LIGHTING CONDITIONS AND STIMULUS DURATION AND THIER EFFECTS ON QUANTIFICATION OF AFFERENT PUPILLARY DEFECTS

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PURPOSE

Identifying an afferent pupillary defect is an essential part of diagnosing and managing various ocular diseases. The following paper discusses and evaluates the pupillary pathway and optimum testing conditions for consistent diagnosis of afferent pupillary defects. In the following study, afferent pupillary defects were quantified through the use of neutral density filters which ranged from .1 to 1.1 log units in .1 incriment steps. Pupillary defects were measured initially in normal room lighting, then in dim lighting, and once again after dark adapting for 5 minutes. In addition, the effects of stimulus duration or pause time was varied from 1 second, 5 seconds, and 10 seconds under dim room illumination and the afferent pupillary defect was evaluated for any change.

In order to accurately assess a pupillary defect one must first have an understanding of the basic anatomy and the mechanisms responsible for pupillary responses, as well as the proper technique for assessment. An afferent pupillary defect occurs when there is a decrease in conduction of light along the afferent pathway. This decrease is conduction is usually caused by damage to the optic nerve from such things as glaucoma, central retinal vein occlussions, and inflammation. An apparent defect can also be seen with dense cataracts, retinal detachments , and retinal and vitreal hemorrhages, as these can also cause a decrease in the amount of light hitting the retinal photorecepetors. As a result of the decreased conduction of light, whether it be caused by optic nerve damage, retinal disease, or dense cataracts, when light is flashed alternately between the right and left eye, an observation of a dilating pupil and/or a constricting pupil can be seen. A better understanding of how this occurs can be understood after examining the pupillary pathway.

In a normal eye, when a light source is directed at the eye, retinal photoreceptors are stimulated. These photoreceptors send impulses to the bipolar cells and then to the ganglion cells which form the optic nerve. From the optic nerve, impulses are passed along to the optic chiasm, where the fibers cross to both the ipsilateral and contralateral optic tracts.[1] This is the reason we see a direct and consensual pupil response. Impulses travel along the optic tract, where they split off just before the lateral geniculate body and head to the superior colliculus in the midbrain. From the superior colliculus, impulses travel to the pretectal nuclueus where they synapse. Neurons are then carried to the edinger westphal nucleus where they synapse marking the ending of the afferent (sensory) path and the beginning of the efferent (motor) path. Efferent impulses enter the orbit via the superior orbital fissure and synapse at the ciliary ganglion then becoming the short posterior ciliary nerves which innervate the sphincter muscles of both eyes.[1] Innervation of the sphincter muscles cause both pupils to constrict equally and simultaneously.

In an eye with a defect, equal and simultaneous constriction of both pupils will not be seen. The affected pupil will still constrict to light as impulses are still sent to all structures of the pupillary pathway; however, depending on the severity of the defect, the impulses will be reduced as compared to the normal eye. The eye with the afferent abnormality can be thought of as "seeing less of the light" and therefore receives weaker impulses resulting in a dilation on the abnormal side and a constriction on the normal side, indicating an afferent pupillary defect.[2] This is most easily identified clinically with the use of the Swinging Flashlight Test.

The Swinging Flashlight Test should be done in a room with dimmed lights. The examiner should be positioned so that they can see both pupils clearly and so that they are not obstructing the patients view of the target. Have the patient look at a distant target that does not involve accommodation, as this can cause unwanted pupillary constriction. Using a bright illumination source, a penlight for example, shine the light for one to three seconds in each eye, one at a time. Assess each pupil independently looking for a direct response. Once the direct response has been evaluated, illuminate one eye for one to three seconds and then quickly pass the light across the bridge of the nose to the other eye for one to two seconds repeating the cycle. If both eyes are stimulated equally, the amount of constriction should remain equal as you swing the light between the two eyes. This tells us that each pupil is receiving a symmetrical and constant afferent input, whether it receives input from direct stimulation or by way of the reflex arc through stimulation of the contralateral eye.[3]

If a defect is present, the abnormal eye is thought of as "seeing less light" as the afferent impulses are weaker in that eye. Therefore, as you swing the the light from the good eye to the bad eye, the bad eye will show a dilation. For example, consider a patient with a normal right eye, but optic nerve damage to the left eye, resulting in an afferent pupillary defect. When light stimulates the right eye, both pupils constrict symmetrically. The right pupil by direct response and the left pupil by consensual response. When we swing the light across to the left eye, the injured optic nerve can not generate as great of an impulse as the right eye and thus the left pupil dilates when the light arrives. Because an equal efferent response is sent consensually to the right eye, it dilates as well and when we swing the light back to the normal right eye more light is impulsed and the pupils constrict. The observation of an initial dilation movement (abnormal response) on one side and a constriction (normal response) on the other side is indicative of an afferent pupillary defect on the dilating side.

The purpose of our study was to determine what effect various lighting conditions, such as normal room illumination and dark adaptation, have on the grading of an afferent pupillary defect using neutral density filters. In addition, how light stimulus duration (pause time) affects grading afferent pupillary defects will be assessed. As an end result, we hope to provide a guideline for optimum stimulus duration and lighting conditions necessary for

EVALUATION OF THE PUPILLARY RESPONSE WITH THE SWINGING FLASHLIGHT TECHNIQUE

NORMAL PUPIL SIZE AND REACTIVITY

↓ shine light in right eye

LEFT AND RIGHT EYE CONSTRICT EQUALLY
(normal)

↓ swing light to left eye

LEFT EYE NO CHANGE OR SLIGHT CONSTRICTION **(normal)**	LEFT EYE DILATES **(abnormal)**

swing light back to the right eye

LEFT EYE NO CHANGE OR SLIGHT CONSTRICTION **NORMAL PUPILLARY RESPONSE**	RIGHT EYE CONSTRICTS **CONFIRMS LEFT AFFERENT PUPILLARY DEFECT**

EYE DISORDERS THAT CAN CAUSE AN AFFERENT PUPILLARY DEFECT

- Optic Nerve Disease
  - Ischemic Optic Neuropathy
  - Optic Neuritis (Retrobulbar or Intraocular)
  - Optic Nerve Tumor
  - Glaucoma
- Central Retinal Artery Occlussion
- Branch Retinal Artery Occlussion
- Central Retinal Vein Occlussion
- Branch Retinal Vein Occlussion
- A Lesion of the Optic Chiasm or Optic Tract
- Amblyopia
- Vitreous Hemorrhage
- Retinal Hemorrhage
- Macular Degeneration
- Retinal Detachment
- Other Organic Retinal Disease

clinical diagnosis of an afferent pupillary defect.

METHODS AND MATERIALS

The study consisted of a thorough evaluation of fifteen patients with a measurable afferent pupillary defect. Subjects were obtained over a six month period from the following offices: Battle Creek VAMC, Saginaw VAMC, Great Lakes Eye P.C., Grand Rapids Ophthalmology and Andersen Eye Center. Assessment of afferent pupillary defects were detected via the swinging flash light test using a transilluminator with a 3.5 volt halogen bulb. The illuminator was held at five centimeters from the eye and at an angle so as not to obstruct the patient's view of a distant target which would elicit an accommodative response or false pupillary constriction. The same instruments, methods of measurements, and end-point definitions were used by both observers during the study to ensure equal and accurate assessment with as little variation in measurement as possible. Once an measurable afferent pupillary defect was detected, the defect was quantified to the nearest 0.1 log unit by using neutral density filters ranging from .1 to 1.1 log units in .1 incriment steps.

The afferent pupillary defect was first evaluated under initial room lighting of approximately 70 footcandles. Light was shone to each pupil consecutively for a pause time of two to three seconds to allow observation of the pupillary response before quickly transferring the beam to the other eye, with a less than one second transfer time. Pause time, the amount of time the light is shone into the eye, and transfer time were kept consistent and equal to prevent assymetric bleaching of the retinal photoreceptor cells which would result in a pseudo afferent pupillary defect. Neutral density filters of appropriate log units were placed in front of the normal eye and the swinging flashlight technique was performed for three cycles until an end point was established. This end-point occurred when the pupils initial constriction and subsequent dilation were symmetrical.

After the afferent pupillary defect was measured in initial lighting, the room lights were dimmed to approximatley 10 footcandles and the pupillary defect defect was assessed again for 3 cycles as described above. The pupils were then assessed by varying the pause times from one second, five seconds, and ten seconds to evaluate the effect of stimulus duration on the afferent pupillary defect in dim lighting. The subjects were then allowed to dark adapt for 5 minutes and the defect was again quantified with neutral density filters and the swinging flash light technique, as previously described, for three cycles.

SUBJECTS

Fifteen patients were included in our study on the variability of afferent pupillary defects. These patients were collected from the following eye care providers and were prospectively tested after informed consent was obtained: Battle Creek VAMC, Saginaw VAMC, Great Lakes Eye P.C., Grand Rapids Ophthalmology, and Andersen Eye Clinic. The

mean age for these patients was 66.7 years, ranging from age 47 to age 81. Eight of these were male patients with a mean age of 67.1 years and seven were female patients with a mean age of 66.8 years. The most common diagnoses in our study, out of a total of eight, were secondary to central retinal vein occlussions (3) and primary open angle glaucoma (3). Other causes of afferent pupillary defects in our study included: retinal detachment (2), anterior ischemic optic neuropathy (2), age related macular degeneration (2), optic atrophy - unknown etiology (1), retinal hemorrhage with subretinal neovascularization (1), and central retinal artery occlussion - ischemic (1). Two patients were excluded from our study due to an afferent pupillary defect which exceeded our neutral density filter limit. There were no other exclusions of patients from our study.

RESULTS AND DISCUSSION

Since the presence of an afferent pupillary defect is essential in the evaluation of the severity of many diseases including optic neuritis, central retinal vein occlussions, central retinal artery occlusions, assymetrical glaucomatous damage, retinal detachment and hemorrhages, and ischemic optic neuropathy, it is important clinically to be able to assess a defect with as little variability in measurement as possible. The purpose of this study was to determine the effects that various lighting conditions and light stimulus durations have on the evaluation of an afferent pupillary defect. As evidenced from our study, lighting conditions play a significant role in the variablity of afferent pupillary defect measurements. If room lighting is not at appropriate levels, such as all or most of the overhead lights on, a defect may not even be detected.

From the results of our study, it is shown that evaluation of pupillary responses in bright or normal room illumination can lead to a non-detectable pupillary defect or a much less quanitification of a pupillary defect than when evaluated in dim light. This was true whether the defect was severe or subtle. As is shown in Table 1 of our results, an afferent pupillary defect neutralized with a .8 filter in dim lighting, was difficult to detect in bright lighting and was quantified as a .3 defect. Another patient had presented with an afferent pupillary defect quantified as .2 with the neutral density filters in dim lighting; however, the defect was undectable in bright illumination. Most likely, this variability is caused by constriction of the pupils in bright lights. This results in small pupils and a decrease in pupillary excursions in bright light making the evaluation difficult and inaccurate.

Our study found optimum lighting conditions for the most accurate assessment of pupillary response should be a dimmed room with just enough light to allow the observer to evaluate the pupillary response. When the lights were dimmed, the pupils were allowed to

RESULTS OF THE STUDY ON AFFERENT PUPILLARY DEFECT ASSESSMENT UNDER VARIABLE LIGHTING CONDITIONS AND STIMULUS DURATIONS
(TABLE 1)

	DX	LIGHTING CONDITIONS			STIMULUS DURATION		
MALES:		**INITIAL**	**DIM**	**DARK ADAPTATION**	**1S.**	**5S.**	**10S.**
73	OA	0.3	0.5	0.4	0.5	0.5	n/a
48	RD	-----	0.2	0.2	0.2	0.3	n/a
47	RD	0.4	0.7	0.7	0.6	0.6	n/a
72	POAG	0.8	1.0	1.0	1.0	1.0	n/a
68	AION	0.3	0.7	0.8	0.7	0.7	n/a
81	ARMD	0.2	0.5	0.6	0.5	0.4	n/a
81	CRVO	0.2	0.6	0.6	0.7	0.7	n/a
67	CRVO	0.3	0.6	0.6	0.6	0.6	n/a
FEMALES:							
50	POAG	------	0.5	0.5	0.5	0.5	n/a
67	RET HEM	0.2	0.4	0.4	0.4	0.4	n/a
62	CRVO	0.3	0.4	0.4	0.4	0.3	n/a
63	ARMD	0.4	0.7	0.7	0.7	0.7	n/a
78	POAG	0.4	0.8	0.8	0.8	0.7	n/a
70	AION	0.3	0.8	0.8	0.8	0.8	n/a
74	CRAO	0.5	1.0	1.0	1.0	1.0	n/a

FACTORS THAT CAN ALTER THE ASSESSMENT OF AN AFFERENT PUPILLARY DEFECT

Eye Glasses (with and without tints)
Eye Drops (mydriatic or miotic drops)
Medications (anticholinergics, decongestants, etc)
Accommodation or Fixation of Near Targets
Emotions
- Excitation
- Depression
- Fatique
- Drowsiness
- Alertness
- Anxiety

Uniocular Blepharoptosis
Poorly Reacting Pupils
Small Pupils
Assymetric Bleaching of the Retina
- Through Unequal Light Stimulation

dilate slightly making them bigger and easier to assess. It was also easier to assess the amount of pupillary movement seen while neutralizing the defect. Consequently, the more detectable the pupillary movement is, the more accurate the quantification is. An argument that a more accurate and consistent quantification of a pupillary response is performed in dim lighting can be made by Table 1. For example, a patient with end stage primary open angle glaucoma had an afferent pupillary defect which was quantified in bright lights as .4; however, in dim lights the defect was measured at a .8, which seemed to correlate better with the amount of damage caused to the optic nerve.

When measurements of the pupillary defects were taken after five minutes of dark adapatation, no significant changes were found. With the exception of two patients, the pupillary defect did not change after dark adaptation. The small change found while neutralizing the defect of those two patients is most likely attributable to a small variation in pupillary movement and a defect which was borderline. Limited by .1 log units, if a patient falls between a .7 and a .8, then the observer must make thier best assessment. For example, the 68 year old patient with anterior ischemic optic neuropathy measured a .7 in dim light and a .8 after dark adaptation. If rechecked it may be found that this patient's defect was actually between .7 and .8; however, because of the limitation of the filter density of .1 steps, the defect was quantified as a .7 in dim and a .8 after dark adapting. This can be referenced in Table 1.

Light stimulus duration or pause time was also evaluated. This is a essential aspect of grading afferent pupillary defects. After thoroughly investigating pause times of one second, five seconds, and ten seconds, our study showed that a stimulus duration of one and five seconds did not change significantly the quantification of an afferent pupillary defect. The ten second pause time, however, was inconclusive. When light was incident upon the eyes for ten seconds, a pupillary release was evident causing both pupils to dilate. This can cause the appearance of a pseudo afferent pupillary defect or alter the apparent severity of the pupillary defect. This release is most likely caused by sphincter muscle fatigue, which occurs after over-exposure of two to three seconds. This muscle fatigue results in a reflex dilation and a pseudo-afferent pupillary defect.[3]

Although our study did not show a significant clinical change in the assessment of the afferent pupillary defect when stimulus duration was varied between one and five seconds, interpretation of the swinging flashlight test tends to be more difficult when the stimulus duration is less than two seconds. Within the first two seconds of pupillary illumination, both constriction and dilation occur, thus the amplitude of pupillary movements is at its greatest.[3] This makes assessment of abnormal pupillary reactions more difficult to

detect. In addition, if one considers measurement error using a stimulus duration of one second, one eye or the other is very likely to receive unequal stimulation resulting in assymetric bleaching and a false grading of an afferent pupillary defect.

In our study, the clinical measurement of the pupillary defect did not change with reference to one and five second pause times; However, the examiners found a stimulus duration of five seconds represented the optimum pause time investigated. Stimulus duration of five seconds allows the examiner an additional four seconds to view pupillary excursions without changing the clinical grading of the defect. In addition, the amplitude of pupillary movements are reduced after two seconds, allowing an afferent pupillary defect to manifest. The above conclusions about pause times can be referenced in Table 1.

Summarizing, our study concluded when quantifying an afferent pupillary defect, the observer should be in dim room lighting while perfoming the swinging flashlight technique. An attempt at observation in normal room illumination can yield unreliable results. The flashlight should be directed into the eye for a period of five seconds with less than one second transfer time between the two eyes for optimum observation of pupillary response. Light stimulation of longer or lesser duration can lead to pseudo afferent pupillary defects or absence of a defect. Although this study used a small population of subjects, it was designed to provide enough evidence to differentiate the effects of variable lighting conditions and stimulus durations.

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