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Electromagnetic Energy and Cataracts

MILTON M. ZARET

Director of Research
Zaret Foundation
1230 Post Road
Scarsdale, New York
and
Clinical Professor of Ophthalmology
New York Medical College
Valhalla, New York

INTRODUCTION

Ophthalmology has frequently been at the leading edge of state-of- the-art technology by incorporating both contemporary scientific electromagnetic theory and advanced electronic research techniques into the pragmatic practice of clinical medicine. Concomitantly, eye injury can provide the first sign of hazards.

The biological effects of laser/maser radiation were first reported in 1961 (1). In 1965, Zaret (2) described row the special features of various laser/maser irradiation could be applied to ophthalmology and other specialties of medical practice for therapeutic, diagnostic, or research purposes, and most of these applications have now been realized. Also in 1965, Zaret (3) described potential health hazards for humans, including cataractogenesis, that could occur as a consequence of direct or indirect exposure either to laser/maser irradiation itself, or to the electronic smog created by either the laser/maser generating circuitry or other electronic equipment.

EMBRYOLOGICAL AND ANATOMICAL FACTORS

It is necessary to describe the embryology and anatomy of the intraocular lens to permit an understanding of the cataractogenic process in humans. The lens is composed of a non-rigid colony of cells, known as the lens substance, enclosed within a tight fitting elastic-like membrane known as the lens capsule. The lens is located within the anterior segment of the eye immediately behind the iris and its pupillary opening, and is approximately 1 cm in diameter and 0.5 cm at its widest depth along its optical axis.

The cells of the lens substance are not totally transparent. Instead, like other embryonal cells, their protoplasm has a translucent appearance so that the lens substance is visible when viewed in a dark surround while being directly illuminated as by a slit-beam of light at an oblique angle of incidence to the viewing axis of a biomicroscope. *In vivo*, the aqueous in front

of and the vitreous behind the lens are ordinarily optically empty, and thereby serve as the dark surround. The obliquely placed slit-beam of light provides an illuminated, thin cross-section of visible lens substance through the thickness of the lens. The various depths, from either the anterior or posterior surface under observation, can be determined by the focal plane of the biomicroscope. Relative distances from the anterior or posterior refringent edge of the optical beam can also be determined.

Under normal conditions the capsule is not visible by slit-lamp examination because it is thin (a few microns thick), and not relucent. However, its site is always recognizable by slit-lamp examination because it is demarcated by the edge of the lens where the light beam is entering or exiting. The normal capsule is elastic and it exerts a compressive force on the lens substance which can thereby be molded to permit focusing an image of regard onto the retina. The capsule also acts as a Donnan's membrane permitting selective permeation of dissolved metabolites into and waste products out of the lens.

The capsule elasticity compresses the lens cells against each other relatively compactly, and the sparse extra-cellular fluid is ordinarily not perceivable. Occasionally a small sphere of fluid, termed a vacuole, is formed and may be observable by slit-lamp biomicroscopy for a few weeks or months. Such a transient microscopic vacuole represents the largest collection of extra-cellular fluid ever found in the normal lens.

Also occasionally, dot-like opacities located in the lens substance can be observed by slit-lamp examination. These, too, are usually present only for weeks or months and are believed to represent salt precipitates temporarily present in the extra-cellular fluid. Although vacuoles and dot opacities can both be observed by slit-lamp examination as if they were lens substance defects, neither are, of themselves, relatable to cataractogenesis.

From its earliest recognizable embryonic stage of development as a discrete organ of accommodation, at about the one- month stage of fetal evolution, the lens has all of the elements that it will ever contain. It has become sequestered from the rest of the eye and separated from direct connections to the circulatory system so that, thereafter, no cells can enter or leave it. Although still microscopic in size, the lens is predestined to grow throughout life only by multiplication and differentiation of the cells contained within its capsule.

Soon after this stage of its evolution, the life-long organization of the lens becomes recognizable. Lining and in intimate direct contact with the entire anterior capsule is a unicellular sheet of cuboidal epithelial cells. Filling the remainder of its volume are the primitive or primary lens fiber cells (lens fibers). At the equator of the lens, new lens fiber cells, termed secondary lens fibers, are formed by differentiation of the peripherally located (around the equatorial circumference) epithelial cells. As the secondary lens fibers are forming, they become elongated with one end extending posteriorly into the potential space between the pre-existing lens fibers and the posterior capsule, and the other end extending anteriorly into the potential space be tween the pre-existing lens fibers and the posterior (inner) surface of the unicellular sheet of cuboidal epithelial cells that separate the lens fibers from contact with the anterior capsule. Thus,

the oldest lens fiber cells are located in the center of the lens and the newest are at its periphery. When partial opacification of a small region of the lens substance occurs, it may be possible to determine the approximate time during life when the event took place by the depth of the opacity from the surface of the lens.

Secondary lens fibers, although retaining their original dimensions in cross-sectional diameter, nevertheless continue to grow in length until, near the anterior and posterior poles of the lens, they abut against the ends of similar lens fibers coming from the opposite direction. When that occurs, axial lens fiber growth stops, apparently because of contact inhibition.

When new lens fiber formation occurs the sheet of epithelial cells enlarges concomitantly by cell multiplication. The cells retain their embryonal size and cuboidal shape, and each cell remains in contact with the capsule anteriorly so that the areal enlargement of the unicellular sheet continuously extends to the equatorial region of the enlarging lens. There, the most peripheral epithelial cells continue in the process of forming new secondary lens fibers throughout life. In this fashion, the growth of the lens continues at a slow rate, by the addition of new lens fibers.

With continuing addition of lens fibers, suture lines occur at the anterior and posterior poles of the enlarging lens where contact inhibition takes place. Some sutures become visible by slit-lamp biomicroscopy because the tips of the elongated fibers do not make complete contact with other fibers, and the space becomes filled with extra-cellular fluid, which has a different index of refraction from lens fibers. Such visualizations of the sutures can appear to be defects in the lens substance.

Once fully formed, lens fibers can be compressed tightly against each other. The older, more centrally located lens fibers normally appear to be more transparent or less relucent than the younger, more peripherally located lens fibers. This results in a difference in luminosity of lens fibers so that at different depths, different regions can be identified. Generally, the lens can be divided into two regions according to luminosity, a nucleus at its center and a cortex at its periphery. At either site, side-to- side adhesions or axial adhesions of small groups of adjacent lens fibers can occur. They give rise to the slit-lamp appearance of banding and riders, respectively. When larger areas are affected, a localized haze can be seen.

All of the above-mentioned types of lens imperfections or defects were diligently searched for as part of the ophthalmic examinations I performed on several thousand scientists and technicians who worked in radiant-energy environments. For present purposes, the principal concern will be with alterations in the appearance of the central or nuclear portion of the lens, the posterior polar subcapsular sutural region of the lens, and the capsular surface or refringent edge of the lens. Other pathological features of the lens will also be discussed.

PHYSIOLOGICAL OPTICS

It is appropriate to define the manner in which the lens is involved with normal visual

function. The lens is suspended by circumferentially located zonules attached to the peripherally located ciliary muscle which controls focusing by its state of tonus. The lens lies immediately behind and in the plane of the iris whose posterior surface rests against the suspended lens's anterior capsular surface. The center of the pupillary aperture in the iris is practically co-axial with the optical axis of the lens.

The posterior surface of the iris is lined with a layer of densely pigmented cells which also line the inner surface of the ciliary body and the outer portion of the retina. These pigmented tissues are comparable to the black, light-tight lining of a camera; they minimize the entry of stray light that could interfere with the quality of perceived images. Thus, all of the light useful for vision enters the eye through the pupil.

Under ordinary conditions of vision, the object of regard must be illuminated sufficiently so that its reflected light can be imaged onto the macular portion of the retina. Simultaneously, the remaining ambient light entering the eye from the remainder of the field of vision must be kept below intensity levels that would interfere with the discriminatory functions of the macula. A major method for adjusting the amount of light inside the eye is by the tonus of the iris musculature controlling pupil size. Thus, the pupil does not have a fixed diameter, but instead fluctuates, averaging approximately three millimeters in diameter.

The major refracting surface of the eye is the cornea , but the fine focusing is accomplished by alteration of the curvature of the lens via the process of accommodation. It is mediated by the elasticity of the lens capsule via the tension maintained on it.

Thus, two major factors that affect visual acuity are the ambient background light within the inner eye, and the refractive state of accommodation.

CATARACTS

The term "cataract" has different meanings in different contexts. Many scientists (including a few ophthalmologists) label as a cataract any lens defect acquired by a laboratory animal during the course or as a consequence of an experiment. On the other hand, there are a few ophthalmologists who maintain that a lens opacity in humans is a cataract only if it reduces visual acuity. Visual acuity is only one of many measures of visual function. It is inadequate and frequently misleading to rely on visual acuity alone as the measure of cataracts. For the reasons given below, a better, more clinically useful definition is that a cataract is any type of lens defect that interferes with visual function (4).

Light rays emanating from an illuminated object of regard enter the lens only through the pupillary aperture, and these occupy only the axial and paraxial projection of the pupillary area through the lens. In other words, only the central axial 20% of the lens volume is used for visual acuity determination. The remaining 80% of the lens substance lies in the relative penumbra and umbra of the iris while viewing an object of regard. Under these conditions, if there are any opacities of the lens substance located in the penumbral or umbral zone and such opacities are

illuminated by a peripherally located light source, then those opacities become secondary luminaires within the eye thereby producing glare and reducing visual acuity. Thus, visual acuity may be excellent under the contrived dark-room conditions usually used for testing, but may be considerably impaired under the ordinary life situation of driving at night or into the direction of a low-lying sun.

Another optical factor to consider is the nodal point of the lens. This lies in the lens substance, along the optical axis, near the posterior pole in the region of the posterior suture. As will be discussed later, this is the site where posterior polar cataracts occur. Prior to opacification of the lens substance, extra-cellular fluid can collect at the nodal point. This can produce a prismatic optical effect at the interface between the lens fibers and the extracellular fluid, resulting in monocular diplopia or polyplopia with each of the multiple images having a visual acuity of 20/20.

Another factor to be considered is the state of refraction for distance vision. Ordinarily, corrective lenses are prescribed to provide a visual acuity of 20/20, which is the pragmatic equivalent of placing the retina in focus with infinity. Abnormal hydration of the lens causes it to become swollen and an infrequently encountered stage of this is known as intumescence. This results in an abnormal increase in convex curvature which then induces a relative myopia. Under that circumstance, visual acuity can still be corrected to 20/20, but a change in the basic eyeglass prescription is needed. Subjectively, if a patient were nearsighted he would think his vision got worse, if the patient were emmetropic (normal vision) he would become nearsighted, and if the patient were farsighted he would think his vision got better.

An additional factor, capsulopathy in its early stages, may also induce an aberration in visual function without materially affecting visual acuity. In this example, consider that the posterior refringent surface of the lens is defined by the capsule, and our immediate concern is limited to that portion of the capsule situated along the optical axis of the eye and lying within the areal projection of the pupil. Should this part of the capsule, which is only a few microns thick, exhibit the optical qualities of becoming recognizable by slit-lamp biomicroscopy as if it were roughened, thickened and faintly opacified, it would behave optically as if it were a microscopically glazed and thin diffraction filter. As such, it would not reduce visual acuity, but the patient nevertheless would describe vision as if it had a misty or hazy veiling even though visual acuity was still correctable to 20/20.

Although more factors could also be discussed concerning abnormalities in visual function that can occur without a reduction of visual acuity, a final item to be presented here concerns anomalous change in transmission of color through the lens. The chromatic content of an illuminated object being imaged on the macula is ordinarily the same for both eyes of an individual so that there is no perceptible difference in color value when comparing one eye to the other. Just as irradiation can cause all of the other examples listed above, it can also cause a change in the colorific value of some lens proteins, thereby causing that lens to act as a partial color filter and thus affect the perceived hues of the observed object. Should the lens of one eye

receive more irradiation producing this effect than the other eye, then the patient's color sense can be different for the two eyes, a condition I termed anisometachromatopsia (5,6).

The net result of examining the factors of pathophysiological optics of the lens, as described above, is to recognize that there are many anomalies of visual function in addition to visual acuity. Any one or more of these factors could be due to an early stage of radiant-energy injury, and thereby be the harbinger of an evolving incipient cataract. Prevention of an advanced stage of radiant-energy cataract can frequently be achieved. However, that usually depends upon establishing the diagnosis of lens injury early, before visual acuity has degraded appreciably. This is especially true where, while visual acuity is still correctable to 20/20, there has been an interference with some other function, such as anisometachromatopsia, intermittent misty or hazy vision, temporary blindness from a light in the field of vision such as oncoming lights during night driving, or occasional diplopia or polyplopia. In such cases, and especially if the lens pathology is present only in one eye, the probability exists of either preventing a loss of visual acuity or significantly delaying its onset or the future need for cataract surgery by identifying the irradiation source and protecting the patient from additional exposures (7).

The difficulty of relying solely on visual acuity as a test for degradation of visual function was demonstrated clearly by Zaret and Snyder (8) in 1977 when they chronicled how an air traffic controller with 20/20 visual acuity in each eye repeatedly put aircraft into mid-air collision courses because of other disturbances in visual function. Also in 1977, Zaret and Snyder (9) presented evidence in an air traffic controller that the earliest symptom of a radiant-energy cataract was a transient visual problem, such as looking through a misty fog or wet glass. This finding of altered visual function antedated by several years the subsequent development of reduction in visual acuity. Moreover, in 1979 Zaret (10) described the case of an airline pilot who, despite having a visual acuity of 20/20 in each eye, nevertheless exhibited polyplopia during the landing of a commercial airplane.

ELECTROMAGNETIC ENERGY AND CATARACTOGENESIS

Duke-Elder discovered that exposure to radiant energy, including long-term direct exposure to sunlight, was either the causal or a contributory etiological factor for most cataracts acquired after birth (11). His data antedated the electronic revolution, and my data has been collected after its inception, so that there have been both qualitative and quantitative changes in the composition of electronic smog. Nevertheless, my data supports his original thesis, adds detailed refinement to some of his observations, and extends our knowledge into other regions of the electromagnetic spectrum that formerly were not, but now are, clinically important.

Much of what appears here is in large part the result of my unique personal experience, during the past 35 years, investigating some aspects of radiant-energy cataractogenesis under both laboratory and clinical research conditions. An important aspect of this work is the unprecedented opportunity it afforded me to investigate actual situations where cataract

problems were occurring. This permitted discovery of differing types of cataractogenesis at varying stages of their evolutions, institution of preventive measures designed to reduce or eliminate additional exposures, and follow-up investigations by repeated ophthalmological surveillance of the subjects to determine the subsequent course of the cataractogenic process and the effectiveness of the preventive measures.

The following account represents an integration of some classical teaching that I found to be valid with some new information. The reader should bear in mind that there are a number of different mechanisms that can ultimately result in cataracts, and that several different pathways may be operant simultaneously or sequentially. Some specific mechanisms will be presented as being primarily an ionizing radiation effect, an ultraviolet radiation effect or a nonionizing radiation effect, while recognizing that none of these mechanisms occurs in the total absence of the others.

IONIZING RADIATION

One concept of ionizing radiation injury of the lens involves mutagenic changes that are usually expressed as aberrant lens fiber formation, resulting in some of the newly forming lens fibers become aborted in that process. The aborted cells either drift or are propelled by succeeding normally developing lens fibers into the posterior subcapsular space, and after 3–5 years start to collect at the posterior pole of the lens where they can be recognized as a posterior polar cataract.

ULTRAVIOLET RADIATION

One currently popular concept of how ultraviolet radiation may induce cataractogenesis is based upon partial, *in situ* denaturation of cytoplasmic protein contained within lens fiber cells. The evidence is derived from *in vitro* analysis of extracted lens substance protein where a chromophoric change to a brown color can occur following exposure to ultraviolet radiation. This was the theorized rationale whereby the nuclear sclerosis type of cataract, which is ordinarily gray, sometimes turns brown. Nuclear sclerosis and its occasional derivative, brunescent cataract, are considered by many ophthalmologists to be stages of the aging process and, as such, are ordinarily described as senile cataracts. However, senescence can occur without any evidence of nuclear sclerosis and I have examined many older patients who never exhibited nuclear sclerosis or other evidence of cataractogenesis. So, if nuclear sclerosis is a sign of senility, it is a sign of abnormal aging and abnormal aging, itself, can be an irradiation effect.

NONIONIZING RADIATION

Nonionizing radiation cataract *per se* has evolved as a recognizable clinical entity only since World War II, which fueled the electronic revolution and led to the subsequent explosive proliferation of military, intelligence, industrial, communication, medical, educational, consumer product, and amusement-related electronic devices. This type of cataract is dependent upon

exposure to radiant energy of any frequency in the nonionizing spectrum. Its distinctive feature is lens capsule injury.

Capsulopathy, at its inception, is recognizable by slit-lamp biomicroscopy as the appearance of roughening, thickening and opacification at the refringent edge of the lens. The fact that acquired capsular cataract could serve as a signature of nonionizing radiofrequency injury of the lens, especially when all other differential diagnostic criteria were also satisfied, was discovered and first reported in 1964 by Zaret (12), and independently verified by Bouchet and Marsol (13) in 1967.

IRRADIATION RELATIONSHIPS

When a cataract is produced by exposure to a specific spectral band of electromagnetic energy, it is common to find other changes in the eye. These include posterior polar cataract induced by ionizing radiation, brunescent cataract from ultraviolet radiation, and capsulopathy, an early stage of capsular cataract from nonionizing radiation.

These distinctions are based on clinical findings in humans who, during their lifetimes, have experienced variable exposures to a wide variety, both qualitatively and quantitatively, of irradiations. To some degree, prior irradiations are both additive and cumulative. A more detailed discussion of many of these factors as well as a classification generally suitable for any type of radiant energy cataract is given elsewhere (4).

CATARACTS INDUCED BY NONIONIZING RADIATION

It was noted earlier that lens fibers are generally packed tightly together, side by side with parallel axial alignment. This structural regularity accounts for not only the optical-quality transmission of images to the retina, but also for the normal appearance of the lens itself. However, following irradiation, because adjacent contiguous lens fibers are kept in a practically immobile contact with each other, the cell walls can adhere to each other as if some contact points between these cells have agglutinated. Then, should hydration occur in such a localized region of the lens, thereby converting the potential extracellular space between lens fibers into a real space, this could result in the tissue appearing as if it were a syncytium because the regular parallelism of the adjacent lens fibers had been altered.

Thereafter, whenever this syncytial region of the lens substance is illuminated, it causes internal scatter and reflection of the illuminating light, resulting in a misty or hazy opacified appearance in that region of the lens. Such hydration in the lens substance can be the initial stage of cataractous change, and it ordinarily precedes protein coagulation. When this type of opacification results from hydration alone, the opacity will seem to disappear when the slit-lamp beam and viewing optics are aligned co-axially.

Ordinarily, the space between the posterior capsule and the posterior surface of the lens substance is optically clear. But it is an area where aberrant lens fibers can collect, frequently in

a decaying or liquefying state. Their proteolytic products cause opacification of adjacent le ns fibers, resulting in clinically recognizable posterior subcapsular opacities. When such opacification interferes with visual function, it is termed posterior subcapsular cataract (PSC). This form of PSC can frequently be observed in its early stages to be separated from and not in direct contiguous contact with the capsule when it arises from exposure to ionizing irradiation.

Another form of posterior subcapsular opacification occurs in which the cataractogenic process begins at the capsule itself. This process is a capsulopathy and it also can occur in the anterior capsule. Eventually it can result in reduced visual function, at which time it may be recognized as a capsular cataract.

Another feature of capsulopathy is that patchy areas of the capsule appear to lose their ability to function effectively as a selectively permeable membrane, thereby permitting a greater than usual local fluid transudation. This results in the accumulation of vesicles lying in contact with the capsule. Vesicles differ from vacuoles in many ways such as being macroscopic in dimension. They are usually present in large numbers, and rarely disappear completely. Vesiculation is another sign of abnormal hydration and it usually leads to cataractous changes in the lens substance. In contrast, vacuoles are seldom found in clusters, are usually resorbed, and generally do not result in cataracts.

Capsulopathy exhibits a variable slit-lamp appearance during its development. It first appears as if small, scattered regions of the capsule are roughened and thickened. Later, as the number and size of these areas increase, confluence of regions occurs and opacification of the capsule becomes evident and then denser. This process leads to a variety of localized forms that are variously described as a honeycomb (especially when accompanied by vesiculation), brush-mark, lace-cloth, spider web, breadcrumb, peppered-surface, depending upon the imaginative perception of the observer. Eventually, a coalescence of some of the areas occurs as the process becomes more widespread, resulting in sheet-like areas several millimeters long. However, the entire lens capsule never becomes uniformly or homogeneously dense white. Instead, even when almost the entire lens capsule appears to be involved, some areas of clear capsule can still be recognized. Most ophthalmologists would recognize this end stage as capsular cataract, formerly an extremely rare condition, but now appearing to increase in prevalence commensurate with the electronic revolution.

Acquired posterior polar cataracts usually originate as a small volume of fluid in the subcapsular space. Although it appears to be optically empty , like a vacuole or a vesicle, it can differ because it is not spherical. Moreover, it acquires an irregular outline and may become somewhat lobulated as it enlarges and becomes invaginated into the lens substance. Subsequently, as a consequence of new lens fibers encompassing it from behind, it becomes partially incorporated within the lens substance at the posterior suture in or near to the optical axis of the lens. (It is sometimes referred to as an “oil drop” cataract, but this is misleading because that conjures a false impression of the composition of the fluid.) It is primarily an extremely localized area of phakohydritis. Should it spread, as it usually does, more and more of

the adjacent lens fibers will undergo localized lens protein coagulation as the cataractous process enlarges. Ultimately, a disturbance in visual function occurs and occasionally this may progress to a stage where surgery is required. Because it is located near the optical nodal point of the lens, it can give rise to monocular diplopia or polyplopia due to prismatic effects.

Still another type of lens substance opacity that can be acquired following irradiation is nuclear sclerosis. It is often erroneously referred to as a "senile" type of cataract because it has long been known to increase in prevalence with advanced chronological age. However, as discussed earlier, nuclear sclerosis especially in a pre-senile age group (i.e., under age 65–70) can itself be considered to be a sign of premature, abnormal aging and pathological aging, by itself, can be a sign of radiation injury.

Additional types of lens-substance defects can also be observed by slit-lamp biomicroscopy. These include opacification along the linear axis of adjacent lens fibers, and a change in relucency over a larger segment of the lens which is usually pie-shaped with the apex towards the optical axis. These defects depend upon the geometry of the lens fibers, and on their state of hydration and coagulation.

The role of radiation-induced phakohydritis (as well as the absence of lens inflammation, termed phakitis) was described in 1974 (4). Here, a few pertinent factors will be reviewed relating to whether the hydration was immediate or delayed, localized or widespread, and reversible or irreversible.

It is rare for the entire lens to be irradiated homogeneously, or for the effects to be present uniformly throughout the lens. Thus, intumescence of the entire lens (hydrops) is an extremely uncommon finding and, when present, is usually due to nonionizing radiation, much less frequently due to ionizing radiation and, in my experience, rarely, if ever, due to ultraviolet radiation.

The sudden appearance of intumescence ordinarily signifies that the lens has been irradiated recently. Usually, intumescence is localized to specific regions of the lens such as to a quadrant following beta radiation therapy for recurrent pterygium, to a partial posterior capsular vesiculation following radiofrequency irradiation, and to posterior polar hydration following X-ray irradiation. In these instances, partial intumescence ordinarily becomes apparent from months to years following exposure. Once established, however, it usually remains irreversible. Nuclear sclerosis is a form of partial intumescence, just sufficient to produce a syncytial-appearing arrangement of the lens fibers of the nucleus.

Generalized intumescence of the lens can be caused by many factors, such as radiation, trauma, toxic substances, and metabolic sources, and it can follow a variable course from being effervescent to being irreversible. However, in most types of phakohydritis, the etiological factors can usually be estimated if searched for diligently.

DIAGNOSTIC CRITERIA

It is an error to ascribe all of the perceived biological effects to only one physical factor simply because it may be easy to detect and measure that factor (14). For example, consider ionizing radiation where biological effects of concern such as mutagenesis or cataractogenesis are not apparent until years after the suspect exposure, and where the measured parameter and presumed sole etiological factor is ion-pair formation. Because the physical models and the physicists' mathematics were so elegantly constructed, it appears that an important physical–biological phenomenon escaped notice. Namely, in addition to stripping electrons, the irradiation also pumps other electrons into higher energy orbits without ejecting them from their atoms. When the higher-energy-level electrons revert to their original, stable energy state, emission of nonionizing radiation occurs. In other words, some of the incident ionizing radiation energy is converted, *in situ*, into nonionizing radiation. Nonionizing radiation can also be cataractogenic.

It has frequently been recognized that cataractogenesis itself can proceed via any one or more of several pathways, singly, sequentially, or in various simultaneous combinations. Cataractogenic exchange energy relationships are not so simplistic as to be describable solely in terms of electron pairs created by ionizing radiation, or as biochemical degradations stimulated only by specific spectra such as that of ultraviolet radiation.

Even more misleading is the concept that, for humans, the cataractogenic potential of chronic exposure is relatable only to some contrived temperature quotient. Microwave cooking may indeed result in acute cataractogenesis due to a thermal injury. Fortunately, however, such instances are rare, and are usually so dramatic in occurrence as to be diagnosed readily; but one should never accept an overt burn as the threshold for radiation injury, *per se*. Instead, the ordinarily encountered human cases of nonionizing radiation cataractogenesis are delayed in onset, and are caused by repeated or chronic irradiation at field intensities either too low to be perceived directly, or too brief in duration to result in recognizable elevation in core or local temperature.

The diagnostic standard applied to human pathology is not scientific certitude, but rather the test of medical reasonableness. Nevertheless, the first cluster of human nonionizing radiation cataract cases that I discovered had an exposure commonality and a specific endpoint pathological criterion response, lenticular capsulopathy, that fulfilled all the qualitative criteria of scientific certitude better than any past or proposed experimental protocol. The relevant cases involved individuals where the viewing (and exposure) was performed only with the dominant eye through a peep-hole, and where the contralateral non-exposed eye then served as a control for comparison. This provided the best possible investigative model, had it been done intentionally, for determining the specific cataractogenic effects of radiation in humans.

From other clusters of cases exhibiting similar pathological changes in the lens capsule, two important additional discoveries subsequently became apparent. The first was that nonionizing radiation was cataractogenic throughout its spectral distribution. This includes leakage from

microwave ovens (7), from cathode-ray tube display units initially used by air traffic controllers (8-10), from those units now in widespread use by office workers and school children (5,6), and from radio transmissions such as citizens-band radios and hand-held walkie-talkies as used by policemen and firemen.

The other important observation was the total absence of any clinical relationship between delayed-appearing cataractogenic effects of chronic exposure to nonionizing radiation and intraocular temperature. Thus, the health-safety criteria for nonionizing radiation in the United States, all of which are based on presumptive thermal effects, are meaningless. The appropriate methodology for monitoring human injury is different from that which has been generally applied. When a lens is injured by irradiation, it will ultimately display a pathological response that may proceed to cataract formation despite the inappropriate assumptions that underlie the present applicable regulations.

Cataracts are not born fully formed and, as mentioned above, may evolve via several different pathways. The PSC is an example. At its inception, minute gray opacities take origin in the lens substance at a few scattered loci near, but separated from, the capsule. These may remain stationary for long periods of time without interfering with vision, but they ordinarily increase in number, size and density and eventually interfere with visual function. They may be diagnosed as PSC by ophthalmologists when these are first observed or when they first interfere with vision. Since ophthalmologists ordinarily do not make such precise distinctions between opacities and cataracts located in the posterior subcapsular region of the lens substance, it is understandable that interpretation of the results of clinical investigations may be confusing.

An epidemiological study of lenticular imperfections in the eyes of a sample of microwave workers compared to a control population was undertaken because cataracts were known to be occurring in microwave workers, especially radar technicians and scientists working in both military and civilian occupational environments (15). Prior to this study, there were two important unknowns—what was the earliest indicator of impending radiofrequency cataractogenesis, and what exposure time duration for occupational environments was required before that pathological process reached a stage where the diagnosis of cataract could be established with medical certainty? I found that posterior subcapsular cataract, as diagnosed by most ophthalmologists, was an indicator, and it would take about 20 years before that became evident. A recent report by Hollows and Douglas (16) confirmed both findings: posterior subcapsular cataract was found to be the earliest indicator of impending, occupationally-induced, nonionizing radiation cataractogenesis.

DISCUSSION

There is no question that chronic exposure to nonthermal levels of both ionizing and ultraviolet radiations can be cataractogenic, and that no data exist upon which meaningful human safety can be formulated for either form of radiation. The situation regarding nonionizing

radiation is exactly the same.

The only published data that implies otherwise was provided by Colonel Appleton, then the chief ophthalmologist in the United States Army and staff ophthalmic consultant to the Army's Surgeon General, who together with McCrossan, an optometrist at Fort Monmouth, New Jersey, conducted an ophthalmic epidemiological study in which they claimed that the microwave environments at Fort Monmouth were not cataractogenic (17). However, Frey's statistical analysis of Appleton and McCrossan's data (18) revealed that there actually was a difference between their exposed and control groups, and that their conclusion should have been the opposite of what was stated (19).[1]

CONCLUSION

Here we have touched on a few highlights. Although it is gratifying to find that a few major discoveries of mine have been substantiated, like the confirmation by Bouchet and Marsol that capsular cataract can be the result of injury by nonionizing radiation as well as the confirmation by Hollows and Douglas that a valid epidemiological study would demonstrate that chronic exposure to nonionizing radiation would result in an increased prevalence of posterior subcapsular opacities. Nevertheless, there is still much more information to be acquired.

It has become apparent that our societal need is not quantity of research, but instead, our need is for better quality and relevance. That can only be achieved by enlisting the aid of our best, independent scientists.

It behooves the scientific community interested in these matters to adopt a code of ethics in order to protect society from narrow parochial and partisan vested interests, without ignoring the relative importance for and contribution to society of those very same vested interests. Predictably, the next major test will evolve around the medical diagnostic use of nuclear magnetic resonance imaging where Smith (19), in preliminary experiments, has demonstrated a cataractogenic effect in bovine lenses.

[1] There were major flaws in the study by Appleton and McCrossan. They designated as "controls" workers at risk of eye injury due to radiations other than microwaves. The control group was composed entirely of individuals who worked with "laser, xenon arcs, ultraviolet and welding equipment (to include plasma torches)"—all of which are recognized to be potentially cataractogenic. Also, they used an unorthodox examination technique that renders some lens opacities invisible, and included a score for iridescence of the lens capsule, a finding that has no diagnostic significance nor comparative value and only served to dilute the significance of positive findings.

More incredible than the report itself, was the subsequent failure of the editors of the publishing journal. After initially accepting for publication a Letter-to-the-Editor from me containing a detailed description of the defects in the Appleton and McCrossan article, the editor, for unstated reasons, subsequently refused to publish my letter. This raises some serious scientific and ethical questions for the Surgeon General of the Army (who did not affix his usual disclaimer) and for the ophthalmic journal editor, who appears to me to have relinquished his proper role.

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