than 5 seconds in trials 2 to 4; 14 animals were excluded for this reason.
8. A number of animals jumped completely out of the apparatus in trials 5 to 7. This behavior, which never occurred in the trials before treatment, was considered a strong indication of retention; such responses were therefore treated as equivalent to maximum step-down latencies and are accordingly included in the 10-second criterion shown in Fig. 1.
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12. Work supported by grants from the Medical Research Council, London. We thank Dr. L. Weiskrantz for advice.

10 October 1966

Comparison of the Effects of Striate Cortex and Retinal Lesions on Visual Acuity in the Monkey

Abstract. *Acuity falls sharply and predictably in man as fixation is shifted away from the test stimulus. If the same "eccentricity" function applies to the monkey, then it can be shown that striate cortex lesions produce a smaller acuity impairment than is predicted by electrophysiological maps of the projection of retina onto the cortex. It is seen in this study that retinal lesions of the fovea and adjacent parafovea produce a more severe drop in acuity than corresponding cortical lesions, and therefore the surprisingly slight effects of the latter cannot be explained in terms of a relatively higher parafoveal acuity in the monkey. The discrepancy between retinal and cortical effects is unlikely to be due to the development of "supersensitivity" at the edge of the cortical lesions. An explanation is proposed in terms of lateral spread of information at retinal and/or geniculate stages of the visual system.*

It is perhaps not surprising that so little quantitative work has been carried out to assess the effects of partial lesions of the "visual" cortex (area 17) in animals, since this would involve the determination of changes in restricted regions of visual space. Considerable progress has been achieved in measuring the detection of small, brief flashes of light (*1*) in restricted regions of the visual field, but the method is very time-consuming. An alternative approach was also undertaken by us based on a simpler but less direct method of testing (*2*). In man, visual acuity falls off sharply and predictably as the stimuli are shifted away from the fovea (Fig. 1, solid curve), this function being roughly correlated with the relative density of cones (*3*). A bilateral lesion of that portion of a monkey's striate cortex to which the macula projects ought, therefore, to produce a measurable drop in acuity. The actual drop in acuity can be used as the basis for an inference about the size of the actual field defect and would also allow one to test various aspects of the point-to-point theory of retino-cortical projection. The present study extends our earlier results and, more importantly, by the inclusion of retinal lesions, establishes a base line against which to assess the cortical effects.

In our first study (*2*) it was found that the drop in "minimal separable" acuity produced by appropriate striate cortical lesions in rhesus monkeys (*Macaca mulatta*) was less than that predicted if the lesion produced an absolute scotoma for striped patterns and

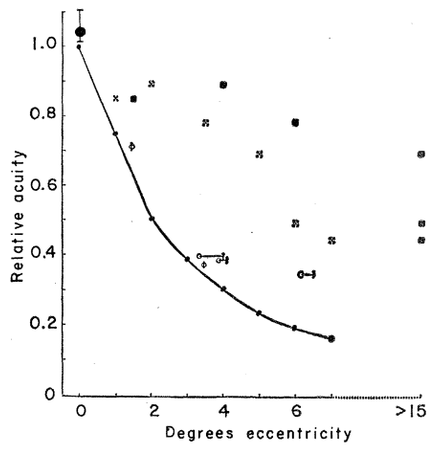

Fig. 1. Relative acuity as a function of distance of test stimulus from fovea. Solid curve: function for normal human observers with identical test situation as used with monkeys. Open circles: relative acuity following retinal lesions of given radius, measured from destroyed fovea as center. Vertical strokes indicate maximum radius, as described in text; ×'s, relative acuity following striate cortex lesions for fields estimated from Talbot and Marshall (*8*); squares, relative acuity following striate cortex lesions for fields estimated from Daniel and Whitteridge (*7*).

if the relation between foveal and parafoveal acuity is the same in monkeys as in man. That is, we estimated the predicted size of field defect from the electrophysiological map of the projection of the retina onto the cortex (Fig. 2) and compared the reduction in acuity with the value predicted by the human eccentricity curve shown in Fig. 1. It will be seen that the human curve falls below all of the points actually obtained (as in Fig. 1).

But, of course, it is possible that the parafoveal acuity of the monkey is relatively better than that of man, although there are no anatomical grounds for any such expectation. Accordingly, we carried out the same type of study with rhesus monkeys that had been subjected to binocular retinal lesions. As before (*2*), the acuity was measured by determining the density of vertical stripes which could just be discriminated from a homogeneous field equated in flux. The stripes were Moiré fringes generated by pairs of diffraction gratings, placed at a distance of 1 m from the animal's testing cage. The animal could secure food reward by pulling in the appropriate stimulus trolley. The width of the stripes was varied in a systematic fashion (according to a program usually called a "titration schedule") until a rigid criterion of stability had been achieved over 600 to 800 trials. Details have been published (*2*).

The acuity was tested preoperatively and again postoperatively, and the results expressed for each of six animals in terms of "percentage relative acuity," which is the ratio of preoperative to postoperative acuity × 100. Mean preoperative acuity was 0.60 minute of arc, with a group range from 0.54 to 0.69 minute.

The retinal lesions were made by a Zeiss xenon-arc photocoagulator, while the animals were anesthetized with Nembutal and while their pupils were dilated. The lesions were reconstructed from photographs of the retinas in the anesthetized animals. Several views were taken of each eye, and only those with the lesion centrally placed were accepted as being optically undistorted. Large composite projections were made of each fundus and these were used to assess the size and locus of each lesion, and more particularly, the shortest distance between the edge of the intact retina and the estimated point of the destroyed fovea. In previous measurements in intact animals, in which the perimeter (*1*) and a measur-

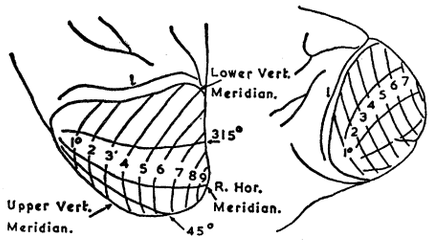

Fig. 2. Electrophysiological map of monkey striate cortex, as adapted from Talbot and Marshall (*8*).

ing ophthalmoscope were used, it was determined that the distance between the center of the disc and the fovea lay within a range of 17° and 18°, and 18° was taken as the distance in subsequent calculations, since that yielded the largest estimate of retinal size (and hence the smallest difference between retinal and cortical lesion results). In one animal ("Dart," Fig. 3) with a very small lesion falling directly on the fovea, it was found that the fovea-center disc distance was 3 times the short axis of the disc and 2.25 times the long axis of the disc, ratios which conform closely with that measured by us from sketches of the monkey retinas by Polyak (*4*). On the assumption of a center disc to fovea distance of 18°, the disc turns out to be 6° by 8°, which is close to the dimensions assessed directly by us in the monkey with a measuring ophthalmoscope.

We assumed the fovea in all animals to be 3 short disc diameters or 2.25 long disc diameters away from the center of the disc, and arcs with these radii were drawn on the composite projections. The two arcs invariably lay close together, as they should have done, and a mean radius of the two was calculated. The position of the fovea on the mean arc was determined by examining the distribution of retinal blood vessels, since the fovea lies in the center of a small region which is free of blood vessels. This can be estimated within fairly close limits, but in our final determination we also included a value for the *maximum* distance between a point on the arc and the edge of the functional retina, to allow an estimate of the largest possible lesion.

The metric for measuring effective lesion size was derived from an average of three independent measurements in each eye, based on the assumption that the short axis of the disc is 6°, that the long axis is 8°, and the edge of the disc to the fovea is 15° (since the edge can be measured more accurately than the center). Finally, two further relevant facts must be mentioned, which were taken into account in our estimates. First, the lesion was assumed to extend to the farthest visible edge, even though the typical lesion had the appearance of a crater with a lip around it. Histological evidence, still incomplete, suggests that receptors are destroyed to the farthest visible edge, even though optic nerve fibers are intact within the lip. Second, as is well known, the optic nerve fibers arising from regions distal to the fovea and disc sweep around the fovea in a circuitous path before entering the disc. In effect, a symmetrical lesion around the macula will be functionally asymmetrical if such fibers are assumed to be destroyed as they course through the macula. An estimate of this asymmetry was made by enlarging a drawing of the fiber pathways (*4, 5*) to the same scale as our composite projections, and superimposing them.

The reconstruction of the retinal lesions is shown in Fig. 3. The white spot in each lesion indicates the estimated destroyed fovea, and the arrow indicates the nearest point of intact retina. The shaded areas represent only the directly damaged regions and not more remote regions rendered dysfunctional by the destruction of fibers passing through the lesions. However, the nearest edge of the intact retina was estimated only after such fiber damage was taken into account by the superimposition method just described above. Under each set of eyes in the figure is the relative acuity. The values are plotted on Fig. 1 as open circles, with the vertical strokes indicating the maximum possible size of lesion, given only that the fovea lay *somewhere* on the arc described above. Total reconstruction was impossible for the left eye of one animal ("Squib"), and its result is not plotted. It should be mentioned that two of the animals had their field defects for the right eye measured in the perimeter, and the results conformed closely to those predicted by the reconstructions.

As is evident, the retinal lesions produce a greater acuity reduction than the striate cortex lesions, and therefore it cannot be the case that the effects of the latter can be accounted for purely in terms of a greater parafoveal acuity in the monkey than

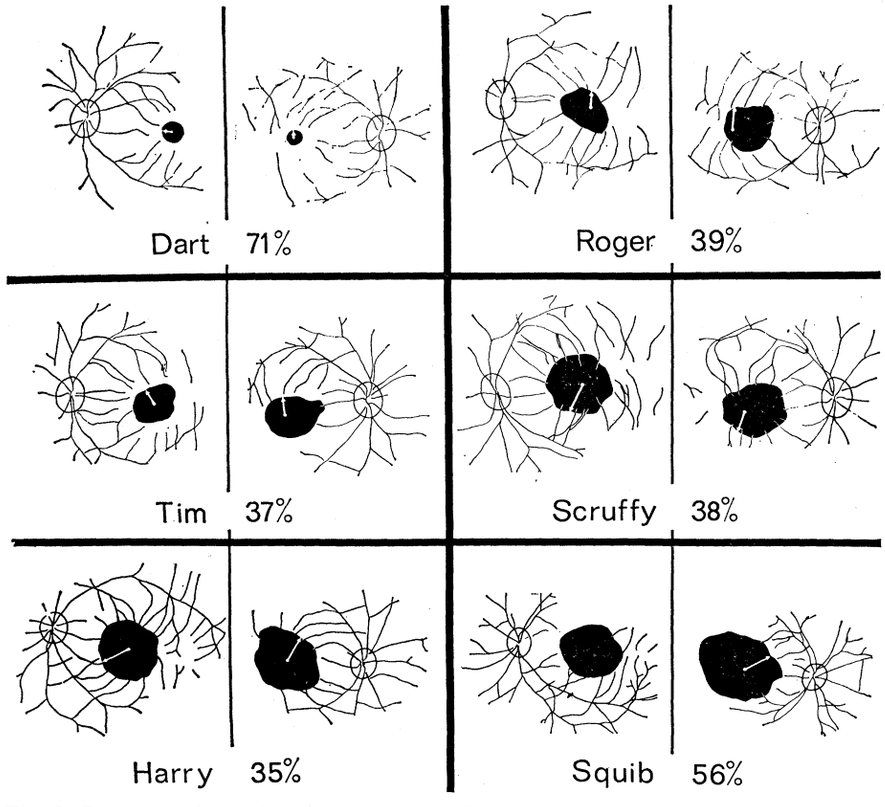

Fig. 3. Reconstructions of retinal lesions. White arrows connect the estimated position of the destroyed fovea with the nearest point of intact retina, according to method described in text. Percentages refer to relative acuity. Left eye is shown on left, right on right for each animal.

in man. The retinal lesions produce acuity reductions slightly smaller than that predicted from the human eccentricity curve, although it should be noted that the subsequent acuity of unoperated control animals shows a slight improvement over original preoperative values: relative acuity in these controls has a mean of 104 percent, as shown by the large circle displayed at 0° on the abscissa of Fig. 1. This would tend, in effect, to displace the entire human eccentricity curve slightly upward. Our results are to a degree consistent with that of Yarczower *et al.* (*6*), who report a drop in acuity after a "foveal" lesion in a single stump-tailed monkey. No reconstruction of the lesion, however, was provided by them, and the relative acuity (16 percent) was so low as to suggest either that their lesion functionally affected much more than the fovea or that the stump-tailed macaque is very different from the rhesus macaque. Or, possibly, the animals were incompletely trained, as suggested by their relatively high value of preoperative acuity (1.4 minutes).

It should be stressed that the discrepancy between the retinal and cortical results is conservatively based: at each doubtful juncture we made assumptions which increased the retinal lesions and decreased the cortical lesions. Therefore, if the present results are mistaken they are probably an underestimate of the discrepancy.

The failure of the electrophysiological map to predict the acuity results following striate cortical lesions has several possible explanations. It could be that the map displayed in Fig. 2 itself is wrong. The only other data for the monkey on which a map could be based are those of Daniel and Whitteridge (*7*). By integrating their curve relating "magnification factor" to degrees of eccentricity one can obtain an estimate of linear distance on the cortex for degrees of eccentricity in the field. When this is done the resulting cortical lesion would be expected to affect even larger regions of visual space, and the results (squares in Fig. 1) depart even further from the retinal results. But it is worth mentioning that other internal evidence provided by Daniel and Whitteridge indicates that their map should more closely approximate Talbot and Marshall's (*8*) map than in fact is achieved by using their magnification factor data, and they themselves strongly imply in their discussion an endorsement of these features of Talbot and Marshall's map which are relevant to the present point.

An attractive explanation of the results which would preserve the "point-to-point" concept would be the development of partial "denervation supersensitivity" at the edges of the cortical lesions, thereby making them able to respond to smaller differences between signals (*9*). But this explanation seems unlikely, since, in a further experiment, we found that the effects of combined retinal and striate lesions on acuity (involving the same regions of visual space) were equivalent to a retinal lesion alone. The supersensitivity argument should predict that the combined lesion be less deleterious than the retinal lesion alone. Projections from the retina to the midbrain or nonstriate cortex might also be able to carry the appropriate information so as to compensate for a striate cortical lesion. But, if so, the results clearly do not fit the hypothesis that such a projection has a fixed capacity so far as acuity is concerned, and one is inclined to examine simpler hypotheses. One hypothesis which neatly fits the facts stems from the definite knowledge that at various stages of the visual system, including the retina, ample opportunity exists for the interaction of neighboring regions, as manifested, for example, in "lateral inhibition." If information transmitted along such pathways of interaction (at a stage prior to the cell bodies of the lateral geniculate body) could be exploited by an animal with a cortical lesion, the obtained acuity results could easily be explained. The actual size of the difference between the retinal and striate cortical reduction results would give an indication of how far laterally information could have an influence. Another prediction which stems from this hypothesis is that the size of the field defect following a cortical lesion ought to be smaller than that predicted by the electrophysiological map. Exactly this result has been found by one of us (A.C.): the field defect measured perimetrically was found gradually to have shrunk over the course of the first few postoperative years, even though it originally was of a size corresponding closely to the one predicted by the map.

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14 September 1966

Evoked Pressure Responses in the Rabbit Eye

Abstract. *In the rabbit, a sensory stimulus of low intensity evokes a characteristic transient intraocular rise in pressure with an amplitude as great as 10 millimeters of mercury. This α-adrenergically mediated phenomenon occurs concomitantly with a general arousal response and appears to be caused by contraction of the orbital smooth muscle of Müller.*

Characteristic intraocular pressure responses as great as 10 mm-Hg in amplitude have been recorded in the eyes of conscious rabbits following low-intensity sensory stimulation. Electroencephalographic (EEG) changes indicate that this evoked rise in pressure occurs concurrently with a general arousal response. Thus, intraocular pressure parallels other physiological changes accompanying arousal in almost every system of the body (*1*). The latency, rate of rise, and rate of decay of evoked intraocular pressure transients are practically independent of stimulus parameters. However, habituation to a constant, periodically repeated sensory stimulus can often be observed, as with any stimulus which loses its novelty. Smaller replicas of the characteristic pressure wave sometimes appear spontaneously. These "internally" triggered events are accompanied by a K complex in the EEG. The evoked intraocular response was observed in each of the 32 New Zealand albino rabbit eyes that were tested.

In these unanesthetized and be-