pattern falling on the retina is altered by passive displacement of the eye or by scanning eye movements, lambda waves are produced. In the latter circumstance, there is no blurring of the visual image; but in the former, blurring does occur. Hence, lambda waves are unlikely to be concerned directly with the mechanism for suppression of blurring during eye movement.

This simplified method of stimulation of the visual system is of interest because it obviates change in total luminous flux as well as the complication of voluntary movement of the eyes. Such voluntary movement may lead to difficulties, as is indicated by some other recent studies in which we have observed that changes in the potential are already occurring in the cortex at the time of or just before eye movement. These may be similar to the readiness potentials (*3*) which occur in association with voluntary movements of the limbs.

After considerable investigation, we believe that the lambda response is more closely related to the visually evoked potential than to inhibitory mechanisms in effect during scanning eye movements.

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7 October 1966

Recovery of Memory after Amnesia Induced by Electroconvulsive Shock

Abstract. *Electroconvulsive shock given to rats immediately after one-trial avoidance learning produced a significant amnesic effect 24 hours later; this amnesia had largely disappeared in further retention tests 48 and 72 hours after treatment. This result puts in question a basic assumption implicit in most memory consolidation studies that such amnesic effects will be permanent.*

Animals given an electroconvulsive shock (ECS) shortly after a single avoidance learning trial will show little or no evidence of learning when tested 24 hours later (*1*). The usual interpretation of this effect is that the ECS has disrupted certain neural processes essential for the establishment of a memory trace. Such an interpretation is based on the assumption that any amnesic effects of this sort will be permanent: for, if ECS has effectively prevented the formation of a memory trace by occurring within the critical period required for this process, then the retention deficit observed 24 hours after treatment should be equally apparent at any other time of testing. Apart from an early experiment by Worchel and Narciso (*2*) and a more recent one by Chevalier (*3*), this assumption has never been systematically examined, yet it is of critical importance for memory consolidation theory.

Worchel and Narciso tested rats 4 days after a series of six massed ECS treatments were administered immediately after a criterion learning trial. The rats showed no impairment in relearning a 14-unit T-maze, although significant impairment had been noted when the rats were tested 24 hours after treatment (*4*). While these results were attributed by the authors to the temporary retroactive effects of the treatment, they could equally well have been due to the temporary proactive effects of the massive dose of ECS used in this experiment, or to a combination of both effects. Chevalier was unable to find any differences in the extent of amnesia for a nonspecific avoidance response (reduction of locomotor activity in an apparatus where shock had been given) in different groups of mice tested 1, 7, or 30 days after a single footshock-ECS treatment; he concluded that the amnesia was permanent. However, the results of the following experiment indicate that under certain conditions there can be a dramatic recovery from the retroactive effects of ECS. In this experiment ECS was administered after footshock consequent upon a step-down response, the interval between footshock and ECS was three-fifths of a second, and all animals were tested for retention 24, 48, and 72 hours after treatment.

Subjects in the experiment were 124 male albino rats of the Wistar strain, aged 90 to 100 days. Each cage housed 4 or 5 rats with free access to food and water. The apparatus was similar to that first described by Jarvik and Essman (*5*). It consisted of an uncovered 44 cm square compartment with walls 46 cm high, made of aluminum lined with matt black plastic. The floor was constructed of stainless steel rods (0.24 cm in diameter) set 1.27 cm apart. In the center was a 9-cm square platform raised 7.5 cm above the grid floor and illuminated from above by a collimated light source. Footshock could be delivered through the grid floor for 2.0 seconds by means of a scrambler delivering 10 pulses/sec to each rod at approximately 0.4 ma. The ECS (50 cycles a-c at 35 ma for 0.20 second) was administered by way of modified crocodile-clip ear electrodes from a constant current machine using the principle of the Pittsburgh electro-shock apparatus (*6*).

In each daily trial earclips were attached to each rat; it was then placed on the platform and its step-down latency (that is, the time spent on the platform) was recorded to within .01 second. On the first 3 days preliminary training trials were given in which the rats were permitted to explore the apparatus for approximately 10 seconds after stepping down. On the 4th day differential treatment was given as follows: Group 1 (FS, $n = 34$) received 2.0-seconds footshock through the grid floor immediately on stepping down from the platform; Group 2 (FS-ECS, $n = 40$) received immediate footshock, followed 0.6 second later by ECS; Group 3 (ECS, $n = 24$) received ECS only, 2.6 seconds after stepping down; Group 4 (NT, $n = 26$) received no treatment after stepping down. All ECS animals were removed from the apparatus while still unconscious and they recovered in their home cages; the other animals were returned to their home cages after approximately 10 seconds on the grid. On each of the three subsequent days retention trials were given with the same procedure as had been used in the preliminary training trials.

The usual criterion of retention in a step-down experiment is the length of time animals remain on the platform the day after footshock treatment (here termed step-down latency). In our experiment there was considerable variation in the post-treatment step-down latencies of the FS group,

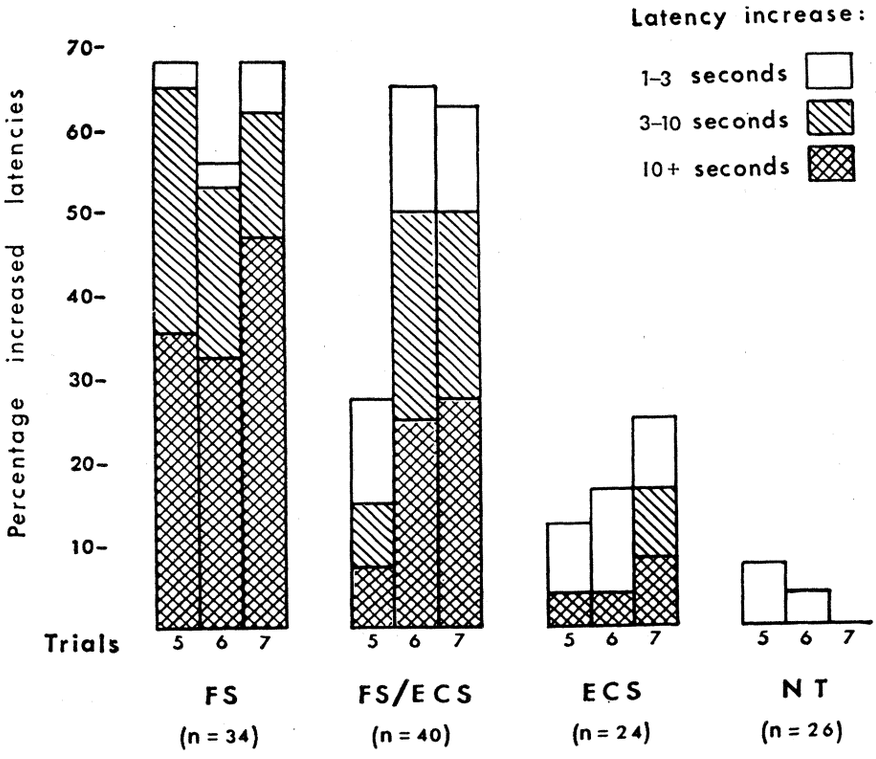

Fig. 1. Percentage of animals showing retention in three successive tests. Retention is indicated as individual increases of more than 1, 3, or 10 seconds over longest pretreatment latencies. FS, footshock; ECS, electroconvulsive shock; NT, no treatment.

relatively few of these animals having the very long latencies normally reported in experiments of this sort. This may be a function of interstrain differences, and also of the time of day used for testing—in this experiment all testing was carried out at night. Because latencies for all rats in the trials before treatment were consistently short (median 1.02 seconds for trials 2 through 4), we considered that even a moderate increase over previous latencies would indicate some degree of retention. Accordingly retention was assessed in this experiment by comparing each rat's step-down latency in trials 5, 6, and 7 with its own longest latency in trials 2 through 4 (*7*). Increased latencies were classified according to whether they exceeded 1, 3, or 10 seconds. The percentage of rats in each treatment group meeting each of these criteria in the three retention trials is shown in Fig. 1 (*8*).

The effect of principal interest emerging from these results is that the retention deficit clearly apparent in the FS-ECS group on the 1st day after treatment has largely disappeared in the two subsequent trials. In the first retention test (trial 5), 68 percent of the FS group met the 1-second criterion, 65 percent met the 3-second criterion, and 36 percent met the 10-second criterion, as compared with only 27.5 percent, 15 percent, and 7.5 percent, respectively, for the FS-ECS group (all χ^2 significant beyond the .01 level). This result is comparable with those obtained in similar experiments when retention is tested 24 hours after treatment. There is, however, very little evidence of impaired retention in the FS-ECS group in the next two trials. By the 3rd day of retention testing (trial 7), the proportions of these animals reaching the 1-, 3- and 10-second criteria have risen to 62.5 percent, 50 percent, and 27.5 percent, respectively. It is worth noting that of the 72.5 percent of FS-ECS animals failing to meet even the 1-second criterion on trial 5, as many as 28 percent have reached the 10-second, 58.5 percent the 3-second, and 69 percent the 1-second criteria on either or both of the subsequent trials.

The FS-ECS group as a whole shows a significant increase in step-down latency from trial 5 to trial 7, the mean latency rising from 3.42 seconds on trial 5 to 9.18 seconds on trial 7 ($t = 2.89$, $P < .01$). In contrast, the comparable increase (from 10.7 to 12.5 seconds) for the FS group is not significant ($P > .25$). As can be seen from these mean latencies, the FS-ECS animals were showing approximately the same degree of avoidance on the final day of testing as that of the FS group on the day after treatment ($P > .25$).

All ECS animals included in these results had full tonic extensor fits. The interval between footshock and ECS (0.6 second) used in this experiment was shorter than in most of the comparable studies, and the ECS intensity (35 ma) and duration (0.2 second) were similar. It would seem, therefore, that the amnesic effect regularly found by other investigators when testing 24 hours after treatment may not reflect a permanent absence of the memory trace.

It is possible that the recovery found in our study is due to a "shrinkage" of amnesia as described for retrograde amnesia following concussion (*9*); such a shrinkage would be predicted from the model proposed by Weiskrantz (*10*). An alternative possibility is that the recovery depends on reexposure to the testing situation and that learning is taking place. Such learning would depend on there being some minimum retention of the traumatic properties of the situation on the first day of testing.

The results for the group receiving ECS only were significant. Previous studies (*11*) have suggested that while a single ECS is not aversive, repeated treatments may become so; the aversive effect has been found on the 3rd or 4th day of repeated treatments. Since we have found significantly more evidence of retention on the 3rd day of testing than on the 1st ($t = 2.34$, $P < .05$), after a single ECS, it is possible that the results of the earlier studies may be due not only to the repetition of ECS but to a latent and strengthening trace set up by the first ECS in the series.

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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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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

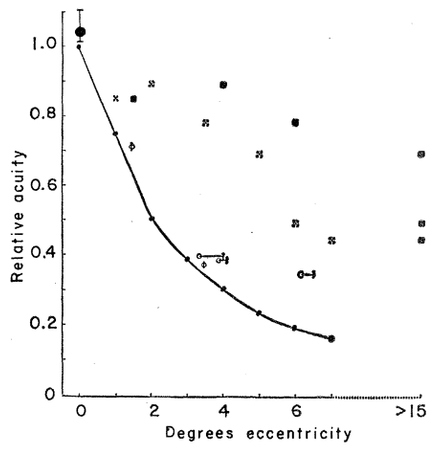

Fig. 1. Relative acuity as a function of distance of test stimulus from fovea. Solid curve: function for normal human observers with identical test situation as used with monkeys. Open circles: relative acuity following retinal lesions of given radius, measured from destroyed fovea as center. Vertical 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-