Sixty-nine cases of optic nerve atrophy were examined electrophysiologically. 42% displayed enhanced ERGs together with reduced or absent visual evoked potentials. Eight cases of these with conditions resulting from blunt head injury, brain tumor, encephalitis and multiple sclerosis are presented in detail.

The distribution of the b-wave and a-wave amplitudes of the pathological ERG was studied and compared with normal ERGs. The b-wave was taken as criterion for retinal sensitivity for reasons explained. Possible underlying mechanisms of the electrophysiological data are speculated considering that 55% of the cases displayed reduced ERGs. The data seem to support the hypothesis that in the intact visual system impulses propagated along centrifugal optic nerve fibers inhibit retinal activity at the bipolar cell level, an effect rivaled by the growing retinal sensitivity during dark adaptation. In the absence of the efferent effect in optic nerve involvement, the ERG recovery would be unrivaled resulting in enhanced ERGs. An inhibition of this assumed inhibitory feedback on the retina by light adaptation is postulated and supported by evidence from animal experiments found in the literature. This hypothesis and alternative hypotheses are applied to the cases examined.

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The occurrence of reduced ERGs is explained by an involvement of the bipolar and receptor cells due to a progressive degeneration of possibly existing intraretinal centrifugal fibers. In favor of this is the significant diminution of the a-wave in contrast to the generally normal a-wave values found in cases with enhanced ERGs as compared with the ERG of normal persons.

INTRODUCTION

The participation of the retinal ganglion cells in the clinical electroretinogram (ERG) is generally considered negligible. Experimental evidence and clinical findings in conditions where the ganglion cells were degenerated (Granit & Helme, 1939; Karpe, 1945; Noell, 1953; Müller-Limroth, 1959; Brown, 1968), point to the first two neurons in the retina as being responsible for its elicitation. However, experimental and clinical observations show that a disturbance in the impulse propagation through the optic nerve fibers, the axons of the ganglion cells, often causes alterations which are reflected by the ERG in the retinal activity of the corresponding eye. In some cases the ERG becomes larger than that produced in the eye with intact central connections and often reaches 'supernormal' amplitudes (Dieterle & Babel, 1955; Jacobson & Gestring, 1958a, 1958b; Suzuki, 1959; Straub & Rank, 1959; Straub, 1961; Abe, 1962; Nagaya et al., 1962; Gills, 1966a; Feinsod & Auerbach, 1969). In other cases it may remain normal or does not attain normal values (Ebe, 1964).

A disturbance in the nervous conduction between retina and cortex is demonstrated by a greatly reduced or even extinct visual evoked potential (VEP) in the cortex. The existence of centrifugal fibers in the optic nerve, which are thought by some of the authors to synapse with the amacrine cells (Ramón y Cajal, 1892-3; Cowan & Powell, 1963; Wolter, 1965; Wolter & Knoblich, 1965; Brooke et al., 1965; Honrubia & Elliot, 1968), seems to point to a negative feedback system in the normal visual system (Granit, 1955; Dodt, 1956). There are other contrary reports, however, where no negative feedback was found, which make the subject controversial (Brindley & Hamasaki, 1962; Mita, 1962; Ebe et al., 1964; Brindley & Hamasaki, 1966).

Because of these differences in opinion, it seemed to us necessary to evaluate the clinical material of 69 cases of optic nerve atrophy examined up to this date in our laboratory, and to try to draw some conclusions as to the function of the intact visual system. In this electrophysiological study, the data of all cases