HEALTH RESEARCH ABSTRACT SUBMISSIONS #10

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College *	College of Medicine
Department *	Ophthalmology and Visual Sciences
Title of Research *	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.

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