is produced by the product of a physical process only, as distinct from an enzymatic or chemical one, as we normally conceive of it. The observation of a thermal luminescence and semiconductivity for dried chloroplasts has recently been reported and interpreted as consistent with the idea that the chloroplasts have some of the properties of semiconductors (*8*).

The asymmetry of the signal itself (*7*), together with the fact that the decay and probably the growth is not a simple, single logarithmic one even at −140°C, seems to indicate that more than one species is responsible for the over-all signal which we see. Among these species may be the chlorophyll triplet (as mentioned earlier), the trapped electron, and, finally, some species of free radical resulting from the direct dissociation of a chemical bond in the absorption act. While it is possible to suppose that the cooling would enhance the lifetime of the chlorophyll triplet to the extent of hours, it does not seem likely. We are thus left with the trapped electron and the possibilities of a dissociated bond.

It is perhaps worth noting that, whatever the nature of the unpaired electron producing this signal, its coupling with the lattice around it must be rather poor in order that it can produce a signal as narrow as the one we see. This suggests that it is located in a rather delocalized pi-type of orbital. It is to be expected that improvements in technique will lead to a more precise identification of the variety of unpaired electrons which almost certainly result from the illumination of the photochemical apparatus in plants.

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Effect of Psychiatric Disorder on Visual Thresholds

Following a wartime observation that neurotic patients tended to obtain lower scores than normal subjects on tests of "night vision," several experiments (*1*) have been undertaken in which the night vision of normal, neurotic, and psychotic groups has been compared. A fairly consistent finding of this research is that, on the average, psychiatric patients have raised intensity thresholds during the course of dark adaptation (*2*).

The most general effect of psychiatric disorder is, apparently, to elevate the dark-adaptation curve along the log-luminance axis without changing its shape (see Fig. 1). The extent of the displacement varies somewhat from one investigation to another between about 0.1 and 0.5 $\log_{10}$ unit. The effect of psychiatric disorder seems to be equivalent to that of placing a neutral density filter in front of the subject's eyes, thus necessitating a greater amount of light to produce a threshold response.

In this respect, the effect of psychiatric disorder may be compared with that of anoxia, which also tends to shift the dark-adaptation curve along the intensity axis without affecting its shape, by amounts varying between 0.1 and 0.4 $\log_{10}$ unit, depending on the degree of anoxia (*3*). A similar effect has been produced by insulin hypoglycemia (*4*) and by alcohol (*5*). In all three cases, the most plausible explanation of the effect seems to be that these conditions lead to a depression of central nervous activity, either in the retina or at higher levels of the visual system.

It seems possible that some of the effects of psychiatric disorder on visual thresholds may also reflect changes in the nervous system of a similar type. Oxygen deficiency has from time to time been reported to occur in psychotics and to a lesser extent in neurotics (*6*), while Gellhorn *et al.* (*7*) have reported more insulin in psychotic patients than in normal subjects, under stress conditions. More recently Shagass and Naiman (*8*) have provided evidence of a reduced central excitability in hysterics, as determined by their "sedation thresholds" for sodium amytal. Although the evidence on the physiological side is far from satisfactory, it would seem that a hypothesis of lowered central excitability in the visual system merits at least as much consideration as possible alternative explanations of a more psychological nature phrased in perceptual terms (for discussion, see *2*).

The implications of the experimental results and the suggested hypothesis may be considered from several points of view. As far as visual research on dark adaptation is concerned, it should be noted that psychiatric disorder does not in general alter either the *shape* of the dark-adaptation curve or the total *range* of adaptation; hence, it seems unlikely that the actual mechanism of adaptation is affected. The shift of the curve along the intensity axis can be accounted for in terms of a depression of activity in the visual pathways, completely unrelated to the dark-adaptation process itself. The results do not, therefore, conflict with the photochemical theories of dark adaptation proposed by Hecht and others, nor do they provide any evidence for neural mechanisms in dark adaptation. They may, however, like the studies of anoxia, suggest the need for a broader basis for visual theories by forcing photochemical theorists to make explicit and develop factors that are implicit in their equations and regarded as secondary or constant (for example, the efficiency with which the conducting system deals with the products of photochemical reactions).

From the broader viewpoint of psychiatry, abnormal psychology, and neurophysiology, the results are perhaps of greater interest, for they may provide further evidence of the value of visual thresholds as indicators of changes of excitability of the central nervous system. As McFarland (*9*) has pointed out, visual thresholds appear to have certain advantages over other psychophysiological functions as quantitative indices of "physiological imbalance" with regard to sensitivity, to the precision with which the physical measurements involved can be made, and to the fact that the subject is not aware of the changes in the physical intensity of the stimulus that are necessary for him to see it, since at threshold the stimulus always has the same appearance. However, like critical flicker frequency (CFF), now being widely used for this purpose, absolute thresholds taken during dark adaptation are not specifically related to any particular physiological stress or abnormal condition. This would seem to reduce their practical value in psychiatric diagnosis.

From the results so far obtained, there seems no immediate likelihood of distinguishing, on the basis of threshold measurements alone, psychotics from neurotics, hysterics from depressives and psychopaths, or, for that matter, psychiatric patients suffering from functional disorders from patients having various organic diseases. In consequence, considerable caution is necessary when results are interpreted from the psychiatric viewpoint and, as Simonson and Brozek (*10*) point out elsewhere in their review of studies of critical flicker frequency, "As a rule, additional information obtained by independent methods is desirable for more specific correlations."

In spite of this, the possibility should not be overlooked that more detailed ex-

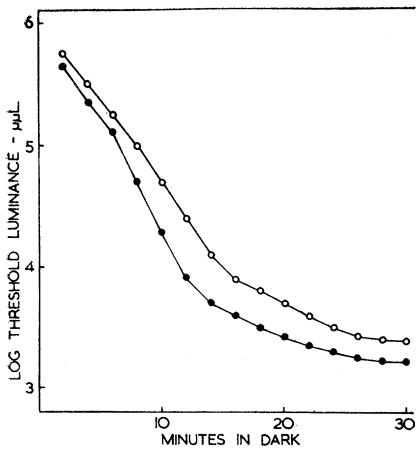

Fig. 1. Mean dark-adaptation curves: ○, psychiatric patients; ●, normal subjects (see *2*).

perimental analysis of the responses of different psychiatric categories may reveal at least statistically significant differences between certain diagnostic groups, not only with respect to the position of the dark-adaptation curve on the intensity axis, but also with respect to its shape. Observations in two experiments (*11*) suggest, for instance, that hysterics and anxiety states may differ in the level of their dark-adaptation curves, and one or two hysterics have produced curves that differ in slope from the normal. Such differences suggest that, at least in certain cases, the mechanism of dark adaptation may be impaired, and detailed investigation of these cases may be of considerable interest from the viewpoint of visual research as well as psychiatry. It is possible, for instance, that neural mechanisms operating in dark adaptation may be affected, perhaps via the centrifugal fibers known to exist in the optic nerve. In view of recent work on the "reticular activating system," both in regard to centrifugal control of the sense organs and its role in psychiatric disorder (*12*), such a possibility cannot be dismissed. Worth noting also is the further possibility that differences in dark-adaptation thresholds between anxiety states and hysterics may be paralleled by differences observed between normal subjects classified according to temperamental type (*13*).

With regard to the experimental implications of the general hypothesis suggested in a preceding paragraph, one might expect, on the basis of previous psychophysical research on vision, that changes would occur in the curves that relate a number of visual functions other than absolute thresholds to intensity. Thus, one would expect the curves that relate critical flicker frequency, brightness discrimination, and visual acuity also to be shifted along the intensity axis by psychiatric disorder. The extent of the shift should in each case be similar to the amount of displacement of the dark-adaptation curve, since it is a function of the density of the hypothetical filter that was assumed to have been placed in front of psychiatric patients' eyes. These "deductions" are all susceptible to experimental test.

One final point that requires emphasis concerns the magnitude of the differences so far obtained that can be attributed to the effects of psychiatric disorder. As was pointed out in a preceding paragraph, in certain cases the difference amounts to as much as 0.5 $\log_{10}$ unit, but in other cases it may be as little as 0.1 $\log_{10}$ unit. Although the differences were statistically significant under the experimental conditions in which they were observed, they must be related to the whole body of knowledge concerning the visual mechanism if they are to be seen in proper perspective. Compared with the enormous range of values (1 to 10^6) through which the visual threshold can vary during light and dark adaptation, the changes are very small indeed. Because it seems likely from studies of physiological stresses (*14*) that the observed changes represent the limits of variation that can be expected from disturbances central to the photochemical system, it is clear that refined experimental techniques will be necessary to measure them accurately and to assess their significance in relation to physical and other sources of variation, including individual day-to-day variation, which is often of the order of 0.3 $\log_{10}$ unit (*15*).

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Histochemical Distribution Pattern of Respiratory Enzymes in the Liver Lobule

Knowledge concerning the function and distribution of enzymes has been gained primarily from procedures involving the structural disintegration of tissue. However, recently developed methods have permitted the localization of specific enzymes in tissue sections. This study deals with the histochemical distribution pattern of four respiratory enzymes—succinic dehydrogenase, cytochrome oxidase, diphosphopyridine nucleotide diaphorase (DPN-diaphorase), and triphosphopyridine nucleotide diaphorase (TPN-diaphorase), respectively—in the liver lobule of higher mammals.

The enzymes concerned are intimately linked to the tricarboxylic acid cycle, which is believed to be the major pathway for the oxidation of many metabolites. Hence, the histochemical study of these enzymes may provide some general information concerning the structural organization of tissue respiration. Furthermore, it may help to elucidate the biochemical anatomy of the liver and its possible significance in the development of localized lesions in liver injury.

The histochemical demonstration of the succinic dehydrogenase system (*1, 2*) and of the DPN- and TPN-diaphorases (*3*) is based on the enzymatic reduction of tetrazolium salts, which serve as indicators of local reductase activity. On reduction, the water-soluble, colorless tetrazolium is converted into a water-insoluble, colored formazan at the site of enzyme activity. The intensity of staining appears to give a rough indication of the amount of enzyme activity present. To achieve specificity of the individual assay, tissue sections are incubated in a medium that allows only the particular enzyme under study to operate on the reduction of the added tetrazolium salt. These methods satisfy most of the criteria that have been adopted for the specificity and reliability of histochemical stains, and they are thoroughly discussed by Farber, Sternberg, and Dunlap (*3*).