Congenital Color Blindness

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By Henry Cadan, Brooklyn, N.Y.
11 Plaza St.

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CONGENITAL COLOR BLINDNESS

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This article advances no claims as to the cure for congenital color blindness. It is written for the sole purpose of calling to the attention of the medical profession the fact that constructive research may be possible in color blindness in order to rehabilitate that group of congenital color defectives who are prevented from pursuing those fields of endeavor in which they are best qualified.

The subject is timely, in view of the fact that the physical requirements in almost all the branches of military service demand normal color perception. Fortunately, the percentage of color blindness found at recruiting stations is low enough to prevent a serious shortage in our armed forces. Yet, because of this handicap, many men of otherwise perfect officer calibre are prevented from serving in those departments in which they would find themselves to be best qualified.

Congenital color blindness has probably existed as long as the human race. However, the first recorded case known to authors dates back to the *Philosophical Transactions* of 1777, and is that of a shoemaker who could not distinguish certain colors. The next best known case in the last century is that of the English chemist, Dalton, who was red-blind, and the first to study his color sense by the spectrum. The first who entered into a systematic study of this subject was Siebeck. He used colored papers and glasses, sometimes prismatic diffraction colors and colored worsted.

Although Siebeck covered almost the whole ground as far as the theoretical side of the subject was concerned, the practical side was untouched until George Wilson published a series of papers read before the Royal Scottish Society of Arts in 1855, under the title *On Railway and Ship Signals in Relation to Color Blindness*. In 1877 Holmgren published his work on color blindness and its relation to railroads and maritime conditions.

In November, 1880, the Council of the Ophthalmological Society of the United Kingdom appointed a committee to consider defects of sight in relation to public safety, with the result that among 18,088 persons at different periods of life and in various social positions, 4.76 percent of males and 0.4 percent of females were found to be color blind to some extent.

It is easy enough to define color blindness as that defect of the eye through which it is unable to distinguish between certain colors, while in other respects the eye is normal. The greatest difficulty arises when we undertake to classify the different kinds.

In May's *Diseases of the Eye,* the principal theories advanced to explain color vision and its derangements are those of Young-Helmholtz, Hering and Edridge-Green.

The Young-Helmholtz theory assumes that there are three sets of color-perceiving elements in the retina, each of which, if stimulated alone, would give rise to a sensation of one of the fundamental colors, namely, red, green and violet, and that all other colors arise from a combination of these. With a defect of one of these primary perceptions, a color will be seen as if composed of the remaining two only. According to the color which is deficient, the patient is said to be red blind, green blind or violet blind.

The Hering theory is that the color sense depends upon the chemical changes in the three different visual substances in the retina, namely white-black, red-green and blue-yellow, by the decomposition and restoration of which substances the sensations of color are produced. According to this theory, color blindness is caused by the absence of one or two of these visual substances. If one is absent, the patient is either red-green blind (frequent) or blue-yellow blind (rare). If two are absent, nothing but the white-black substance is left, and the patient has total color blindness, everything appearing grey.

The Edridge-Green theory supposes that a photograph is formed in the retina by the decomposition of the visual purple in the rods. This chemically stimulates the ends of the cones, causing a visual impulse to be transmitted through the optic nerve fibres to the brain. It assumes that this impulse differs in quality according to the wave length (color) of the rays of light producing it. There is a special centre in the brain to distinguish these differences.

Regardless of the theory one accepts, it is generally conceded by the various authors that color perception depends, therefore, on three vital factors:

1. A photochemical substance in the retina, which may be the visual purple, or closely associated with it; 2. the afferent nerves, which convey the impulse through the optic nerve to, 3, that area of the brain cells, probably the cuneus, whose specific energy is for us identical with a sensation of color.

We conclude from the foregoing, therefore, that the retina is only the selecting organ, while the brain alone is the perceiving one.

With regard to photochemical substances, it must be admitted that they are only hypothetical. But, as we have already found one substance in the retina that is very sensitive to light, i.e., the visual purple, we may well suppose that there are other chemical substances yet to be discovered by actual experiment. Perhaps we may find that the visual purple itself plays an

important part in color vision, as suggested by Ebbinghaus.

Since visual purple consists of rhodopsin and iodopsin, which two substances can further be identified as a protein and vitamin A. Deficiencies of vitamin A, therefore, could play an important part in defective color perception. It has never been established just where the defect lies in congenital color blindness. Opinion is divided as to whether the defect lies in the retina or in the brain. Because of such a divided opinion, I decided to attack the problem empirically, using any therapy which, in my opinion, would aid in correcting the defect in the retina or in the brain.

1. Vitamin A, already mentioned, is a factor in the visual purple metabolism.

2. Vitamin B complex, although not clearly established, is a factor in nerve tissue metabolism.

3. Iodine, to stimulate the general body metabolism and because of its antisclerotic effect.

4. Electrical stimulation in the form of low frequency-low tension currents, the physiological effects of which produce a circulatory enhancement, thereby accelerating metabolism of the tissues to which the current is applied. There is also an increase in the neurogenic response. The low frequency-low voltage currents directly stimulate the motor and sensory nerve endings.

In the case of stimulation of the eye, the following objective and subjective results would occur:

(a) A contraction of both the voluntary and involuntary musculature in the area of the electrode.

(b) A conductivity to the brain of the various nerve sensations. When applied to the eye, a patient experiences a sensation of burning, heat and light-flashes with each make and break of the current, especially marked when interrupted faradism is used.

Because of the seriousness of the undertaking to rehabilitate rejects for defective color perception, I felt that those who could differentiate the primary colors, but not the shades of color whose luminosities were fainter, would be most suitable for experimentation.

There are many rejects of the socalled red-green blind class who can distinguish primary color, but who fail in distinguishing the numerals or characters in the pseudo-isochromatic charts of Stilling, or the charts originated by Ishihara.

Forty-five patients suffering from mild red-green blindness were treated. After six to fifteen treatments, thirty-five passed a re-examination in various branches of military service, having previously been rejected because of defective color perception. Two patients improved, although not sufficiently to be judged normal. Eight patients discontinued after one or two treatments.

On re-examination of those patients previously rejected by the examining physician, all standard methods for detecting color blindness were used. The methods used included the Edridge-Green lights, the worsted yarns and the charts of Stilling and Ishihara.

Method of Treatment

1. Seventy-five thousand units of vitamin A daily by mouth.

2. Subcutaneous injections of one-half c.c. of vitamin B complex plus additional vitamin B complex by mouth.

3. Five drops of tincture of iodine by mouth, once daily, during the course of treatment.

4. Low voltage-low frequency therapy, consisting of five minutes of interrupted faradism, and ten minutes of sinusoidal current to each eye and to toleration.

5. An intensive study of colors and color patterns during the course of treatment.

The attempt in the above treatment was first to correct the metabolism of the color substance in the retina and, secondly, to attempt an education of the color perception area of the brain.

Before commencing treatment, an ophthalmoscopic examination of the eye should be made. Arteriosclerosis and vascular diseases contraindicate treatment by electrotherapy because of the increase of the circulation and increase of local pressure of the blood in and around the area of the eye during the course of treatment. Fortunately, the age group applying for treatment rarely showed vascular degenerative changes.

In view of the fact that the subjects of my experiments have been of such recent origin, it is impossible to prognosticate at this time as to just how long the improved condition will sustain itself. However, those subjects who were found to be normal in December, 1941, were re-examined in July, 1942, and still showed normal color perception.

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