EYE DISEASE AND COLOR DEFECTS*

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INTRODUCTION

It is sometimes disconcerting to the student of vision to find that his new ideas are in fact rediscoveries of old ones. Nowhere is this phenomenon more striking than in the field of color defects where not only Maxwell and Helmholtz but also Helmholtz's great students, von Kries and König, enjoyed phenomenal insight. Before the turn of the century, these scientists described and defined not only the congenital but also the acquired color vision defects which accompany eye disease. Subsequently, study of acquired color vision defects declined. François and Verriest (1961) summarized the status of the area in Volume 1 of Vision Research:

"... the acquired defects were so neglected that the facts already known were almost completely forgotten and eventually cases of acquired deficiency were even considered as congenital."

How much was lost can only be appreciated by a brief review of the early work in color defect.

In the field of color vision defects, von Kries is associated with the analysis of abnormal color matches. He (von Kries, 1897) differentiated three types of color matching abnormality: (1) a widened match characterized as a reduced normal system, (2) a shifted match characterized as an absorption system, e.g. by the interposition of spectral filters before the visual photopigments, and (3) a shifted match characterized as an alteration system, e.g. by a fundamental change in shape or spectral locus of one or more of the photoreceptors. The reduction and alteration systems saw immediate application to congenital dichromacy and anomalous trichromacy. However, all three mechanisms are important in acquired color vision defects. The absorption system occurs in depositions of material in the lens or cornea; the alteration system occurs in macular disorders as a result of receptor disorientation; and the reduction system occurs in optic nerve disorders.

König (1897) described the first cases of "blue-blindness"; these were in fact acquired blue defects in eye diseases. Hereditary cases were only found at a later date. König also described acquired red-green color vision defects and is famous for his design of a spectral color mixing device.

The tradition continued with Nagel, who was a student (and the son-in-law) of von Kries. Nagel (1907) gave his name to the instrument used to measure the Rayleigh equation and called the device an "Anomaloskop". He used spectral fields similar to those used by Lord Rayleigh (1881) who had discovered anomalous trichromacy. Nagel described the color matching technique used for classification of congenital color vision defects. Nagel also emphasized the importance of control of the field size in studying color defects. This, like many other earlier observations was to be rediscovered in the 1950s (Jaeger and Kroker, 1952) and again in the 1970s (Smith and Pokorny, 1977). Nagel (1905) also described a red-green color defect in optic neuritis. Helmholtz's students were by no means friends. von Kries and Nagel looked down on König although today no one would question König's contributions.

Köllner (Fig. 1) combined these observations with his own clinical experience and derived his famous rule for acquired color vision defects: blue defects are characteristic of retinal disorders and red-green defects are characteristic of optic nerve disorders. This rule remains valid today despite occasional contradictions.

Köllner (1912) summarized his work in the book: "Die Störungen des Farbensinnes, ihre klinische Bedeutung und ihre Diagnose" ("The Disturbance of the Color Sense, its Clinical Meaning and Diagnosis"). When we collaborated with members of the International Research Group on Color Vision Deficiencies 65

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years later on a book on color defects, we were pleased to discover how current Köllner's book remains.

COLOR DEFECT AND EYE DISEASE PRIOR TO 1960

The early studies of congenital color defects were continued by Engelking (also a student of von Kries), Franceschetti, and Trendelenburg, among others. The 1920s saw English and American physicists and psychologists pursuing the study of normal color vision and agreeing on international standards for photometry and colorimetry. This story was told with considerable charm by W. D. Wright (1981, 1984, see also Boynton, 1979). Wright (Fig. 2) and his colleagues (Wright, 1946) also provided the delineation of congenital color defects including tritanopia (Wright, 1952). Acquired color vision defects were by contrast neglected.

The 1950s saw a new impetus and new investigators with specific interest in color defects in eye disease. Verriest has emphasized that the 1950s were ripe for new investigation into acquired color vision defects. An important reason lay in Farnsworth's development of arrangement tests using Munsell colors. Pierce (1934) is usually credited with the idea of an easily performed vocational test for color vision. It was however Farnsworth (1943) who translated the concept into reality and described the tests we still use today: the Farnsworth–Munsell 100-hue (FM 100-hue) test and the Farnsworth dichotomous test. The FM 100-hue test has 85 hues of similar chroma and lightness, representing the full color circle. The hues are separated by approximately equal perceptual steps. The task of the observer is to arrange the hues in a natural color order; in practice this is achieved by only 1–2% of the normal population. The hues are arranged in four boxes, presented one at a time. The observer can make errors within a box but cannot make errors between boxes. Thus the test is one of color discrimination rather than color confusion. In contrast, the dichotomous test was designed as a vocational test of the ability to discriminate large color differences (Farnsworth, 1943). The test now known as the Farnsworth Panel D-15, was subsequently developed for use in a test battery in which congenital color defectives with poor discrimination could be separated from those with good discrimination (Farnsworth, 1947; Paulson, 1973). The 15 hues represent the entire color circle. The test can be used to demonstrate color confusion, the inability to distinguish colors on opposite sides of the color circle, such as red and green or blue and yellow.

The value of the Farnsworth tests was that they could be mass-produced at reasonable cost, could be transported easily, and could be performed by untrained observers. Also, the colors could be described according to accepted color specification, and a quantitative error score could be determined for the FM 100-hue test and a pass/fail dichotomy for the Panel D-15. Each of these factors combined to make the Farnsworth tests attractive for introduction to clinical testing. It is fitting that the inaugural issue of *Vision Research* was dedicated to Commander Dean Farnsworth. The photograph shown in Fig. 3 is in fact the same picture that was used for that inaugural issue. [In the succeeding issue, François and Verriest (1961) described their use of the FM 100-hue test in eye disease.]

Before the development of the Farnsworth tests there were rather few options available to clinicians. The screening plate tests developed for congenital color vision tests are not suitable for diagnosis of acquired color vision defects. A major limitation was that there were few plates for blue color vision defects. Screening plates were used primarily to demonstrate severe acquired red-green color vision defects. The color matching classification of monochromacy, dichromacy and trichromacy so important in the study of congenital color defects is less applicable to the study of acquired color vision defects. The alternative is psychophysical measurements of color discrimination. Such measurements are often not performed since they require specialized equipment with highly trained technical personnel. Further, the demand on the observer is also high, making the tests unsuitable for routine use in a patient population.

There was a decade of promising developments in the study of acquired color vision defects prior to the initial publication of *Vision Research*. Engelking's student Jaeger (Fig. 4) started a brilliant career in Heidelberg. Jaeger's contributions included studies of congenital color defects emphasizing the effects of field size in dichromacy (Jaeger and Kroker, 1952) as well as documenting residual color vision in achromatopsias (Jaeger, 1950, 1951, 1953, see also the recent review by Jaeger and Krastel, 1983). Jaeger studied the blue color defect in dominant optic atrophy—an apparent con-
tradiction to Köllner's rule (Jaeger, 1954) and defined the pseudo-protanomaly (red-shifted Rayleigh match) that occurs in central serous chorioidopathy (Jaeger and Nover, 1951).

Verriest (Fig. 5) began his studies of acquired color vision defects in the Eye Clinic in Ghent using the FM 100 hue and the Farnsworth Panel D-15 tests, studies to which in proper European fashion, his Professor assumed the senior authorship (summarized in François and Verriest, 1961). Based on these and other methods of examination, Verriest (1963) proposed a new classification for acquired color vision defects. Verriest's scheme included a Type I acquired red-green defect, characteristic of cone degenerations, a Type II acquired red-green defect characteristic of optic nerve disturbances (except dominant optic atrophy) and a Type III acquired blue-yellow defect. The Type III defect was the most frequent and occurred in many disorders including pigmentary retinal degenerations, glaucoma, papilledema and dominant optic atrophy, choriotretinal inflammations, retinal vascular disease and cataract. Wright in the preface to Pokorny et al. (1979) pointed out that the term "blue-yellow" is inaccurate since the defect does not involve confusions on the blue-yellow but on the violet-yellow axis. In such defects, discriminations mediated by short wavelength sensitive (or "blue") cones seem affected. A better term might be "blue" color defect or "violet-yellow" axis. Table 1 shows a revised version of Verriest's classification and in deference to Wright we follow his suggestion and call the defect the Type III blue defect.

Lakowski (Fig. 6) (Lakowski, 1958) published a landmark paper demonstrating that color discrimination performance was age-dependent. The etiology of this developmental deficit certainly includes the progressive yellowing of the lens as well as possible sensory and performance factors. Lakowski emphasized a variable which is of great importance in clinical assessment. In laboratory psychophysics individual variability is often ignored, one or two observers are usually considered sufficient to characterize a visual function. This approach is justified since great care is usually taken in the experimental design to ensure that the observer serves as a "null instrument". Clinical work in contrast involves a great diversity of observers and clinical tests often contain performance factors. There is a problem in deciding if poor performance on a color vision test is caused by eye disease or by some other factor. Lakowski used pseudoisochromatic plate tests and the Pickford–Nicolson anomaloscope (Pickford and Lakowski, 1960). He recalls that his paper received a less than enthusiastic reception by the English physicists and physiologists. Its importance however was immediately evident to Verriest (1963) who within 5 years, had published age norms for the FM 100-hue test.

Two laboratories began psychophysical measurements on patients with eye disease. Jaeger's student Grützner measured spectral sensitivity and chromatic discrimination functions in patients with eye disease, and showed the enlargement of the neutral zone centered at 580 nm in optic nerve disorders. He again emphasized the peculiar nature of dominant optic atrophy and used the anomaloscope to demonstrate the enlarged matching ranges and shift to scotopic matching in macular degenerations.

Table 1. Classification of acquired color vision defects*

<table>
<thead>
<tr>
<th>Verriest type</th>
<th>Discrimination defect</th>
<th>Visual acuity</th>
<th>Wald–Marré type</th>
<th>Disorder</th>
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<tbody>
<tr>
<td>Type I</td>
<td>Red-green</td>
<td>Mild</td>
<td>Moderate</td>
<td>IIb</td>
</tr>
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<td></td>
<td></td>
<td>Severe</td>
<td>Severe</td>
<td>III degeneration</td>
</tr>
<tr>
<td>Type II</td>
<td>Red-green + blue</td>
<td>Mild</td>
<td>Mild</td>
<td>IIIa</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate</td>
<td>Moderate</td>
<td>IIIb (post optic disc)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe</td>
<td>Severe</td>
<td>III</td>
</tr>
<tr>
<td>Type III</td>
<td>Blue + Pseudo-PA</td>
<td>Mild</td>
<td>Mild</td>
<td>I</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mild</td>
<td>Mild</td>
<td>I</td>
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<tr>
<td></td>
<td></td>
<td>Moderate</td>
<td>Moderate</td>
<td>Ib</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Pigmentary degeneration</td>
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<td></td>
<td>Glaucoma, papilledema</td>
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<td></td>
<td></td>
<td>Dominant optic atrophy</td>
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<td></td>
<td></td>
<td>Cataract</td>
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</tbody>
</table>

*This is a revised version of Verriest's classification (Verriest, 1963; Pokorny et al., 1979). The most usual correlations of Verriest defect, acuity, Wald–Marré defect and disease category are shown. There are always exceptions to be noted in individual cases.
Much of this work was summarized by Jaeger and Grützner (1963) and later by Grützner (1973). At the same time Cox, after training in Wright's laboratory started measurements of wavelength discrimination (Cox, 1960, 1961).

Many other investigators were active. We have tried to emphasize only those who were to set the stage for the next 25 years. Among those who have published reviews of the status of the field prior to the appearance of Vision Research, we can mention a few more names. Waardenburg, a Dutch geneticist interested in ophthalmic disorders collaborated with the Swiss ophthalmologist Franceschetti in a review of genetic disorders affecting the eye (Waardenburg et al., 1961). Franceschetti and François summarized the status of chorioretinal degenerations in a 1963 report to the Société Française d'Ophthalmologie. This report was translated into English and updated following Franceschetti's death, eventually appearing in English in 1974 (Franceschetti et al., 1974). Krill in his regrettably short career emphasized the importance of correlating the ophthalmoscopic appearance of the fundus with the assessment of color vision and the electrophysiological function in hereditary retinal disorders. His review of visual function in hereditary retinal disorders was finished by his colleagues (Krill, 1977).

COLOR DEFECT AND EYE DISEASE
THE LAST 25 YEARS

When Vision Research first appeared, the scene was set for a major expansion of research in the color defects accompanying eye disease. So much has happened that we cannot describe each of the individuals who are today engaged in clinical color vision research nor mention the many studies that have been published during the past 25 years. We prefer to highlight a few ideas, people and events that have set the direction for the future. These include the formation of a society whose members are devoted to research in color vision defects, the continued interest in development of easy and rapid color vision tests, the development of psychophysical tests based on particular advances in color vision theory, and advances in the use of color matching for the analysis of eye disease.

The International Research Group on Color Vision Deficiencies. This group originated at a meeting of the Association Internationale de Couleur in Stockholm in 1969. The original idea was to foster research and communication in the study of color vision defects by providing a forum of communication between clinician and basic scientist and between researchers of all nations. Additionally the directors of the Society urged the collaboration that resulted in the publication of Congenital and Acquired Color Vision Defects (Pokorny et al., 1979), summarizing methods and results in color defects since the time of Kollnér. The society has also been important in promoting the commercial development of anomaloscopes and new color tests.

The main activity of the I.R.G.C.V.D. is the biannual meeting. The first meeting held in 1971 in Ghent, Belgium was specifically devoted to acquired color vision defects. Since then, meetings have been held every two years in several European countries including Great Britain (twice), the Netherlands, Italy, Germany, Switzerland and France. The proceedings of these meetings constitute a record of the development of research in acquired color vision defects for the past 15 years (Verriest, 1972, 1974, 1976, 1978, 1980, 1982, 1984, 1986). As yet no meeting has been held in the Americas or Japan, but one is planned for the U.S. in 1987 and the membership has been invited to Argentina. The I.R.G.C.V.D. also sponsors local meetings in countries and localities to which the entire membership cannot travel. Local meetings have been held in East Germany and are planned for Japan.

The I.R.G.C.V.D. has provided a forum of communication between East and West. The Japanese have long been interested in color vision defects. Stillling invented the concept of the pseudoisochromatic plate in the late 1800s, publishing the first edition in 1878. Shinobu Ishihara, an ophthalmologist, designed his ingenious plates for detection of color defects while visiting Stillling's laboratories. In the 1960s, interest in basic color vision and congenital color vision defects was fostered by the visits of Japanese scientists to the U.S. scientists, for example Munehira Akita to Clarence H. Graham and Mitsuo Ikeda to Robert M. Boynton for their graduate work. Mathew Alpern has hosted a succession of eminent Japanese scholars including the Kitaharas, Ohba, and Torii. Yasuo Ohta has been an important influence in color vision research in Japan and has been active in the I.R.G.C.V.D. from its earliest days. More recently Hiroshi Ishikawa and his colleagues have made significant contributions to...
Fig. 1. Hans Köllner, M.D., an Assistant at the University Eye Clinic of Berlin.

Fig. 2. W. D. Wright, D. Sc., A.R.C.S., D.I.C., Emeritus Professor of Applied Optics at Imperial College, University of London.

Fig. 3. Dean Farnsworth, Commander in the U.S. Navy.
Fig. 4. Ernst Engelking, M.D. (seated), Professor of Ophthalmology at Heidelberg University Eye Clinic with his student Wolfgang Joeger, M.D., the present Professor at Heidelberg University Eye Clinic.

Fig. 5. Guy Verriest, M.D. of the Ophthalmology Department at the State University of Ghent.
Fig. 6. Romuald Lakowski, Ph.D. of the Psychology Department at the University of British Columbia.

Fig. 7. Marian Marrè, Doz. D. Sc. Med. of the Carl Gustav Carus Medical School, Dresden.

Fig. 8. The authors.
the study of oongenital and acquired color vision defects (Verriest, 1974, 1980, 1984). Thus one finds an active tradition of color vision research in Japan, including both basic and clinical color vision research. Unfortunately, relatively little of the Japanese research has yet appeared in English-language journals, the exceptions being the papers appearing in the proceedings of the I.R.G.C.V.D.

The meetings of the Society are characterised by an exceptional social program. Members have their favorite locations and memories. This year one member recalled for us with great fondness his visit to Verdi’s home near Parma. We remember a reception at the van Gogh museum in Amsterdam, where some 100 people had the entire museum to themselves.

Farnsworth tests. Farnsworth’s impact continues to be felt. Statistical analysis of FM 100-hue test errors (e.g. Kinnear, 1970; Aspinall, 1974), axis analysis (e.g. Helve, 1972; Smith et al., 1976; Kitahara, 1980; Smith et al., 1985), and new age norms (Verriest et al., 1982) have appeared. The FM 100-hue test is now universally acknowledged as the most sensitive test of chromatic discrimination available that is appropriate for widespread use in clinical populations. The test was recently given to over a thousand diabetic patients enrolled in a U.S. controlled clinical trial. New tests based on Farnsworth’s ideas have appeared. The most notable of these are Lanthony’s desaturated Panel D-15 and Lanthony’s New Color test (Lanthony, 1974; 1978a, b).

Screening tests. Screening plate tests are still widely used. The Hardy–Rand–Rittler Pseudoisochromatic plates, one of the few to contain plates for blue defects have been out of production for many years. The last few years have thus seen a search for new screening tests appropriate for acquired color vision defects. There are new pseudoisochromatic plates for acquired color vision defects (Ichikawa et al., 1983). Frisén and Kalm (1981) designed a screening test for acquired color vision defects using colored caps of varying saturation.

Wald Marré Functions. Marré (Fig. 7) (Marré, 1973; Marré and Marré, 1978) developed a technique to assess the sensitivity of the cone receptor mechanisms, termed “primary color vision mechanisms”. Her parameters were based on those used by Wald (1964), hence the name “Wald–Marré Color Vision Mechanisms”. Increment threshold spectral sensitivities on chromatic backgrounds provide reasonably good isolation of three color vision mechanisms over a fairly broad (100–150 nm) spectral region. These mechanisms represent the spectral sensitivities of the short (blue), medium (green) and long (red) wavelength sensitive receptors. She used a quantitative analysis to describe three types of color vision mechanism in patients. In Type I, the blue color vision mechanism is solely (Stage Ia) or primarily (Stage Ib) affected. The Type I Wald–Marré pattern corresponds to the Verriest Type III acquired blue defect (see Table 1). In Type II, the red and green color vision mechanisms are affected, either with a normal (Stage IIa) or less affected (Stage IIb) blue color vision mechanism. The Type II Wald–Marré pattern corresponds primarily to Verriest’s Type II acquired red-green defect (see Table 1). Finally in the Type III Wald–Marré pattern, all three color vision mechanisms are affected; this pattern corresponds to the most severe stages of Verriest’s Types I and II acquired color vision defects.

Marré and Marré (1978) correlated their results with the fixation preference, and presumably visual acuity, of their patients. Type Ia patterns are seen with foveal fixation; Types Ib and IIa are seen with parafoveal fixation and Types IIb and III with parafoveal or even extra-macular fixation. Pinckers and Marré (1983) presented a classification of acquired color vision defect relating Pincker’s (1982) functional classifications of retinal dystrophies and Marré’s studies of the effects of fixation preference to the observed acquired color vision defects. More recent developments of the Wald–Marré approach have included de Vries-de Mol’s (1977) development of a continuous tracking technique. Most recently Lutze, Pokorny and Smith (in press) optimized a number of parameters to improve the isolation of the color vision mechanisms as well as permit rapid acquisition of data by untrained observers.

Color matching. Color matching has been regarded as less useful in acquired color vision defects since clear-cut classification into trichromacy, dichromacy or monochromacy is rarely possible. However the von Kries analysis of abnormal color matching behavior can be applied to acquired color vision defects (Alpern et al., 1976). The past 25 years saw the appreciation of the von Kries mechanism of alteration in the analysis of pseudo-protopanomaly. Pseudo-protopanomaly (a red-shifted Rayleigh match) has been recognized since 1951 (Jaeger and Nover,
1951), as occurring in disorders of the choroid and pigment epithelium in which the sensory retina is elevated or inflamed. We showed (reviewed in Pokorny et al., 1980) that pseudo-protopanomaly is closely correlated with receptor disorientation in these disorders. Since choroidal disorders are often accomplished by a Verriest Type III acquired color vision defect, these findings permitted a subdivision of the Type III defect into those with pseudo-protopanomaly and those with a normal Rayleigh match (Table 1).

Another advance in color matching in the 1960s was the development of special purpose equations for the anomaloscope. Pickford and Labowski (1960) used three equations in the Pickford–Nicolson anomaloscope. In addition to the Rayleigh (a match of “red” plus “green” to “yellow”) equation for red-green defects, there were the Engelking–Trendelenburg (a match of “blue” plus “green” to “blue-green”) and the Pickford–Lakowski (a match of “blue” plus “yellow” to “white”) equations. It is clear from their work that these latter equations were very sensitive to aging (Lakowski, 1971) and to acquired color vision defects in diabetic (Lakowski et al., 1973) and glaucoma (Lakowski and Drance, 1979). On theoretic grounds, both equations are also very sensitive to variation in macular pigment density in the fovea centralis (Wright, 1946). The Engelking–Trendelenburg equation was designed to diagnose tritanomaly. However Wright’s (1952) search for tritan observers had not revealed definite tritanomaly and Krill et al. (1970) did not find evidence of tritanomaly in a review of the intervening years. Moreland and Kerr (1978) undertook an experimental study to determine the set of primaries that would minimize individual variability in a tritan equation. The result was the proposed equation

\[ 480 \text{ nm} + 380 \text{ nm} = 439 \text{ nm} + 500 \text{ nm}. \]  

This equation minimizes macular pigment variation (Moreland and Kerr, 1978) and to a lesser extent, lens variation (Zaidi et al., 1982). However, the equation remains sensitive to lens variation and hence to age. Pokorny et al. (1981) preferred to substitute 430 nm for the blue primary since the pair of primaries 430 and 500 nm are near a tritan confusion pair. The discriminations of tritan pairs involve only short wavelength sensitive cones (see Boynton and Kambe, 1980). The Moreland equation has replaced the Engelking–Trendelenburg equation for evaluation of tritan and acquired blue defects.

A number of new anomaloscopes have appeared. In Japan, Ohta and his colleagues (Ohta et al., 1980) developed an interference filter version of the Nagel anomaloscope. This anomaloscope has a number of engineering improvements which make it stable and easy to use. Roth and his colleagues (Roth et al., 1982) developed a filter anomaloscope which allows not only the Rayleigh, but also special purpose equations. Jaeger and his colleagues (Jaeger et al., 1982) and Moreland (Moreland and Young, 1974) also designed multi-purpose anomaloscopes, but neither is yet commercially available.

In addition to new multi-purpose anomaloscopes, there has been an attempt to develop portable anomaloscopes to assess the Rayleigh equation. These have used light emitting diodes (LEDs) as primaries but have not been successful since the commercially available LEDs were not spectrally suitable (Pokorny and Smith, 1984). A recent design (Krastel et al., 1986) promises to surmount these problems and we may soon see a vest-pocket anomaloscope.

Application of modern color theory. An important direction of research depends on advances in basic color vision theory. As pointed out in previous papers in this issue, it is now agreed that color signals are processed in the nervous system in an opponent fashion. It has become customary to speak of the Chromatic Opponent Channels—a red-green opponent channel and a blue-yellow opponent channel. Specific tests have been developed to seek evidence of opponent channel activity in normal individuals (Sperling and Harwerth, 1971; King-Smith, 1975). This approach has now been extended to acquired color vision defects with particular application to optic nerve disorders (King-Smith et al., 1976; King-Smith et al., 1980; Zrenner and Kruger, 1981; Alvarez et al., 1982) and glaucoma (Adams et al., 1982).

The ubiquity of the Verriest Type III acquired blue defect has led investigators to question whether short wavelength sensitive cones are more liable to acquired damage. This question became a major topic of interest at the 1981 Berlin meeting of the International Research Group on Color Vision Deficiencies (Verriest, 1982). A number of investigators remain interested in delineating the loci of acquired blue defects.
SUMMARY

The groundwork for understanding color defects in eye disease was established by the end of the nineteenth century. Thereafter the field was neglected as scientists concentrated on studies of normal color vision and congenital color vision defects. Spurred by the development of the Farnsworth 100 hue-test, interest was renewed in the 1950s. The past 25 years have seen an explosion of interest in color defects in eye disease. The International Research Group on Color Vision Deficiencies has played an important role in this activity. The development of new clinical tests and instruments as well as refinement of laboratory techniques are among the important developments.

REFERENCES


