Vestibular Disorders

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Preface

In no area of neuroscience have recent advances in fundamental research had more profound impact on clinical research and practice than vestibular studies. *Vestibular Disorders* presents the latest viewpoints of both basic and clinical scientists on the disordered physiology of the vestibular system, with goals of understanding its disturbances and providing scientific bases for testing its functions, and for managing patients. The text is divided into four parts that deal with (1) the neurophysiology of orientation; (2) the comparative merits of different vestibular function tests; (3) smooth eye movement disorders; and (4) the diagnosis and management of dizziness. This book is directed at physicians and physiologists who are concerned with dizzy patients. The contents are not encyclopedic; the book is intended to be sufficiently broad to satisfy the needs of experienced otologists or neurologists and to provide a foundation for residents. We have selected topics by expert contributors to highlight recent important advances. Contributors have discussed those aspects of physiology most pertinent to understanding vestibular disorders.

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PART ONE

Recent Concepts of the Neurophysiology of Orientation

1

*Experiments on Vestibular Adaptation and Its Clinical Significance**

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It is well known to the neurotologist that the vestibular system as a whole exhibits a remarkable potential for clinical rehabilitation after pathologic lesions. Even after the extreme insult of unilateral ablation of the peripheral end organ and/or its peripheral innervation, there often is progressive recovery toward apparent behavioral normality, including return of the patient to the work force. Nevertheless, there remains much to learn about the fundamental underlying physiologic processes responsible for this kind of neurologic rehabilitation.

For example, although neurologic lesions can certainly generate central plastic reactions such as neural sprouting, reactive synaptoge-

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nesis, and deafferentation supersensitivity,[7–9, 56, 57] the extent to which these processes need to be brought under behavioral control for full functional recovery remains debatable.[17, 34, 58, 61] Despite these uncertainties, at least some key factors are clear. Thus, even in the absence of any lesion, purely behavioral stimuli can readily be made to induce extensive adaptive plastic changes of reflex function.[2, 26, 33, 40] Moreover, these behaviorally induced changes of reflex function appear to be associated with correlated central neurophysiologic changes of a plastic nature.[18, 29, 43] However, the rehabilitative process is not restricted to these mechanisms. An additional feature is the organized recruitment of allied sensory-motor systems to "make good," or "substitute" for, lasting deficiencies incurred by the primary pathologic impairment.[28, 49] No doubt in practice any call for "action" initiates a multifactoral "search" for "best" solutions to a particular problem, drawing in idiosyncratic fashion from the available repertoire of these physiologic options.[1]

We review briefly first the experimental basis of behaviorally induced vestibular adaptation in normal subjects. Next we describe some recent studies which demonstrate the correction of vestibulo-ocular inadequacy by the synergistic contribution of complementary saccadic eye movements acting in the *compensatory* direction. Finally, we address the clinical relevance of these findings as reflected in the overall rehabilitation of a patient subjected to unilateral 8th nerve neuronectomy due to acoustic neuroma.

REVIEW OF ADAPTIVE PLASTICITY IN THE SLOW-PHASE COMPONENT OF THE VESTIBULO-OCULAR REFLEX (VOR)

Behavioral Experiments

In the Introduction, we questioned the extent to which a patient's plastic neural reactions to an internal lesion could be brought under behavioral control through an active working encounter with the external environment. At first it seemed difficult to conceive how the essentially internal phenomenon of central neural plasticity could, *of itself,* serve to reestablish a proper degree of sensory-motor correspondence[36] at the interface with the external physical world. Presumably for functionally meaningful recovery, the active process of internal neurologic rearrangement would have to be guided by some kind of adaptive informational feedback from the working brain-world interface.

Following these guidelines, it was argued that if such feedback

could be activated in the *abnormal* subject by physical contact with our *normal* environment, then conversely it should surely be possible to induce analogous effects in the laboratory by exposing the *normal* human subject to an *abnormal* encounter with the external environment. It was essentially this "guess," together with the recognized need for active maintenance of even the normal "status quo" (neurologic homeostasis), which first triggered[14] an extended series of experimental studies designed to investigate the potential for purely behavioral induction of adaptive plasticity within the CNS.

Initial experiments utilized the VOR as a model sensory-motor system. Leaning on the outcome of psychologic studies of Kohler,[27] optically reversing prism goggles were used to "ask" the involuntary VOR to reverse itself. The prism goggles were arranged on the head so that when turning, say, to the right, the eyes would have to move smoothly *also to the right* if they were to "see" a stable image of the external world on the retina during head rotation, which is exactly opposite to normal VOR compensation. Short durations of exposure to this demanding situation resulted in relatively large degrees of attenuation in slow-phase gain of the dark-tested reflex (slow phase VOR gain = [compensatory eye velocity]/[head angular velocity]).[15]

Figure 1–1 summarizes results obtained from 7 human subjects. The lower curves show the mean daily reduction in (normalized) VOR gain resulting from two successive 8-minute exposures to mirror reversal of vision during sinusoidal whole body rotation, repeated on 3 successive days. On each day there was a highly significant VOR attenuation amounting to about 20% of the initial condition, albeit with almost (but not quite) complete recovery between days. The top row of control points demonstrates that this attenuation was not due to simple habituation, since exactly the same vestibular stimulus profile (⅙ Hz, 60°/sec amp) conducted *without* vision reversal produced no significant alterations in the dark-tested reflex, as has been confirmed for this stimulus frequency by other authors.[23]

Bearing in mind that Kohler's subjects needed weeks, not minutes, of continuous vision inversion to produce effective functional adaptation in locomotive and sportive activities, we similarly extended our experimental exposures in the reversed vision paradigm. As illustrated in Figure 1–2,A, after about 2 weeks ambulatory activity with continuous left-right vision reversal, the *dark-tested* VOR produced an adaptively altered pattern of ocular response in which both the slow and quick phases of resulting nystagmus became effectively the reverse of normal.[16, 32] This new condition was approached gradually through a combination of progressively reduced gain (upper

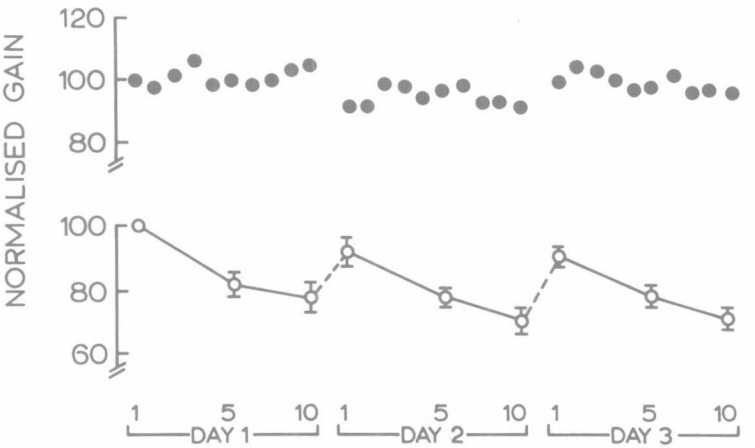

FIG 1–1.

Short-term adaptive changes in the human VOR. Upper control data points define the lack of habituation produced by repeated 2-min exposures to horizontal sinusoidal rotation with normal vision. Each point shows dark-tested VOR gain measured after each 2-min period of normal vestibular-visual interaction. Lower curves show progressive attenuation of dark-tested VOR, with 8 min of reversed visual-vestibular conflict between each point. Note restoration of VOR gain between consecutive daily tests, with a small but significant decline of initial test results on each of 3 consecutive days. (Data from Gonshor A, Melvill Jones, G: Short-term adaptive changes in the human vestibulo-ocular reflex arc. *J Physiol* 1976; 256:361–379.)

curves in each figure) coupled with changes of phase (lower curves) that had a trend toward a reversed response, which would ideally be represented in these figures by a 180° phase shift. Figure 1–2,B, shows similar patterns of change in the cat exposed to very long durations of continuous dove prism reversal of vision.[37] The inset in Figure 1–2 shows samples of original tracings of compensatory nystagmus from the human subject during sinusoidal whole body oscillation in the dark, (a) during a control test and (b) after 14 days of adaptation to the reversed visual-vestibular conflict. Notice the almost complete reversal of the adapted reflex response, although at somewhat reduced gain, denoted by the respective calibration bars.

Important in Figure 1–2 are the following three features: (1) effects were long-lasting; (2) observed changes were adaptive in that they improved the functional goal of retinal image stabilization during head rotation; (3) given adequate time (roughly equal to the duration of the preceding reversed vision experience) a return to normal vision completely restored the normal VOR, although in a somewhat complex

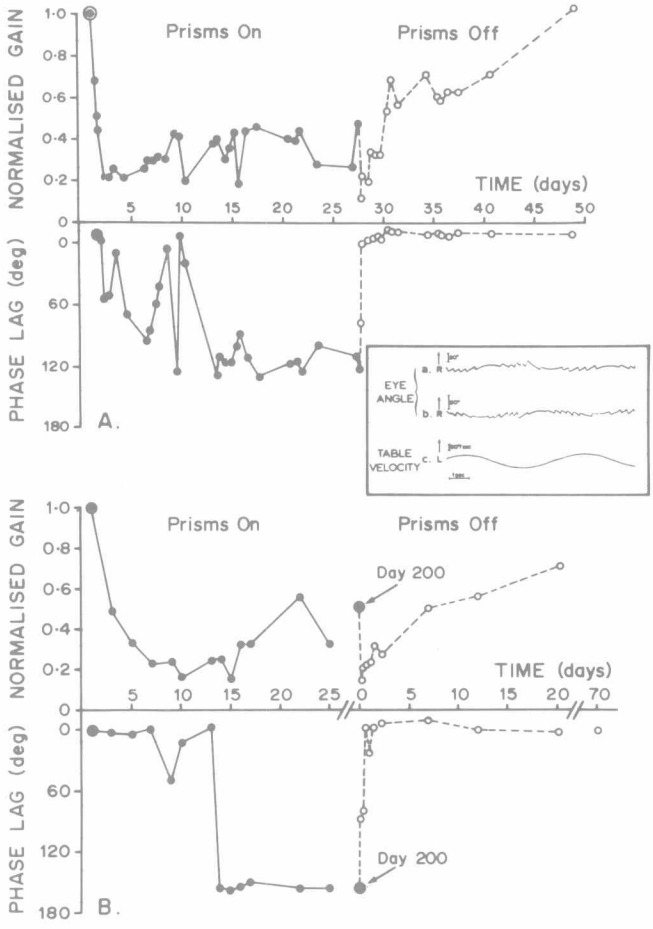

FIG 1–2.

Time course of long-term VOR adaptive changes in gain and phase during (●) and after (○) the wearing of horizontally reversing dove prisms. **A,** man, tested at 1/6 Hz, 60°/sec peak amplitude. **B,** cat, tested at 1/8 Hz, 5°/sec. The phase is registered relative to perfect normal compensation. All tests were conducted in darkness. *Inset* shows samples of normal *(upper)* and adapted ("reversed") nystagmus tested in the dark after 14 days of continuous vision reversal. (Adapted from Melvill Jones G: Plasticity in the adult vestibulo-ocular reflex arc. *Philos Trans R Soc Lond* [*Biol*] 1977; 278:319–334.)

manner.[39] These characteristics led us to adopt the term "adaptive plasticity" to describe the general features of this phenomenon.

Additional animal experiments both replicated and extended the findings of these early studies in the cat[31, 48]; in the rabbit[22]; in the monkey[42]; in the chicken[59]; and in the goldfish.[50]

For example, Figure 1–3 illustrates the systematic augmentation of VOR gain produced in monkeys who wore binocular spectacles which, instead of reversing the seen world, simply magnified it by a factor of 2.[42] After a week or so of wearing these binoculars, the monkey's dark-tested VOR had appropriately nearly doubled itself. This condition was approached approximately exponentially and tended to be retained indefinitely in the absence of further visual-vestibular interaction. However, as in the earlier human experiments, the VOR could be completely restored to normal by reexposure to normal vision and movement, albeit with a somewhat faster time course than the original adaptation. Moreover, reexposure of the same animal to the same adaptive stimuli repeatedly produced an identical time course of VOR gain change, emphasizing the almost "machine-like" nature of the adaptive process. In line with these findings, Miles and Eighmy[42] also showed that substituting miniaturizing lenses for the magnifying ones produced the appropriate effect of attenuating, rather than augmenting, slow-phase VOR gain.

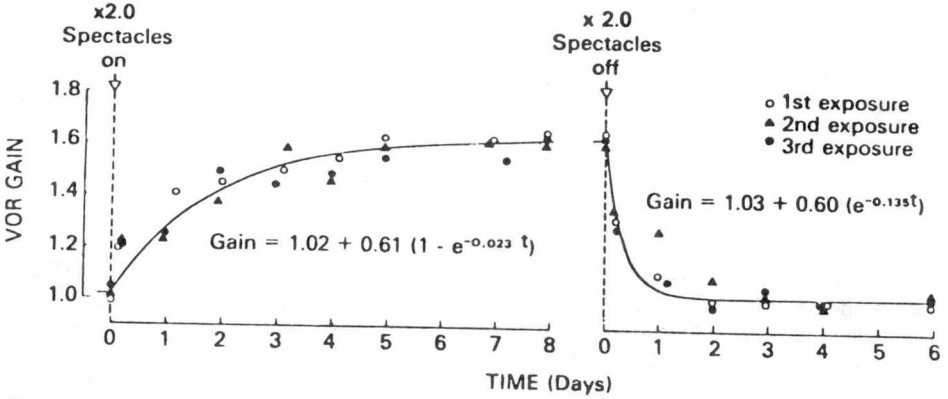

FIG 1–3.

Adaptive enhancement and recovery of VOR gain in a monkey exposed to continuous ×2 binocular vision. The different symbols represent data from the same animal obtained on different occasions. The similarity of the curves they depict emphasizes the "machine-like" characteristics of the adaptive process. (From Miles FA, Eighmy BB: Long-term adaptive changes in primate vestibuloocular reflex: I. Behavioral observations. *J Neurophysiol* 1980; 43:1406–1425. Used by permission.)