AGE-RELATED MACULAR DEGENERATION

A Literature Review

Including the Results of a
Survey of Retinal Specialists

February 24, 1989

by

DANIEL G. IRWIN

WALTER BETTS, O.D.
Project Adviser

The virtual explosion in modern medical technology has had the effect of increasing human life expectancy in the United States by thirty years since the beginning of the century and continues to rise. While this is generally regarded as a desirable outcome, one should not overlook the fact that our ability to successfully treat the various disorders of the ageing body has not risen commensurately.[22] A prime example is ocular disease, namely age-related macular degeneration (ARMD), which will be the focus of this paper.

Age-related macular degeneration is the leading cause of irreversible severe blindness in the western world in people fifty years of age and older.[3] In the United States alone, of those between the ages of 65 and 75 years, at least 10% have lost some central vision as a result of age-related macular degeneration; older than 75, the percentage climbs to 30. The highest prevalence is for those 85 and older. The latter is also the fastest growing segment of the U.S. population and is expected to double in number within the next thirty years.[16,22] Indeed, by the year 2030, experts predict that the number of people 55 years of age and over will be equal to those under 19 years of age.[15]

The magnitude of age-related macular degeneration as a public health problem becomes readily apparent as one reviews the above statistics. The fact that the expansion of optometry as a primary care health profession is coinciding with the growth of this very serious public health problem poses even more of a challenge to optometrists as we approach the twenty-first century. If optometrists are to meet this challenge head on, a

good basic working knowledge of this debilitating disease is an absolute must. An awareness of the most recent literature concerning age-related macular degeneration will aid the practitioner in his or her effort to provide optimum patient care.

Loss or impairment of vision in the elderly is not readily compensated for by any of the other senses, since a decline in all five senses occur.[15] Those individuals at risk for potential vision loss must therefore be identified as early as possible to ensure proper management and follow-up care. Without doubt co-management, or more specifically the ability to make appropriate and timely referrals to ophthalmologists who will do any "treatment", promises to be the optometrist's basic responsility.

In keeping with this thought, this paper will concentrate primarily on the most recent reports in the literature pertaining to age-related macular degeneration. As alluded to above, emphasis will be placed on reporting the optometrists' current role in the management of these patients. The information presented subsequently is not intended to be a lesson on age-related macular degeneration. Rather, it will consist of a basic overview of the disease and highlight the current thought on the major signs and symptoms, and proven as well as possible future treatment of this disease. The latter portion of this paper will deal with the intriguing results of a survey of retinal specialists throughout Michigan, conducted in late 1988. One will assuredly find these results interesting, worthwhile, and practical.

As recently as 1939 age-related macular degeneration was considered an uncommon entity in the medical field. In fact, only 130 cases of the exudative form of this disease were reported in the literature.[21]

It was back in 1885, however, that the term "senile macular degeneration" was actually first coined. At this same time, it was Haab, a pioneer in this area, who first described the clinical observations of pigmentary and atrophic changes that occur with this disease in the macula. This was just the beginning, nevertheless, as the close relationship between macular drusen and disciform macular degeneration continued to be overlooked.[21] It was not until Gass, having performed his "monumental" study on disciform macular degeneration in the 1960's using the newly developed technology of fluorescein angiography, did our current knowledge of age-related macular degeneration begin.[21]

It is interesting that even with all the current knowledge of this disease, there is as yet no universally accepted definition of age-related macular degeneration. When describing this disease, authors tend to concentrate on the three main manifestations of the condition. Drusen, geopgraphic atrophy of the retinal pigment epithelium (RPE) and retinal changes related to subretinal neovascular membranes in individuals over 50 years of age are generally considered age-related macular degeneration.[3]

Bressler et al bring to light an interesting observation related to drusen. That is, since drusen are seen in more than

half the population over the age of 70, there are investigators who prefer to use the term "age-related macular degeneration" for those individuals who exhibit any of the manifestations listed above plus a degree of vision loss.[3] This makes sense when taking the word "maculopathy" into account (the condition is also known as age-related maculopathy). Indeed, the implication is that without manifested visual loss such findings, i.e. drusen, would simply be "age-related" and considered no more important than the onset of presbyopia.

When discussing the progression towards age-related macular degeneration, however, drusen do represent the earliest clinical sign.[3] Histopathologic studies have, indeed, shown drusen to be "associated with and a ... predisposing feature"[8] of ARMD.[4]

Young defines drusen as "extracellular masses of heterogenous composition situated between the basal lamina of the RPE and the inner collagenous layer of Bruch's membrane."[22] Generally patients have normal visual acuity and are asymptomatic. Patients with numerous drusen centrally will occasionally complain of problems reading noted most in dim light, and metamorphopsia.[3]

Currently, the lists of types of drusen is quite lengthy. Bressler, et al categorize them by their morphology: hard, soft, semsolid, basal laminar, and calcified drusen.[3]

The term hard drusen is derived from from their appearance. By ophthalmoscopy, they appear as small, round, flat, discreet yellow-white deposits. The pigmentary changes observed with hard drusen make the ophthalmoscopic detection easier. They may be

associated with overlying RPE hypopigmentation or adjacent RPE hyperpigmentation.[3]

Second, there are soft drusen, which by ophthalmoscopy are generally larger than hard drusen. They present with ill-defined, nondiscrete borders which can merge together forming large confluent areas of degeneration. Confluent soft drusen are thought to be histologically identical to serous detachments of the RPE.[4]

The clinical manifestations of confluent soft drusen and serous detachments of the RPE are varied, however. For example, serous detachments of the RPE are more translucent on biomicroscopic examination than are coalesced soft drusen. Also, with flourescein angiography, confluent soft drusen do not exhibit hyperflourescence at any point during flourescein angiography as do serous detachments of the RPE. Moreover, areas of confluent soft drusen are usually smaller with irregular borders and are more shallow than the classic serous detachment of the RPE. It is important to be able to distinguish between the two as areas of large, soft confluent drusen are more highly associated with subretinal neovascular membranes.[3] Third, there are semisolid drusen. These usually have diffuse borders like soft drusen but appear fairly flat like hard drusen. Sarks hypothesizes that hard drusen may actually progress to semisolid drusen and that this transformation can continue culminating in the formation of soft drusen.[3]

Fourth, there are basal lamina drusen. These present as

numerous, small, uniformly sized, discrete, slightly elevated yellow subretinal lesions. Histologically, basal lamina drusen are similar to Hassle-Henle warts and guttata that occur peripherally and centrally, respectfully, within the corneal endothelium. This type of drusen, unlike the hard, soft, or semisolid type are seen most often in young patients. Yellow exudative detachments, on occasion, have been associated with basal lamina drusen. Visual acuity is rarely affected significantly, even when the fovea is invloved. These detachments can be chronic or resolve. In addition, even though the appearance of these yellow serous detachments are similar to the lesion observed in vitelliform or Best´s disease, the appearance of basal lamina drusen indicate that this "vitelliform-like detachment" is not a manifestation of Best´s disease.[3]

The term "basal laminar drusen" should not be confused with "basal laminar deposits". The latter is a term used to describe the widespread thickening of Bruch´s membrane discerned in ARMD.[3]

Lastly, the term calcified drusen can be used to describe any of those drusen above that exhibit a shiny, glistening appearance. It is thought that they represent a "dystrophic calcification" within such lesions.[3]

The pathophysiology of drusen is a subject that has recently gained much attention when attempting to determine what exactly goes wrong in age-related macular degeneration, even down to the molecular level.

It is known that in age-related macular degeneration the primary lesion occurs at the level of the retinal pigment

epithelium (RPE).[22] Young believes that this is the result of the high rate of molecular degradation that occurs here. As an individual ages, cells of the RPE slowly accumulate sacs of molecular debris, or "residual bodies".[22] These "residual bodies" are actually lipofuscin material and are thought to represent remnants of incomplete degradation of abnormal molecules, which at some point have become damaged within the RPE or derived from damaged molecules of phagocytized rod and cone cell membranes. Over time, these cells enlarge and it is the eventual extrusion of this abnormal material that accumulates in Bruch's membrane, thus the manifestation of drusen and basal laminar deposits.[22, 23]

These "excretions" contribute to the deterioration of the RPE. Visual cells begin dying as a result of the RPE degeneration. Indeed, the primary event of age-related macular degeneration is the deterioration of the RPE. All else occurs secondarily: death of photoreceptors, and the choroidal vasculature, deposition of abnormal material within Bruch's membrane, drusen, pigment mottling, and basal laminar deposits. Hogan has stated that "'the gradual failure of the function of the retinal pigment epithelium is the principal cause of many forms of degenerative macular disease, especially the senile ones.'"[22]

Current knowledge again comes back to the hypothesis that the RPE degeneration is a result of imperfections in the cells' digestive mechanism. The latter causes abnormal molecules to accumulate within the RPE, interfering with the cell's normal

metabolism, initiating the "excretions" mentioned above.

There is currently, however, no evidence that inherited defects in digestive enzymes contribute to the development of age-related macular degeneration. It is believed that when molecular degradation is incomplete, it is because the substrates of a cell's enzymes are abnormal rather than the result of any aberrant enzymes. Supporting this theory is the knowledge that the abnormality of these molecules is not evident until after they have been synthesized.[22]

The above signs discusses thus far, e.g. drusen, Bruch's membrane changes, and basal laminar deposits, are all discerned in the elderly, but to a lesser degree. That is, the major signs of age-related macular degeneration are seen in the elderly, but only in certain individuals do these develop in cell death and severe vision loss.[22] This brings one back to the basis for Bressler et al definition of age-related macular degeneration with vision loss being an important criterion.

Young, nevertheless, puts forth an interesting theory regarding age-related macular degeneration. He believes that the disease may simply be an advanced stage of what he calls "normal ageing" and that as one ages, the probability that cell death will occur increases. Senescence and age-related macular degeneration can therefore be considered elements of a continuum in which one enters the disease state when vision loss is noted.[22]

It would seem then that biological renewal is the main anti-senescence process. This process is, of course, very complex and will vary in different individuals. An important

factor here is genetic variance, but perhaps no less important as a factor in influencing the rate of senescence is the environment.

Environment as a factor in influencing the development of age-related macular degeneration has been thoroughly probed in a recent article by Young.[23] Specifically, he discusses the the relationship between solar radiation and retinal deterioration.

Experimental studies show that the region that deteriorates in age-related macular degeneration - the outer layers - is the same region that is damaged by bright light. Young feels that this is more than coincidence, as he states: "It is a remarkable and provocative empiracal law that **the retinal damage produced by bright light is most severe in precisely the location which deteriorates most rapidly in age-related macular degeneration.**"[23]

It is known that high-energy visible and ultraviolet radiation are capable of producing molecular damage via photochemical means.[23] The theory is that the abnormal molecules that accumulate in the cells of the RPE are actually in large part the result of photochemical damage to rods and cones. To reiterate briefly what was discussed previously, it is these abnormal molecules that accumulate within the RPE cells, extrude into Bruch's membrane and aggregrate in the form of drusen and basal laminar deposits. In addition, the photochemical damage is worsened by the presence of oxygen which initiates free-radical chain reactions.[23] More specifically, the photochemical changes may include formation of the superoxide

free radical. The latter is intrinsically unstable and can initiate chains of lipid peroxidation. This takes on much more importance when considering that the rod and cone outer segments consist of a dense stack of about 2000 layers of phospholipids. Fatty acids are concentrated within these phospholipids, where the latter demonstrate a high concentration of oxygen.[21,23] Oxygen flow is very high in the photoreceptor -RPE complex; indeed, it is higher here than any other site in the body.[23]

According to Young's theory, then, both ocular melanin and a cataractous lens should protect the retina against age-related maculopathy. In fact, various studies have shown this to be the case.

Laboratory studies have shown that the presence of ocular melanin apparently plays a role in protecting the retina from developing age-related macular degeneration. Dilation of the pupil has been shown to reduce the threshold for retinal damage in pigmented rats, indicating that iris melanin is important in reducing irradiance on the retina. Several studies have also demonstrated that blacks contain more melanin in ocular tissues, such as the iris and choroid. Clinical observations and case studies have shown age-related macular degeneration to indeed be more prevalent in whites than in non-whites.[20, 23] This would account for the description of the disease in the past as "'a blue-eyed white man's disease.'"[23]

In one particular study, there was a statistically significant trend for lightly pigmented irides to develop signs of age-related macular degeneration sooner than those with darker irides. The authors' conclusion of this study was simply that

increased ocular pigment decreases the risk of developing the disease.[23] Implicit in these authors conclusion was the factor of genetics. This apparent connection between ocular pigment and development of age-related macular degeneration may help account for what Young terms "the tendency of age-related maculopathy to occur in families, since ocular pigmentation is an inherited genetic factor".[23]

The issue of a genetic factor in the development of ARMD was recently addressed in a clinical study - allegedly the first of its kind - that investigated the occurrence of ARMD in identical twins, determined to be so by genetic testing. The study does mention one report of "'identical looking'" twins with ARMD. No genetic testing, however, was performed to confirm whether they were indeed monozygotic.

In the report, this pair af identical twins reportedly lost central vision within three years of each other. A family history revealed no signs of ARMD in their nine children age 51 to 62. By history, as told by the twins, four out of eleven of the twins' siblings had severe ARMD, and their mother reported having lost reading vision in her seventies, had good peripheral vision, and was told by an ophthalmologist that surgery would do no good. Ocular history of the twins' grandparents, cousins, aunts, and uncles is simply unknown. Lastly, their father, who lived to be 75, did not exhibit the condition.[13]

In hypothesizing the mode of inheritance, the authors determined the liklihood for dominant inheritance to be 51.3%, assuming the father was not a carrier.[13] In their conclusions,

however, the investigators state that this clinical study only suggests the possibility that indeed genetics plays a larger role in the development of ARMD than was previously thought. As with most aspects of this disease, further research is necessary.

Regardless of one's genetic composition, in his article Young also offers several protective measures that supposedly will at least decrease one's chance of developmenting ARMD. Anti-oxidants, such as alpha-tocopherol (Vitamin E), ascorbic acid (Vitamin C), and beta-carotene (Vitamin A) which all must be obtained from one's diet are listed as agents that limit the potentially damaging free-radical chain reactions.[23] The question of whether supplementing one's diet with one or more of these agents can delay the onset or even diminish the severity of age-related macular degeneration has not been adequately investigated. Young points out, nevertheless, that animal investigators have shown that maximizing the retina's defenses against free-radical chain reactions **should** limit the "extent" of photodynamic molecular damage.[23]

Perhaps the more practical measure includes the use of protective sun lenses. For maximum protection, lenses should absorb all blue, violet, and ultraviolet radiation, or all radiation below 510 nm. (at least to 1% or less transmission).[23] No studies have tested this proposition, however. Young is quick to state that lenses with the above absorptive characteristics are in fact currently available, even though most commercial sunglasses fall short of meeting these specifications.[23] Young will supply a list of manufacturers upon request.

While the naturally occurring ocular melanin appears to

offer some protection from the damage to the retina that visible radiation can incur, lipofuscin, is a different story. Unlike melanin, lipofuscin is a heterogenous aggregation of damaged molecules, rather than a naturally occurring substance that is genetically programmed. In addition, unlike melanin, pathways for dissipating energy of electron excitation are not confined within the lipofuscin molecule. Indeed, absorption of near-UV radiation is re-emitted throughout the cytoplasm of the RPE cells at the blue and yellow-orange wavelenths. Consequently, it is thought that there may be many uncontrolled side effects of these reactions. This all becomes quite significant when considering the senescent retina. Within the latter, lipofuscin may exceed melanin by amounts of five to ten times [23]. In addition, it is interesting to note that in the absence of disease, lipofuscin first becomes apparent within the RPE by 10 years of age. By 40 years, the number rises to 8% of the cytoplasmic volume of the cells, and by 80 years, this figure rises to over 20%. [12]

When making the connection between melanin protection against radation damage and the formation of lipofuscin, one would think that lipofuscin, or the formation of "residual bodies" would be less in blacks than in whites. Indeed, this was recently confirmed. [23]

What then about cataractous lens protection? In various studies, those individuals suffering from age-related macular degeneration exhibited clearer lenses than those without the disease. [23] The theory is that radiation absorbed in the lens

cannot reach the retina and possibly harm it. Indeed, the yellowish pigments of nuclear cataracts absorb the high-energy blue and violet photons which are known to be especially damaging to the retina. Furthermore, a prelimary report suggests that there are significantly more signs of age-related macular degeneration (e.g. drusen and pigmentary changes) in the aphakic eye as compared to the phakic eye in individuals who have had unilateral cataract surgery.[23]

Of great concern to researchers and practitioners alike is not only the events that potentially lead to this disease, but the severe vision loss that can occur with age-related macular degeneration. It is generally accepted that there are primarily two forms of the disease: geographic or areolar atrophy, and neovascular or exudative macular degeneration, also known as the dry and wet forms of the disease, respectively. Of course, both can cause a devastating loss of vision.

Patients with geographic atrophy usually present with one or more areas of atrophic RPE in addition to drusen. Ophthalmoscopically, one can discern a concurrent atrophy of the choriocapillaris as well. As has already been established, areas of geographic atrophy are usually accompanied by a loss of overlying photoreceptors, since the latter are metabolically dependent upon the RPE. This, of course, is responsible for the vision loss that occurs in these cases. The choriocapillaris as well undergoes atrophy as a result of the loss of RPE. Support for this hypothesis comes from laboratory studies in rabbits. When the RPE of these animals were selectively destroyed, the choriocapillaris subsequently atrophied.[3]

Geographic atrophy may occur in one or both eyes. Few studies have, however, focused on the nonexudative stage. One retrospective study did determine that patients with bilateral drusen are at some risk for developing exudative maculopathy (8% within three years).[3] Nevertheless, Bressler et al believe that future prospective studies are necessary to investigate the incidence rates for those individuals who progress from the nonexudative to the exudative stage of the disease.[3]

As alluded to above, the bulk of the research has focused on the exudative form of age-related macular degeneration. The main reason for this is that the majority of patients experiencing severe vision loss do so as a result of developing the exudative stage of the disease. The Framingham Eye Study found that the nonexudative form of age-related macular degeneration accounted for about 80% of their cases whereas the exudative form of the disease were only 8%. Yet, of all the eyes in the Framingham Eye Study with age-related macular degeneration who exhibited vision of 20/200 or worse, 79% had the exudative form of macular degeneration. Other studies have also supported the findings of the Framingham Eye Study.[3] The conclusion to be drawn from these studies then is that while most patients with age-related macular degeneration do not progress beyond the nonexudative stage of the disease, those who do go one to develop the exudative stage are at the greatest risk for developing severe vision loss.

Indeed, those patients who experience severe central vision loss from age-related macular degeneration usually do so from the development of neovascularization and related exudative

processes. These include choroidal neovascular membranes (CNVM), serous or hemorrhagic detachment of the RPE, vitreous hemorrhage, and the culmination of these processes, the fibrovascular disciform scarring.

The exudative feature that has perhaps garnished most attention is the choroidal neovascular membrane (CNVM), also known as a subretinal neovascular membrane (SRNVM). When viewed ophthalmoscopically, CNVM appear round to oval and have a greenish-gray color.[3] Associated findings may also include subretinal lipid exudate, subretinal blood, detachment of the sensory retina, and even a portion of an ophthalmoscopically visible CNVM.[3, 18]

When an individual, who already exhibits signs of age-related macular degeneration, presents with a sudden or recent loss of central vision, which would include blurred or distorted vision or a central scotoma, the practitioner should immediately suspect that a neovascular process is occurring. A flourescein angiogram should be obtained as soon as possible to determine if first, a CNVM exists, and second, if it is treatable.

A complete understanding of the pathogenesis of choroidal neovascularization is currently unknown. Experts generally agree that they are largely the result of the thickening of the inner portion of Bruch's membrane in conjunction with soft drusen which presumably predispose Bruch's membrane to develop breaks through which new vessels from the choriocapillaris can invade. This hypothesis is firmly supported by the neovascular findings in other retinal diseases such as pathologic myopia, angioid streaks, and presumed ocular histoplasmosis syndrome, where

breaks in Bruch's membrane are observed.[3]

There are, however, two findings that indicate there must be a stimulus other than a break in Bruch's membrane initiating choroidal neovascularization. First, breaks in Bruch's membrane have been observed in eyes with age-related macular degeneration where no neovascularization can be identified. Second, studies have shown that endothelial cells have all the enzymes necessary for the digestion of a basement membrane such as Bruch's. This interesting finding would indicate that it is possibly the endothelial cells of CNVMs that actually produce the break in Bruch's membrane.[3]

Choroidal neovascular membranes that are associated with age-related macular degeneration tend to lead to greater macular damage than those associated with other pathologies for what Bressler et al cite as three reasons. First, CNVMs associated with ARMD tend to be located under the center of the foveal avascular zone (FAZ), thus ruling out treatment in most cases. The FAZ is variable, but has been determined to be generally 0.4 to 0.6 mm in diameter. This corresponds to an area slightly larger than the foveola as defined by the absence of rods.[4] Second, subfoveal CNVMs tend to be larger when initially detected. And third, the latter may be true because the underlying pathology of ARMD is diffuse rather than focal as it is, for example, in presumed ocular histoplasmosis syndrome.[3]

The pathogenesis of CNVMs in ARMD is, in practice, of secondary importance since the flourescein angiographic appearance is critical in the management and treatment of

patients exhibiting CNVMs. Choroidal neovascular membranes can be divided on fluorescein angiography into two groups: those that are poorly defined and those that are well-defined, where the entire extent of the membrane can be determined from the angiogram. This is important since clinical trials have shown benefit only in treating those CNVMs for which the entire extent is known since adequate treatment entails the photocoagulation of the membrane in its entirety.[3]

Current photocaogulation treatment of CNVMs is excluded when the membrane is poorly defined or when it located within 200 microns of the FAZ. When one considers these limitations, the numbers of successfully treated patients is actually quite small. To illustrate this, 50% of all CNVMs associated with ARMD present as poorly defined and of those well defined CNVMs, 50% are located with 200 microns of the foveal center. As one can readily discern, only about 25% of CNVMs associated with ARMD are treatable by the ophthalmologist.[3]

The size of the CNVM also is apparently related to the visual prognosis in eyes with ARMD. A recent study found that the larger the CNVM at initial observation, the lower the initial visual acuity. In general, it was found that even with relatively good entering visual acuities, patients with a subfoveal CNVM exhibited a poor visual prognosis.[10]

The fundamental principle of treating choroidal neovascularization is that of laser photocoagulation. Such treatment for ARMD has been shown to be effective for only a small percentage, as previously mentioned, of patients who present with a well-defined CNVM that is located more than 200

microns from the center of the FAZ. These criteria stem primarily from the results of a randomized clinical trial - a part of the Macular Photocoagulation Study (MPS) - that looked at the use of blue-green argon laser photocoagulation for ARMD.[2] The MPS recently published a three-year follow-up study indicating that treating extrafoveal CNVMs with blue-green argon laser photocoagulation is still beneficial. The authors, by studying consecutive visual acuities measurements, concluded that blue-green argon laser photocoagulation delays severe loss of vision is ARMD for an average of 18 months.[1]

The question remains as to what to do with patients who present with CNVMs that either are not well-defined or are subfoveal.

The factors that may make a CNVM difficult to define on angiography may include leakage that is poorly defined or blocked in part by possibly a serous detachment of the RPE , subretinal fluid, or turbid blood or pigment. The failure to identify a CNVM in its entirety precludes any treatment, since this may lead to incomplete photocoagulation. In addition, it must be established that the CNVM is located at least 200 microns from the center of the FAZ, since photocoagulation treatment has been substantiated only for such cases.[3]

The visual prognosis for patients with subfoveal CNVMs is also not very good. In one retrospective study, development of a CNVM within the FAZ was most often associated with a poor visual outcome where approximately 70% of affected individuals showed a visual acuity of 20/200 or worse.[3] It is important to note also

that of all untreated extrafoveal CNVM, nearly 75% eventually extend under the center of the FAZ.[3]

The use of krypton red photocoagulation has shown some promising results in the treatment of subfoveal membranes. The principle behind the use of krypton red laser photocoagulation is based on the fact that such light (647nm.) is minimally absorbed by foveal xanthophyll and oxy-hemoglobin, but is maximally absorbed by melanin. Another advantage of red krypton in many cases is that this wavelength traverses a nuclear sclerotic lens better than the other available wavelengths.[18]

Decker et al in their study - which was also part of the Macular Photocoagulation Study Group - studied treatment of subfoveal CNVMs by red krypton laser by placing them into groups: I - juxtafoveal CNVMs which were considered to be 1-250 microns from the center of the FAZ, and II - subfoveal CNVMs which were located under the center of the FAZ. The percentage of eyes that improved by two lines or at least did not worsen was 62% (16 of 26) for group I, and only 28% (7 of 25) for group II. It was further noted that in seventeen of the 25 eyes treated in group II, closure of the neovascular membrane was achieved. In this same group, in five eyes vision either improved or remained unchanged; yet no eye maintained a post treatment acuity of better than 20/100.[5]

With these findings in mind, these researchers conclude that treatment involving the subfoveal area should be restricted to eyes that show a pre-treatment acuity of worse than 20/100. The authors also conclude that although not all the eyes treated in this study improved or stabilized, a

majority did. In addition, treatment of juxtafoveal CNVMs seems to yield better results than the natural course. Given the poor visual prognosis of CNVMs located inside, but not under the center of the FAZ, treatment "seems justified".[5]

A recent clinicopathologic study reported findings in a case of ARMD with a CNVM treated with krypton laser photocoagulation in an eye with a posterior chamber intraocular lens. The findings included, contrary to previous studies, a full-thickness retinal scar secondary to retreatment with a krypton laser. Although this retina exhibited myopic thinning and was attenuated from previous laser treatment, these researchers conclude that an implication to be drawn from this histopathologic study is that krypton red photocoagulation can cause a full-thickness retinal scar" and "must be considered when retreatment is undertaken in the macula."[9]

Bressler et al discuss three potential complications that may accompany treatment with krypton red laser phtocoagulation. It is thought that each may be related to increased absorption in the choroid of the light energy due to a lack of absortion of red light in the inner retina. The first of these complications is choroidal hemorrhage during treatment. The second is delayed perfusion of choroidal vessels. Due to the rapid recovery of normal choroidal perfusion, it is thought that a vascular spasm rather than vascular destruction occurs. This has been noted several times with no permanent loss of vision. The third complication that may occur is a tear of the RPE.[3]

Further research is currently being conducted by the

Macular Photocoagulation Study Group of juxtafoveal and subfoveal CNVMs which should yield valuable additional information.[3]

Following photocoagulation of a CNVM associated with ARMD, an individual is still to be monitored closely given the high likelihood of developing recurrent neovascularization. In the MPS of extrafoveal CNVMs, recurrent neovascularization occurred contiguous to the photocoagulation scar in 53% of the successfully treated eyes and independent to the scar in 8% of the eyes.[3, 17] Overall, 43% and 52% of the eyes suffered a recurrence after one and two years, respectively.[17] After two years, recurrence was found to be rare if it had not occured already. In addition, as might be expected, eyes with recurrence had a worse visual acuity than eyes showing no recurrence.[17] Despite the recurrences noted in treated eyes in the MPS argon blue-green group, it should be noted that treated eyes exhibited better visual acuity outcome on average than did untreated eyes.[19]

In the MPS discussed above, cigarette smoking was the only significant risk factor for CNVM recurrence. Other documented risk factors include CNVMs located closer to the center of the fovea on initial presentation and very lightly pigmented CNVMs. Currently, predictions as to which eyes will suffer a recurrence cannot be made accurately.[3] It is therefore suggested that careful monitoring of patients who have received argon blue-green laser photocoagulation of a CNVM be implemented, particularly in the first year following the treatment. The MPSG advises Amsler grid testing and routine retinal evaluation, as well as fluorescein angiography as needed.[17]

Laser treatment, when discussed with a patient, should be described as a method of arresting further vision loss rather than a method of improving vision. The goal of such treatment, it should be stressed, is to completely obliterate the CNVM while minimizing laser damage to the fovea. In addition, it is important to explain that a central field defect that already exists due to a CNVM will be larger after treatment, even if the membrane is successfully elimated.[18]

The occurrence of serous detachments of the RPE is also a complication many patients with ARMD experience. On ophthalmoscopy, the appearance of a serous detachment of the RPE is that of "...a round or oval, yellow-orange, sharply demarcated mound."[3] The appearance comes from the well-demarcated dome-like blister that occurs when the firm attachment of the RPE to Bruch's membrane becomes compromised due to the diseased RPE-Bruch's membrane complex. With prolonged detachment of the RPE, the physiologic pumping mechanism of the RPE cells becomes compromised allowing fluid to enter the subretinal space. The development of a sensory retinal detachment then occurs. The normally loose adherence of the sensory retina to the RPE provides little resistance to the spread of subretinal fluid allowing the sensory retinal detachment to extend beyond the border of the RPE detachment.[3]

It has been shown that among patients under age 55 who present with a pure serous detachment of the RPE, the visual prognosis is excellent with 90% showing no loss in vision. In addition, "almost none" of these patients developed any

neovascular complications.[3] Patients age 55 and over with serous detachments of the RPE and drusen, however, exhibit a significantly higher risk of neovascular problems and loss of vision.

Bressler et al are quick to point out, nonetheless, that presently the benefit from photocoagulation treatment for serous detachments of the RPE is not clear. Current information suggests that the prognosis for treatment of serous detachments of the RPE in patients with ARMD, regardless of age, with photocoagulation is no better and is possibly worse than the prognosis for the natural course of this entity.[3]

It should be reiterated here that in many cases when a CNVM is difficult to define it is due to a serous detachment of the RPE. In such cases treatment is usually ruled out. When treatment of a CNVM is possible, however, and there is a treatable serous detachment of the RPE, photocoagulation should be viewed with caution given the severe risk of a tear of the RPE. The latter can occur spontaneously as well. Regardless, the location and extent of a tear are unpredictable and may result in a drastic reduction in visual acuity if it involves the fovea.[3]

When a CNVM does exist, a secondary complication that can arise when bleeding occurs is a hemorrhagic detachment of the RPE. Just as with a tear of the RPE, vision loss will occur if the detachment involves the fovea.[3]

A hemorrhagic detachment of the RPE appears as a dark green or red, elevated mound when the blood is confined to the subretinal pigment epithelial space. In fact, a hemorrhagic

detachment of the RPE is sometimes mistaken for a choroidal melanoma. The two can be differentiated by the observation that on fluorescein angiography, a hemorrhagic detachment of the RPE blocks choroidal flourescence, while a choroidal melanoma will generally exhibit some hyperflouresence within the lesion. In addition, the presence of drusen in the fellow eye is helpful in making a correct diagnosis.[3]

If the blood from the hemorrhagic detachment of the RPE is able to "dissect" through the edge of the detached RPE, the color of the lesion will appear as red since the blood is able to reach into the subsensory retinal space. When this occurs, blood may even break through the sensory retina resulting in a vitreous hemorrhage.[3]

When a vitreous hemorrhage occurs, the most frequent symptom is a sudden loss of vision. Some patients, however, will report severe pain followed by vision loss. Pain is experienced due to the distortion of the numerous nerve fibers within the choroid when the hemorrhage occurs.[3]

A vitreous hemorrhage secondary to a CNVM resolves in about 75% of patients. Eventually, the posterior pole can be viewed and neovascular ARMD can be confirmed. Any patient, however, with a vitreous hemorrhage in one eye and signs of ARMD in the other should be suspected of having a CNVM in the eye with the vitreous hemorrhage, and further testing is warranted to rule out conditions such as rhegmatogenous retinal detachment, choroidal melanoma, and other less frequently encountered causes of vitreous hemorrhages.[3]

When choroidal neovascularization goes untreated, the ultimate result is the formation of a fibrovascular disciform scar that replaces most of the outer sensory retina and RPE that rest between the retina and choroid. The lesion may vary in color from "white to yellow to brown to black" depending on the extent of the retinal pigment epithelial hypertrophy.[3] Of course, once a disciform scar has developed, photocoagulation treatment is of no proven benefit. Fluorescein angiography is warranted only to differentiate between a CNVM and scar in the event there is some doubt.[3]

By the time an individual with ARMD reaches the point of exhibiting disciform scarring, patient questions surrounding the etiology of this disease will almost certainly have been raised. Aside from those concerning treatment, questions regarding etiology of ARMD are indeed perhaps the most commonly asked by patients. Dr. Weiter, writing in Archives of Ophthalmology, believes that not every person who is at risk for developing the disease will do so because it, ARMD, is most likely the result of other independent, yet-to-be determined factors. The bulk of his editorial focuses on a possible nutritional factor, namely the Newsome et al study of using oral zinc in ARMD.[21]

It is known that metabolically zinc plays a very important role in several enzyme systems of the chorioretinal complex.[3,14,21] This is the reason that Newsome and his fellow colleagues undertook a prospective, randomized, double-masked, placebo-contolled study. Visual acuity outcome was observed in 151 individuals with drusen or ARMD who were administered oral

zinc. While this is the first controlled oral intervention study of this kind, the results revealed a positive, although limited, effect in ARMD. The study consisted of follow-up of 12 to 24 months. Some eyes in the zinc treated group lost vision. Overall, however, this group had "significantly less vision loss" than the group given placebos.[14]

This proposed relationship between oral zinc and "less vision loss" takes on greater significance when considering the essential role zinc plays in human metabolism. Zinc is the second most abundant element in the body and the most common in the eye.[14] Zinc, in fact, plays a major role in the metabolism of the retina. The RPE-choroid complex has the highest concentration of zinc of anywhere in the body. In addition, since the RPE has a very high metabolic rate it is not inconceivable that zinc may play an important role here.[21]

Furthermore, it has been shown that zinc is deficient to various degrees in at least some groups at risk for the development of macular degeneration. A survey conducted nationally by the U.S. Food and Drug Administration found that individuals 60 and older were likely to have a diet deficient in zinc, as well as other nutrients.[14] Newsome et al also comment on the interesting observation that females were found to be more likely to be zinc deficient while at the same time exhibiting a "reported preponderance in macular degeneration."[14]

Newsome et al, nevertheless, are quick to point out that it

is "definitely premature" to advocate the widespread administration of oral zinc since there can be potentially serious side effects such as anemia, and worsening of cardiovascular disease.[14] Dr. Weiter is in complete agreement with this cautionary tone as well as the statement put forth by Newsome et al that the issue of using oral zinc for ARMD definitely warrants further investigation.

In reading the article by Newsome et al, this reviewer could find no reference to the method with which visual acuity was determined. One might assume that Snellen acuity was utilized. A recent study, however, investigates contrast sensitivity in ARMD and suggests that the method used in assessing visual acuity may play a role in determining the results.

More specifically, in the study mentioned, low-contrast charts were used to test individuals with drusen in an effort to determine if visual deficits exist that may not be discernible with standard Snellen charts. These researchers compared performance on Regan letter charts* between 52 eyes with drusen and Snellen acuity of 20/20 and 27 control eyes. Those with drusen read fewer letters than the control group on all the charts used. This difference increased as the contrast of the charts was decreased. In addition, the loss of acuity correlated with the severity of drusen.[11] This was, however, only one aspect of this study.

Twenty-one eyes with 20/20 Snellen acuity and drusen were

* Regan letter charts are similar to Snellen charts except that the test letters are available in varying degrees of contrast.

also tested with the Ginsberg contrast sensitivity chart** and compared with age-matched normal controls. A loss of both contrast sensitivity in the higher spatial frequencies and peak contrast sensitivity were associated with increasing drusen.[11]

These results suggest that patients with drusen may indeed have a vision deficit even though they may be able to read 20/20 on a Snellen chart. With this in mind, the authors suggest that contrast sensitivity testing may be useful in measuring acuity loss in those patients with drusen. The latter statement becomes significant when realizing that it has been found that a loss in contrast sensivity - at least in part more efficiently detected using the low-contrast Regan letter or Ginsberg contrast sensivity charts - has more influence on the mobility of the low vision patient that does a loss of acuity.[11]

The astute observer would discern a contradiction between what the authors of the study concerning contrast sensivity in ARMD and the definition put forth by Bressler et al previously. Bressler et al purport ARMD to be the sum of drusen **and** a measurable vision loss. Yet the study looking at contrast sensitivity found a measureable vision loss - using methods other than Snellen acuity - in those patients with drusen only. Perhaps in future reviews this discrepancy will be reconciled.

When an individual enters an optometrist's exam room, the

** A Ginsberg contrast sensitivity chart consists of printed sine wave gratings with orientation either vertical or titlted to the right or left.

signs and symptoms leading to the early detection of ARMD can be numerous and varied. Indeed, the importance of early detection of ARMD cannot be overstated.

With the advent of successful - albeit limited - treatment of exudative ARMD with laser photocoagulation, early detection of neovascular processes increases the chances of identifying patients with a CNVM that is in a potentially treatable position.[6] Patients with early stages of ARMD , those for whom one eye has already been impaired by the disease, those at risk for developmenting the disorder (i.e., drusen in the macula), as well as post-laser treatment patients, should evaluate their central visual fields daily with the Amsler grid performed monocularly.[4,18] Should their be a disturbance on the Amsler grid, or a decrease in distance or reading vision, an eye exam should be performed at the earliest feasible time, again, in an effort to detect potentially treatable neovascularization membranes as early as possible.[6]

A patient´s history of visual loss and symptoms is very valuable information in raising suspicion of a CNVM. Patients with neovascularization will usually present with a sudden loss of vision whereas those with the the dry form of the disease will complain of a gradual loss of vision over a period of months.[7] Other critical symptoms include metamorphopsia - the distortion of lines - and micropsia - the minification of objects. These symptoms can be elicitied by history or Amsler grid testing.[4, 7]

An asymmetry in Snellen acuity between the eyes that is difficult to explain should also alert the practitioner to view

more carefully the poorer eye. Likewise, a patient exhibiting a deterioration in vision (i.e., 20/25 to 20/80) should raise suspicion of a possible CNVM.[7]

For those patients successfully treated by laser photocoagulation, daily self-testing with an Amsler grid is mandatory due to the possibility of a recurrence of the neovascular membrane. In the absence of any symptoms, an ophthalmologic exam should be performed every six months.[4]

At some point, an optometrist may find him or herself confronted with a patient who is suffering from the geographic form of the disease, or an untreatable CNVM. At this point, it is important to reassure the patient repeatedly that loss of central vision does not progress to total blindness. It should be emphasized that patients with ARMD rarely lose their peripheral vision and can be helped to retain - or regain - their independence.[4]

Patients who experience irreversible loss of central vision should be advised about the availability of the various low vision devices. The practitioner should either prescribe the necessary devices or refer the patient to one who is qualified to do so.[3, 4]

A note of caution is perhaps in order with regard to low vision devices. The success of any such instrument is highly dependent on several factors, namely patient motivation, and should not be looked upon as treatment for ARMD. It should be stressed that the patient still retains **usuable** vision and every effort made to improve their quality of life.

The extreme complexity of ARMD mandates that the eye care

practitioner know not only the obvious late changes of this process, but the subtle early signs. Therefore, dilated pupil evaluations are essential in the care of those at risk.

In many cases, a patient's subjective complaints will indicate to the practitioner that a more detailed view of the macular region is warranted. The preferred method of obtaining the necessary magnified stereo view of the macula is to use a slit-lamp biomicroscopy with a planoconcave fundus contact lens. Since a topical anesthetic and coupling ocular gel is required, any type of fundus photography should be performed before hand.[7]

One should keep in mind that while the use of a direct ophthalmoscope will indicate the location and possibly the type of drusen that may be present, this instrument can lead the practitioner to a false negative conclusion. CNVM's and serous detachments can have a subtle appearance and indeed may not be easily detected using a direct ophthalmoscope.[4] In addition, although blood is usually present with a CNVM, it may be only a tiny spot and therefore easily missed.[7]

To summarize, a routine put forth by Folk is to first examine a patient with the indirect ophthalmoscope. Next look with a direct ophthalmoscope or Hruby lens. This is followed by fundus photography and flourescein angiography. Lastly, a complete slit-lamp examination using a fundus contact lens can be performed.[7]

SURVEY RESULTS AND CONCLUDING REMARKS

As mentioned earlier, part of this project consisted of a survey that was sent out to retinal specialists in Michigan -

eighteen in all - in an effort to gain some insight into the current thought concerning treatment and management of patients with ARMD. Nine ophthalmologists returned the survey in all, not enough to be of any statistical value perhaps, but enough to yield some insight into this disease.

Of those who did return the survey, the majority, however, did not complete the entire questionaire (appendix B). Many comments, such as "The data we give you in a survey like is 'off the top of our heads'. If you had a chart review, the numbers might be different," or one such as "We could get this from the computer. I don't have these figures" were noted. In retrospect, such comments were not surprising. Indeed, I see now that I made a mistake in not emphasing in the cover letter (appendix A) that was sent along with each survey that "off the top of the head figures" would have been adequate. What I was after was, afterall, an **impression** of the specialists' practice.

At any rate, the numbers of individual responses were tallied and can be viewed in appendix B. Many comments were offered so I think a discussion of the results and various accompanying comments might prove interesting and perhaps useful.

It is interesting that a majority of the respondents listed 10% or less as the percentage of ARMD patients treated medically, namely with a laser. To the practitioner, I would not think this figure to be out of line. To the general patient population, however, such a low rate of treatment might be surprising.

Question two, regarding referral to low vision specialists, was interesting in its wide range of responses - <10% to >50%. I

have a feeling, and this is my opinion, that this diversity of responses may reflect a varying attitude that some of these ophthalmologists have toward low vision sevices, and perhaps to a larger extent, their attitude toward optometry since the majority of low vision services are provided by optometrists.

The responses in questions three and four were not overly surprising. In question three, the percentage of CNVMs most commonly listed as presenting as poorly defined, however, was lower than that listed in the literature (20% vs. 50%). With regard to both questions three and four, one wrote "Some of these are treated under certain circumstances." This individual did not elaborate, but the comment is something to keep in mind.

No surprises were unearthed in question five regarding recurrence of CNVMs. An interesting comment was made regarding the frustration of recurring membranes, though. It simply read: "Recurrence is a problem and may be up to 70%".

No real conclusive information was obtained in question six with regard to factors commonly associated with ARMD. One interesting comment read: "Most of the ARMD patients have hypertension ... we have not done this study on our patients."

Patients with geographic atrophy, as surveyed in question seven, should be recalled for routine care every twelve months, according to the majority of responses. One respondent added "As long as they follow VA with Amsler grid". One answered six months, but added "If stable and reliable - 12 months".

Question eight revealed a unaminous "yes" response when inquired whether patients should monitor their vision with an Amsler grid. Again, no surprise here.

For question nine, regarding recall schedule for patients who have dry ARMD of their better eye and disciform degeneration of the other, the responses were about evenly split between six and twelve months. A comment accompanying a "six months" response read: "For first 1 - 2 years after developing disciform in fellow eye. Then once yearly if the patient is reasonably following their Amsler grid".

Once again no surprise responses were noted with question ten regarding the most common symptom that a patient with exudative ARMD reports. The responses were about evenly split between metamorphopsia and decreased vision.

The most common sign discerned in patients who develop exudative ARMD, as reported in question eleven, was about evenly divided between blood and serous detachment. One individual circled SRNVM, but added "(which) leads to serous detachment and exudate".

The responses in question twelve were interesting. The most common instrument selected as most helpful in the early diagnosis of exudative ARMD was the fundus contact lens. This was not surprising, unlike one survey that had only 'direct o'scope' circled. There were two doctors who circled both 'fundus contact lens' **and** 'direct o'scope', which, of course, is fine. One wrote "Slit lamp with Hruby lens" while another commented "Use all". I found it interesting that not only was BIO not circled, but one went so far as to write, with regard to the use of BIO for early diagnosis of wet ARMD, "NO!" I would imagine that the majority of these specialists would use more than one of the instruments

listed in the total treatment of patients with exudative ARMD. Indeed, as mentioned earlier, this is what Folk recommends. However, for the **early diagnosis** of exudative ARMD, the instrument of choice would be, I think, the fundus contact lens used with a slit-lamp biomicroscope.

Of all those who responded to question thirteen asking whether patients with dry ARMD should have flourescein angiography performed annually as a routine test, all responded with "No". After some thought about this question, the results are not all that shocking. That is, if flourescein angiography is not indicated, a patient should not be subjected to it routinely. One respondent even answered "No! Use only when more information is necessary or for treatment".

Question fourteen asks how soon should a patient recently diagnosed as having exudative ARMD be referred to an ophthalmologist. The responses were about what one might expect, i.e., 'immediately' to '1 - 4 days'. With regard to the latter response, one doctor wrote: "Depends on the clinical picture; as soon as possible, but also duration of symptoms can influence decision." It is also worth noting, I think, that one individual was careful to write 'retinal specialist' with an arrow pointing to the word 'ophthalmologist' in the question.

When asked about their success rate with those patients with exudative ARMD who have been treated with a laser, the responses were considerably varied. The individual who responded with '>80%' also added this philosophical note: "What is success? Definition: acceptance of problem by patient, use of low vision aids, social and emotional suport, self care in their own home

and etc." Another, who did not give an answer, wrote: "Very difficult. How do you define success?" As can be seen, the varying responses of this group of individuals reflect their differing views of just what is success.

The next question, number sixteen, I viewed as one of the most intriguing of the questions asked. Asking if early exudative ARMD would be more easily diagnosed if optometrists were legally allowed to use oral flourescein, the question not only raises an interesting point, but cuts into the heart of the ongoing debate between optometrists and ophthalmologists over what the former is capable of doing. It was not surprising, therefore, that all the specialists answered 'No', with one adding "because oral flourescein will only allow late photos - you can't accurrately interpret just late photos, you need a transit phase also". In all fairness, this individual raises a valid point. Another wrote 'uncertain' and commented: "because of false positives and negatives from poor interpretation or poor quality of flourescein angiography".

The last question, number seventeen, did not reveal any surprises. The attempt of the question was to gain some insight into the role race may play in those afflicted with ARMD. As has been reported in various literature sources, caucasians are overwhelmingly more likely to be afflicted with the disease. One practitioner did not respond, instead writing: "predominately white practice".

Throughout this paper, much has been discussed. Age-related macular degeneration, in both optometric and ophthalmologic

circles, is indeed a great disease. That is "great" in the sense that the moral, ethical, and legal obligations to the elderly patient are awesome. The importance of diagnosing ARMD as soon as possible in an effort to maintain the patient's quality of life cannot be overstated. With state optometric laws evolving as they are today, only one thing seems certain: The optometrist's role in the management and treatment of patients with ARMD will increase, not decrease. Barbara J. Jennings, O.D., writing for the Journal of the American Optometric Association, states it her belief that "...optometrists will eventually be allowed to legally perform photocoagulation..."[15] Even with all the advances optometry has made in the last couple of decades, I am skeptical about this comment. I do not doubt that optometrists could perform such treatment, but I believe there would have to be, first of all, drastic changes in how optometrists are trained. Extensive hospital experience would be necessary, first of all, and I do not see this happening in the near future. At any rate, the issue effectively illustrates my point concerning the rapidly changing scope of optometry.

Indeed, optometry **is** undergoing major changes in today's health care delivery system. To best serve our patients, we must keep abreast of the constant updating of literature in our endeavor to maintain an effective working knowledge of the suspected etiology, management, and treatment of age-related macular degeneration.

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APPENDIX A

Date

Ophthalmologist, M.D.
Address
Address

Dear Doctor:

I am currently a Senior Intern in the Optometry Program at Ferris State University and have a request to make of you.

As part of a senior project, I am doing an updated literature review of age-related macular degeneration. In an attempt to make the project as complete as possible, I thought it appropriate that I do a survey of retinal specialists, asking a series of pertinent questions about this condition.

Indeed, enclosed you will find a list of questions about age-related macular degeneration. I would appreciate it very much if you would take a few moments of your time and answer these questions as complete as possible and return the survey as soon as possible in the enclosed self-addressed stamped envelope.

Again, I express my thanks to you for playing an important role in my developing this literature review. Of course, if you have any other literature that you feel might be helpful to me, I would be grateful for that as well.

Sincerely,

DANIEL G. IRWIN
Senior Intern

WALTER BETTS, O.D.
Associate Professor

APPENDIX B

AGE-RELATED MACULAR DEGENERATION SURVEY
WINTER 1988-SPRING 1989

RESULTS*

*Total number of responses are in parentheses under a given possible choice. Written responses are also noted in parentheses.

1. Of the total number of AMD patients you see, what percentage are treated medically, specifically with laser treatment?

<10	10	20	30	40	50	>50
(1)	(4)					

2. What percentage of AMD patients do you refer for Low Vision services?

<10	10	20	30	40	50	>50
	(1)	(1)	(1)			(2)

3. Of those subretinal neovascular membranes (SRNVM) noted with flourescein angiography, what percentage present as poorly defined, thus ruling out laser photocoagulation treatment?

<10	10	20	30	40	50	>50
(1)		(3)	(1)			

4. Of those SRNVM's noted with flourescein angiography, what percentage present within the center of the foveal avascular zone, thus ruling out laser photocoagulation treatment?

<10	10	20	30	40	50	>50
(1)		(1)	(2)			(1)

5. Of those SRNVM's that you treat a.) What percentage recur? b.) Of these, what percentage recur contiguous to the laser scar? c.) Independent to the scar?

a.	<10	20	30	40	50	>50				
			(1)	(3)	(1)					
b.	<10	20	30	40	50	60	70	80	90	>90
								(1)	(2)	(2)
c.	<10	20	30	40	50	60	70	80	90	>90
	(4)	(1)								

6. Which of the following, if any, do you see most commonly associated with AMD? Circle more than one if necessary.

Cardiovascular disease	(1)	(1**)
Hypertension	(1**)	
Smoking		
Family history of ARMD	(1)	(1**)
Hyperopia		
None		
Other _____		

7. In terms of months, how long should patients with dry ARM be recalled for their routine care?

1	2	3	4	5	6	7	8	9	10	11	12
					(1)						(6)

(1 - "6 to 12")

8. Do you believe that all patients with dry ARM should monitor their vision with an Amsler grid?

YES	NO
(8)	

9. In terms of months, how often should patients who have dry ARM of their better eye and disciform degeneration of the other be recalled for their routine care?

1	2	3	4	5	6	7	8	9	10	11	12
					(3)						(3)

(1 - "3 to 6") (1 - "6 to 12")

10. What is the most common ocular symtom that a patient with early exudative ARM report?

metamorphopsia	decreased VA	floaters	other
(1**)	(1**)		
(3)	(4)		

11. What is the most common sign seen in your patients who recently develop exudative ARM?

blood	serous detachment	SRNVM	exudate
(1**)	(1**)	(1)	
(3)	(3)		

12. What instrument(s) are most helpful in the early diagnosis of exudative ARM?

fundus contact lens	90D lens	direct o'scope	BIO
(1**)	(1**)	(1**)	(1 -"NO")
(1***)		(1***)	
(3)		(1)	

13. Should patients with dry ARM have fluorescein angioscopy performed annually as a routine test?

YES	NO
	(8)

14. How soon should a patient recently diagnosed as having exudative ARM be referred to an ophthalmologist?

immediately
(3) (1 - "immediately to days")

1	2	3	4	5	6	DAYS
(1 - "1-2")						
(1 - "1-4")						
(1)						

1	2	3	4	WEEKS

1	2	3	MONTHS

15. What would you list as your success rate with those patients with exudative ARM who are treated with a laser?

>10	10	20	30	40	50	60	70	80	>80
			(2)		(1)	(2)			(1)

16. Do you think that early exudative ARM would be identified at an earlier stage if optometrists had the legal option of using oral fluorescein?

YES	NO	(1 - "UNCERTAIN")
	(5)	

17. Of your total patient load afflicted with AMD (signs and/or symptoms), how would you break down, by race, percentages of those individuals affected?

CAUCASIAN	<10	10	20	30	40	50	60	70	80	90	>90
								(1)		(1)	(3)
BLACK	<10	10	20	30	40	50	60	70	80	90	>90
	(3)		(1)								
OTHER	<10	10	20	30	40	50	60	70	80	90	>90
	(2)	(1)									

/*Indicates multiple reply to a question.