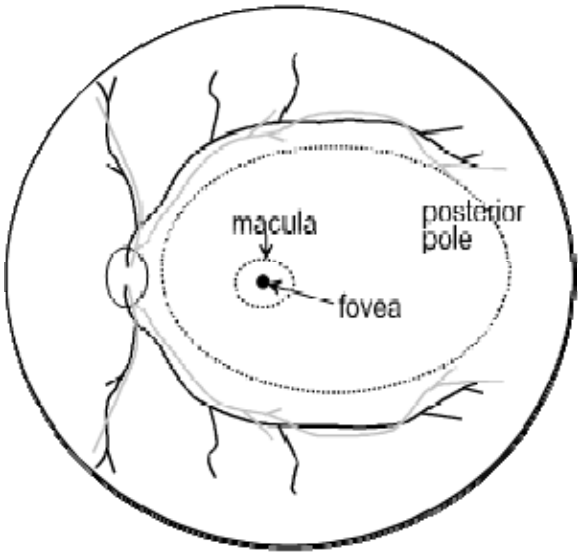
Macular Degeneration - the Essentials

Definitions

Macula: Area of the retina about 3 mm temporal to the optic nerve head. It is responsible for central vision, fine spatial discrimination and color vision.

Fovea: Central depression within macula containing only cones responsible for finest resolution sight.

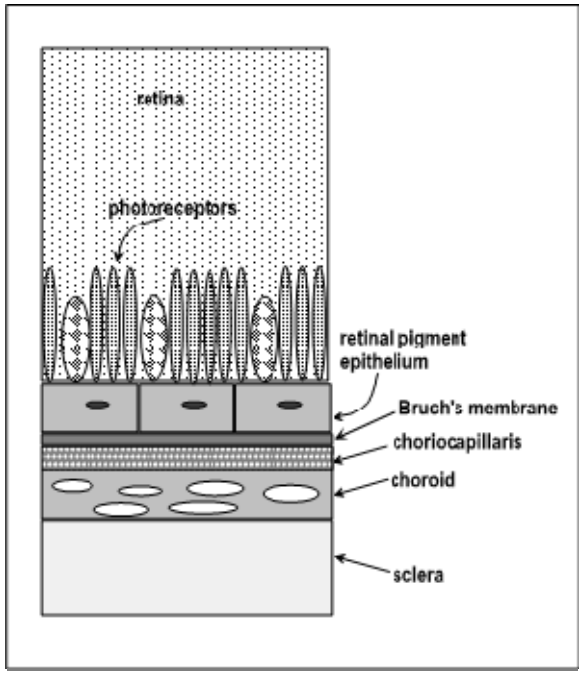
Age-related Macular Degeneration: loss of central vision due to age related changes in the macula.



Epidemiology

Leading cause of legal blindness in patients over 60. Affects 28% of people between ages 75 and 85.

Smoking has been shown to be positively correlated with ARMD.



Etiology/Pathophysiology: Aging results in a number of changes in the retina.

1) *Drusen* - localized deposits in *Bruch's* membrane - the layer between the *retinal pigment epithelium* (RPE) and *choroid*. They are classified by appearance into *hard* (small and discrete) and *soft* (irregular with indistinct edges) types.

2) Retinal Pigment Epithelium Changes - hyper and hypo pigmentation due to atrophy. The RPE serves a number of metabolic functions for the photoreceptors. When the RPE atrophies, photoreceptors lose function.

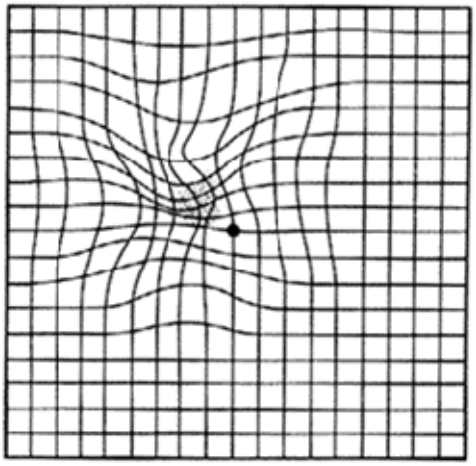
3) Weakening of Bruch's membrane.

The *dry* or *atrophic* form of macular degeneration is characterized by drusen and RPE changes. Visual acuity is variably reduced depending on the location and extent of the changes. Less commonly, a severe form of dry macular degeneration called geographic atrophy can result in total loss of central vision.

The *wet* or *neovascular* form of macular degeneration is characterized by extension of vessels from the choroidal circulation through the weakened Bruch's membrane forming a *choroidal neovascular membrane (CNVM)* or *net*. The new blood vessels leak fluid, lipid and blood under

the retina. This is turn leads to photoreceptor death, subretinal fibrosis and scarring and detachment of the retinal pigment epithelium. Visual loss in the wet form of macular degeneration is dependent on the location of the leaking vessels but is generally more sudden and more severe than the dry form.

Amsler Grid With Distortion



Symptoms: Patients with the dry form of macular degeneration may note gradual mild decrease in central vision and will occasionally notice distortion causing waviness of straight lines (*metamorphopsia*). Patients with the wet form of macular degeneration may notice sudden loss of central vision with a central blind spot (*scotoma*) and severe metamorphopsia.

Treatment: There is currently no treatment for the dry form of macular degeneration.

Until recently, wet ARMD was treated primarily with laser treatment aimed at sealing off and causing regression of the CNVM. The benefit from laser was small and patients often had permanent blind spots as a result of the treatment. This treatment is used much less often in current practice.

A newer treatment called photodynamic therapy (PDT) involves intravenous injection of a photoactivatable dye, verteporphorin, with an affinity for new blood vessels. Subsequent exposure of the CNVM to the correct frequency of light causes activation of the dye molecules and closure of the CNVM. When it works this treatment results in stabilization but rarely improvement in vision.

Currently, most patients with ARMD are treated with antibodies to vascular endothelial growth factor (VEGF). The anti-VEGF antibodies are injected into the vitreous cavity and cause regression of the CNVM with much less scarring than the older laser treatment. This newer treatment actually results in an improvement in vision rather than the slowing of visual loss or stabilization characteristic of older treatments.

Based on current data, patients with ARMD at any stage should be encouraged to stop smoking. A specific combination of vitamins A, E, C, Zinc and Copper has been shown to have some prophylactic value in patients with high-risk characteristics. The downside of this vitamin is minimal (except for patients who smoke for whom the risk of lung CA is increased) . The use of sunglasses in bright sun conditions has not been proved to be of clear prophylactic value but some ophthalmologists will recommend these to decrease chronic photic insult to the retina.

There are many claims that other vitamins and supplements are useful but none have yet been shown to be beneficial in well performed studies. As is often the case with chronic incurable diseases there will always be some practitioners who are willing and able to sell expensive treatments of no proven value.