in darkness. Under normal lighting the adapted response significantly decreased (~70%). Adaptive plasticity is likely to be mediated by the vestibulo-cerebellum.
flocculus, which generates an inhibitory signal that modulates the VOR gain at the vestibular nucleus level. When the flocculus is lesioned the VOR gain saturates at ~1.6 and vestibular plasticity is abolished [13]. Traditional studies of VOR gain adaptation challenge the VOR to change “all at once” and typically make the visual target move at the same speed as the head but in the opposite direction (a x2 stimulus) so that the VOR has to compensate for twice the head velocity – a large change all at once [10-12]. However, non-vestibular motor control studies and auditory perception studies indicate that smaller and incremental error signals in learning tasks drive neural plasticity and learning more effectively than large error signals [14-16]. Schubert et al. [17] showed that the VOR could be robustly adapted in dim light using an incrementally increasing VOR challenge stimulus during active (self-generated) head rotations. ‘Incremental’ versus ‘traditional’ all-at-once adaptation was compared in 7 normal controls and 6 patients with unilateral vestibular hypofunction. The passive (imposed head rotation) and active (self-generated) VOR gain was measured before and after adaptation training. For incremental adaptation, a x1.1 stimulus was used to increase the VOR by 10% and, after a brief rest, a x1.2 stimulus took it a further 10% (20% total), and this was repeated until the x2 stimulus was reached. The total adaptation time was 15 minutes. On a separate day, the VOR gain was similarly measured before and after all-at-once (x2) adaptation training lasting 15 minutes. For both subject groups, traditional all-at-once adaptation resulted in no VOR gain increase, whereas incremental adaptation resulted in significant increases in both normal and patient subjects. A subsequent study showed that the incremental technique could be used to increase the VOR response to one side only [18] which was an important finding because the majority of vestibular patients have a unilateral lesion and so the VOR must be increased for rotations to the injured side only. Increasing the VOR gain for rotations towards the normal side would result in inappropriately large eye movements when the head rotated towards that side. These two studies showed that: 1) significant unilateral and bilateral VOR adaptation occurs after only 15 minutes of training, i.e., versus 3 hours [19], 2) training can consist of only self-generated active head impulses, i.e., versus passive head impulses that require the use of a rotary chair or manual operator, and 3) the VOR adaptation is retained in a darkened (dimly lit) environment, i.e., versus strict darkness. The next steps will be to determine: 1) whether patients with vestibular hypofunction show similar ipsilesional adaptation, 2) the retention period after unilateral adaptation, which is an important question for maintenance training, and 3) outcomes in patients performing regular adaptation training versus current best practice.

REFERENCES


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