

Dark adaptation in retinal abnormalities

A common complaint is that of abnormally reduced vision in dark conditions. Whether this refers to visual acuity, glare, or other aspects of visual function must be determined in order to understand the cause of the complaint and if any alleviation of these symptoms can be considered. One aspect to investigate is whether the complaint refers to a delay in adjusting to a dark environment. This may reflect underlying retinal changes which result in slowed recovery of sensitivity following exposure to light. Of particular interest are the recent findings that patients with age-related macular degeneration and other retinal abnormalities can have severe delays in recovery of sensitivity in the dark¹⁻⁵ even with normal visual acuity and normal photopic and scotopic visual fields. These results suggest that some patients who have normal vision by conventional testing can have profound delays in recovery of sensitivity following light exposure and that this may explain the symptoms of reduced vision in dark conditions.

The paper by Baca and coworkers in this issue contributes to understanding some of the characteristics and delineates some potential underlying physiological mechanisms for this phenomenon. They measured dark adaptation curves following a period of light adaptation in six patients with Best vitelliform macular dystrophy (BVMD). They found that when they tested in the region of clinically normal appearing retina, all patients showed a normal time course of recovery. This was in contrast with their findings in Stargardt macular dystrophy where they had earlier found that all 12 patients tested had abnormally prolonged dark adaptation.⁶ This abnormality selectively involved the later portion of rod adaptation with normal rod-cone break times and normal early rod dark adaptation. This is of interest in light of the histopathological findings.^{7,8} It was suggested that accumulation of lipofuscin-like material within retinal pigment epithelial cells could contribute to this abnormality by impairing rhodopsin regeneration. Since a lipofuscin material accumulates within retinal epithelial cells in BVMD, this was suggested to reveal a fundamental difference in the underlying functional abnormalities. As originally emphasised by this group, the fully dark adapted sensitivities were measured before any bright light exposure in order to determine the true absolute thresholds uncontaminated by any abnormal kinetics of recovery.⁹ This is important because previous investigators had reported normal kinetics of dark adaptation in some forms of retinitis pigmentosa (RP) but this type of abnormality would have been missed without measuring fully dark adapted sensitivities.

Recently the characteristics of abnormal dark adaptation in different retinal abnormalities has furthered our understanding of some differences. For example, while in some forms of RP the kinetics of dark adaptation may be normal even in the presence of depressed final sensitivity, in other forms of RP with known rod opsin mutations, characteristic abnormalities in dark adaptation have been found which can selectively affect the late rod portion in the presence of normal cone and early rod dark adaptation.^{3,4,10} It has been found that patients with age-related Bruch's membrane change may have severe abnormalities in the rate of recovery

of dark adaptation despite normal final sensitivities in some cases. In a group of patients who reported poor vision in dim light but who had normal visual acuity, severe delays in the recovery of dark adaptation were found.⁵ This may be associated with prolonged choroidal filling on fluorescein angiography.^{5,11} In other cases of funduscopically visible change, however, normal rates of dark adaptation have been found.¹² Interestingly, severe delays in both rod and cone dark adaptation have been found in Sorsby's fundus dystrophy in regions of the fundus with visible yellow deposit at the level of Bruch's membrane.¹³ In other regions of the same fundus with ophthalmoscopically normal appearing retina dark adaptation can be normal. The rate of regeneration of rhodopsin in the corresponding retinal locations showed, similarly, changes in kinetics suggesting abnormal metabolic exchange across a thickened Bruch's membrane as the basis for the slowed dark adaptation.

A fuller understanding of the abnormalities of dark adaptation in different forms of retinal abnormalities may explain some symptomatic reports of reduced vision and may lead to better understanding of the underlying pathophysiological mechanisms. The relation between funduscopically visible structural changes and the manner in which dark adaptation abnormalities lead to later loss of visual acuity and visual field loss may provide earlier indications of pathology. These in depth investigations may lead to better understanding of the fundamental mechanisms of visual loss in common conditions such as age-related macular degeneration and provide a framework for devising new therapies.

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- Eisner A, Fleming SA, Klein ML, Mauldin MW. Sensitivities in older eyes with good acuity: eyes whose fellow eye has exudative age-related macular degeneration. *Invest Ophthalmol Vis Sci* 1987; 28: 1832-7.
- Eisner A, Stroumbos VD, Klein M, Fleming SA. Relations between fundus appearance and function; eyes whose fellow eye has exudative age-related macular degeneration. *Invest Ophthalmol Vis Sci* 1991; 32: 8-20.
- Wells J, Wroblewski J, Keen J, Inglehearn C, Jubb C, Eckstein A, et al. Mutations in the human retinal degeneration slow (RDS) gene can cause either retinitis pigmentosa or macular dystrophy. *Nature Genetics* 1993; 3: 213-8.
- Moore A, Fitzke F, Kemp C, Arden G, Keen T, Inglehearn C, et al. Abnormal dark adaptation kinetics in autosomal dominant sector retinitis pigmentosa due to rod opsin mutation. *Br J Ophthalmol* 1992; 76: 465-9.
- Steinmetz RL, Haimovici R, Jubb C, Fitzke FW, Bird AC. Symptomatic abnormalities of dark adaptation in patients with age-related Bruch's membrane change. *Br J Ophthalmol* 1993; 77: 549-54.
- Fishman GA, Farbman JS, Alexander KR. Delayed rod dark adaptation in patients with Stargardt's disease. *Ophthalmology* 1991; 98: 957-62.
- O'Gorman S, Flaherty WA, Fishman GA, Berson EL. Histopathologic findings in Best's vitelliform macular dystrophy. *Arch Ophthalmol* 1988; 106: 1261-8.
- Lopez PF, Maumenee IH, Cruz ZDL, Green WR. Autosomal-dominant fundus flavimaculatus. *Ophthalmology* 1990; 97: 798-809.
- Alexander KR, Fishman GA. Prolonged dark adaptation in retinitis pigmentosa. *Br J Ophthalmol* 1984; 68: 561-9.
- Kemp C, Jacobson S, Roman A, Sung C, Nathans J. Abnormal rod adaptation in autosomal dominant retinitis pigmentosa with Pro-23-His rhodopsin mutation. *Am J Ophthalmol* 1992; 113: 165-74.
- Chen JC, Fitzke FW, Pauleikhoff D, Bird AC. Functional loss in age-related Bruch's membrane change with choroidal perfusion defect. *Invest Ophthalmol Vis Sci* 1992; 33: 334-40.
- Holz FG, Jubb C, Fitzke FW, Bird AC, Pope FM. Dark adaptation and scotopic perimetry over 'peau d'orange' in pseudoxanthoma elasticum. [Letter.] *Br J Ophthalmol* 1994; 78: 79-80.
- Steinmetz RL, Polkinghorne PC, Fitzke FW, Kemp CM, Bird AC. Abnormal dark adaptation and rhodopsin kinetics in Sorsby's fundus dystrophy. *Invest Ophthalmol Vis Sci* 1992; 33: 1633-6.