HEALTH RESEARCH ABSTRACT SUBMISSIONS

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Development of Pupillary Adrenergic Supersensitivity after Pharmacologic Induction of Oculosympathetic Defect

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Introduction & Purpose *

Interruption of oculosympathetic nerve activity induces the development of adrenergic supersensitivity of end–organs. Evidence suggests that this takes 3–5 days, but some report as few as 36 hours. Brimonidine is a selective alpha–2 agonist and will produce a pharmacologic oculosympathetic deficit by inhibiting presynaptic sympathetic neurotransmitter release. Apraclonidine is an alpha–2 agonist with weak alpha–1 agonistic properties that manifest if end–organ supersensitivity is present. The goals of this study are to: 1.) Investigate the time course for development of adrenergic supersensitivity by monitoring pupil size in response to apraclonidine after induction of a pharmacologic oculosympathetic defect using brimonidine and 2.) Establish objective criteria to detect pupillary adrenergic supersensitivity for the pharmacologic diagnosis of an oculosympathetic palsy (Horner Syndrome).

Experimental Design *

In this cohort study, 10 healthy subjects were prospectively studied by administering one drop of 0.2% brimonidine to the right eye (OD) twice daily for 5 days to induce an oculosympathetic defect. The left eye (OS) was used as a control. Adrenergic supersensitivity was assessed by measuring the pupillary response of both eyes to one drop of 0.5% apraclonidine daily and comparing the symmetry of reaction between the pupils to a control population of 100 normal subjects.

Results *

4 of 10 subjects developed pupillary adrenergic supersensitivity during this study period with or without the reversal of anisocoria. These 4 subjects each initially demonstrated supersensitivity to apraclonidine in the brimonidine treated eye starting at 24, 48, 72, and 96 hours, respectively.

Conclusions *

The results of this study suggest that detectable adrenergic supersensitivity of end–organs develops as soon as 24 hours from the onset of a pharmacologically induced sympathetic palsy. Objectively measuring inter–eye asymmetry of pupil reaction to apraclonidine can be utilized as a diagnostic tool for detecting supersensitivity in unilateral oculosympathetic defects irrespective of whether the anisocoria reverses.