

International Symposium for Therapy of Ocular Motility and Related Visual Disturbances

Henry J. Kaminski, MD, and R. John Leigh, MD

A symposium dedicated to the emerging field of therapeutics of ocular motility disorders was held on November 15th and 16th at Case Western Reserve University in Cleveland, Ohio. Abnormal eye movements are of general interest to neurologists because of their diagnostic value, and the literature in this field has burgeoned over the past quarter century. In contrast, interest in treating the visual consequences of abnormal eye movements has been mainly restricted to management of diplopia, which has been the province of orbital surgeons. Bender¹ was among the first to define visual complaints associated with abnormal eye movements and draw attention to the complaint of oscillopsia—illusory motion of the seen world—that is reported by some patients with nystagmus. Recent research has delineated the visual requirements that eye movements must satisfy: proper alignment to allow binocular single vision and relative stability of the line of sight for the image to appear clear and still.² Patients whose abnormal eye movements disrupt single, clear vision do consult neurologists and ophthalmologists, but these clinicians often complain, with justification, that there is no organized body of knowledge to consult with regards to treatment. For example, standard neuro-ophthalmology texts have little to say about treatment for eye movement abnormalities. In fact, there are numerous case reports of patients whose abnormal eye movements have been helped by this or that treatment, but there have been few controlled clinical trials to evaluate such measures.³ Another major deficiency is that treatments have largely been empirical, and there is great need to organize basic knowledge about normal and abnormal gaze control, so that it can be used to guide therapeutic efforts.⁴ It was against this background, and a local interest in eye movements fostered by Dr. Robert B. Daroff, that the conference was organized. Twenty-four investigators, with expertise in both basic science and clinical issues, contributed to this symposium, which addressed both central and peripheral disorders of the control of eye movements, and their visual effects.

Basic mechanisms for normal and abnormal gaze control and effects on vision. Dr. Jean Böttner-Ennever of Ludwig-Maximilians University of Munich opened the meeting by reviewing *current neuroanatomic concepts related to the control of eye movements*.⁵ Of especial relevance to nystagmus are her studies defining the cell groups of the paramedian tracts (PMT), which lie scattered along the midline fiber tracts in the pons and medulla, close to the medial longitudinal fasciculus.^{6,7} The PMT cell groups are unknown to most neurologists but are extraordinary because they receive inputs from all structures that project to oculomotor neurons. Thus, they could function by "monitoring" premotor signals. The PMT cell groups project solely to the cerebellar flocculus and ventral paraflocculus.^{8,9} This part of the vestibulocerebellum is known to be important for responding to "retinal slip" information, but if the cerebellum is to behave as a "repair shop" that ensures eye movements meet visual needs, then it also needs to monitor ocular motor commands. The PMT cell groups may play a key role in this regard, and, if so, it follows that disruption of their projections by disease may deprive the cerebellum of essential information, leading to instability of gaze.¹⁰

Dr. Ulrich Böttner, also of Ludwig-Maximilians University, presented an update on the *pharmacology of the vestibular and ocular motor systems*, an area that has grown rapidly over the past 10 years.¹¹⁻¹⁴ One problem posed by this research is that although many neurotransmitters and neuromodulators have been identified within ocular motor structures, the functional significance of most of these molecules remains unclear. One approach to clarify their roles in the control of gaze has been to study the behavioral effects that follow pharmacologic inactivation by microinjection into specific sites in the brainstem and cerebellum. In this regard, the studies by Dr. Böttner et al.¹² were pivotal in demonstrating the importance of GABA in the control of gaze. They, and others,¹³ targeted the nucleus prepositus hypoglossi - medial vestibular nu-

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From the Department of Neurology, Veterans Affairs Medical Center and University Hospitals of Cleveland, Case Western Reserve University School of Medicine, Cleveland, OH.

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