ON SEEING.

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In the present communication it is intended to consider the fundamental formula of mental excitation processes,

$$H + L = T,$$

in terms of seeing. The equation implies that the characters of a light stimulus L are judged through a second factor H. T is the response of the responding organ (1, 3).

We consider first the known fact that we cannot see clearly insufficiently illuminated objects, and deduce from this that the judge, H, must have sufficient data, or L, to obtain a clear and distinct mental picture of the thing looked at. We thus group together, as mediated by too little L, vague shadowy outlines, dim memories (4), the music of the too-far-away band, and vague sensations of discomfort or subliminal pain (5). Vagueness, then, implies generally too little L.

In the next stage L increases sufficiently to give adequate data for the judge H, and we subdivide the stage into the conscious and subconscious, the latter merging into the former as more H is added by attention. But we cannot predicate any dividing line between one stage and another. Indeed it is a matter of common experience that extra attention, or more H, can make clear what might otherwise be obscure, so that the amount of H probably makes the difference between seeing and observing.* Clear conscious vision, then, implies the application centrally of adequate H to adequate L, received from the periphery.

If L, or the illumination, go on increasing, a stage will be reached where L has grown so large as to leave no room for adequate H. Clear conscious vision of the object whence light comes then ceases to be possible. We obtain consciousness of plenty of light, but lack the power to judge accurately its source. This stage may be called the glare stage.

Finally, L can be so large as to leave no room at all for H. This stage is one of blindness, and it should be noted that our analysis shows two kinds of blindness. In the one the light-receiving

* L should, however, be increased by the time of extra attention.

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apparatus sends either inadequate or no data to the visual centre; in the other, the eye sends to the brain impulses of a quality which prevent the application to them of the factor mediating consciousness.

The reader has probably noted that this consideration of seeing makes clear conscious vision a central phenomenon, whereas, as is well known, it certainly depends also on retinal structure. There is, however, no conflict here, because our result implies that normally only impulses arising from the fovea centralis contain that adequate L to which can be applied centrally the adequate H for clear psychic vision. The impulses from other parts of the retina must either contain too much L to permit of adequate application of H, or else so little L that no amount of H can give a correct judgment from the inadequate data supplied. Light stimuli of a given intensity must thus produce different amounts, or types, of change in the periphery and centre of the retina respectively, and so our next problem is to determine, if possible, the nature of these differences.

The solution of this problem is given, I think, by the dark-adapted or scotopic eye, which, as is well known, gives more distinct vision at the periphery than at the centre. If, then, stimuli of subnormal intensity give at the periphery impulses with adequate L, it follows that normal stimuli will give at the periphery impulses with too much L to apply to them the adequate H for clear conscious vision.

The conclusion reached here that stimuli of a given strength produce different amounts of effect at the periphery and centre of the retina respectively is only partly in accord with current physiological conceptions, in that it does not require, as these do, any difference in the relative amounts of change produced in the two retinal areas by light of different strength. For current explanations of the difference between photopic and scotopic vision would make the periphery more sensitive to dim lights and the centre the more sensitive to the strong. If these explanations were correct there should be some illumination between dim and strong light at which all parts of the eye see things equally well, but though mankind in general, and philosophers in particular, have for thousands of years occasionally watched, or observed, day fade into night and the night pass to day, none has yet recorded that while light was waxing or waning a point was reached where all things in the field of vision seemed equally clear. Such a striking phenomenon would certainly have been noted by somebody at some time or other, and never having heard of it, I conclude it does not exist. At the same time, it should be noted, no other hypotheses than those above called current were possible so long as excitation processes were considered to differ one from the other solely in point of size. My

work shows they possess differences of composition as well as of size, and those differences of composition make all the difference to our interpretations of the facts.(2)

The next inference is that these differing responses to stimuli of equal strength at the peripheral and central parts respectively must be due to differences in the normal balance between ions and colloids in these tissues, the colloids of the peripheral parts possessing a finer state of subdividision when at rest than do the colloids of the central tissues, which, however, in balance, possess more Ca.(2)

If we conceive next the possibility that the light-sensitive elements of the retina could become regularly more and more calcified, such calcification, as work on the heart shows, would be accompanied by a corresponding reduction in the fineness of the state of colloidal aggregation and an increasing resistance to environmental change, which would culminate eventually in stimuli of ordinary intensity failing to excite.(2)

In any system of elements thus gradually becoming less responsive to environmental change, the first to lose that responsiveness entirely will be those which were least responsive at the start and, in the eye, the least responsive normally is the fovea. Hence, if the light-perceiving elements of the retina could become gradually more and more calcified, a stage would eventually be reached where the centre parts of the eye would fail to respond to light or be blind. A drug well known to give central blindness is nicotine, and turning to work which I have done to find out what this drug does to the heart when used in such amounts as smokers or tobacco-chewers get into their systems, we find that nicotine produces two effects, *viz.*, (1) it calcifies the tissues, (2) it renders the tissues much less responsive to environmental change (1). In smoking we do not take enough nicotine to reduce the output of our neural centres, but just that amount which will make them less responsive to environmental change. The smoking author, for example, is thereby enabled to carry on the work in hand without undue distraction by life's little worries (1). The sufferer from tobacco amblyopia, however, has increased too much the resistance of his fovea to environmental change, or light, but should yet possess around the central scotoma an area of clear vision, which should be better than that normally existing there, though I do not know of any observation showing this is so.

The blindness of tobacco amblyopia is thus due to a general loss of responsiveness of the retina to light, the part normally least responsive being the first to cease responding. An entirely different type of blindness would ensue if the retina were generally sensitized to light.

In the latter case moderate sensitization would cause light stimuli of normal intensity to produce as much effect as strong light produces in a normal eye, so that, just as a normal eye is photophobic to strong light, this abnormal eye will be photophobic to normal light, though when we speak of photophobia we imply the reaction of an abnormal eye to normal light.

Photophobia, then, implies sensitization of the retinal elements to light, and if that sensitization go far enough we shall reach a stage where ordinary light is blinding. If, however, all parts of the retina obtain equal increments of sensitization, then the part of the eye which is normally most sensitive to light will be the first to go blind. The periphery, being more sensitive than the centre, would thus be the first to go blind.

But we have used the terms "centre" and "periphery" above as though they were sharply demarcated parts of the retina, whereas we only know one extreme of each. We cannot say precisely where the centre and the periphery are in contact. Accordingly, while we deduce that some part of the periphery, the most sensitive, will go "blind" first, we cannot say definitely from our analysis where that most sensitive part is. If, however, we assume there is a gradual increase of retinal sensitiveness from its centre to its peripheral border, then the blindness we are considering would be shown as a contraction of the field of vision. Since this blindness can be caused by too great an emotional tone in the nerve impulses, we should expect to find it in the hysterical, which we do.

A contraction of the field of vision, due to the causes here considered, should culminate in complete blindness, but the blindness would not be due to loss of retinal capacity to react to light. There would be, instead, such an alteration in the quality of the nerve impulses as would not permit of their being brought to consciousness. Subconscious impulses should remain intact.

Pressure, as is well known, can also excite the elements of the retina, so that with a gradual increase of intra-ocular pressure, as in glaucoma, we could expect also such a gradual contraction of the visual field as occurs.

References.—(1) Burridge, W., *Arch. Internat. de Pharm. et de Thérap.*, 1926, xxxii, p. 216.—(2) *Idem, Quart. Journ. Exper. Physiol.*, 1928, xviii, p. 315.—(3) *Idem, Journ. Ment. Sci.*, July, 1929, lxxv, p. 371.—(4) *Idem, ibid.*, January, 1930, p. 96.—(5) *Idem, ibid.*, January, 1930, p. 101.