to reduce fluctuations in temperature and relative humidity. A few bits of commercial cetyl alcohol sprinkled by hand on the surface of the water provided the resisting film. Imperfectness of the insulation seems to have been the limiting factor on the accuracy and range of the measurements. Reproducibility of the film was also somewhat haphazard.

At a high wind velocity of about 18 mi/hr the acaloric steady-state temperature was raised by 4.4°C as a result of the film, while at a lower wind velocity of about 6 mi/hr, the effect was 3.8°C. The corresponding resistances were $r_a = 4.2$ and 7.5×10^5 °C sec cm²/g; $R_f = 20$ and 22×10^5 mm-Hg sec cm²/g, or, in centimeter-gram-second units, $R_f = 2.1$ sec/cm within experimental error. When the water was heated, the experimental uncertainty in W_a was too large to make it useful in determining the surface temperature through Eq. 3. Equations 1 and 4 were therefore used to estimate the thermal resistance of water (r_w). The values found did not seem to depend appreciably on wind velocity but increased markedly in the presence of film, especially when heat input was small and bulk temperature was close to surface temperature. They ranged from $0.4 \pm 0.15 \times 10^5$ °C sec cm²/g for a clean surface to 1.4×10^5 in the presence of a film when $T_w - T_s \cong 3$ °C and 2.6×10^5 when $T_w - T_s \cong 0.8$ °C. These effects suggest that convection currents are the main factor determining r_w.

Rather surprisingly, the film had no perceptible effect upon the thermal resistance of air (r_a). Thus, the quieting effect of a monolayer (*3*)—the calming of troubled waters—which is so prominent in field tests of evaporation control (*4*) seems not to affect the rate of evaporation under the conditions of these small-scale experiments (*5*).

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Effect of Interruption of the Visual Pathway on the Response to Geniculate Stimulation

Abstract. Optic nerve section or destruction of the lateral geniculate nucleus increased the amplitude and elevated the recovery cycle of the cortical response to lateral geniculate radiation stimulation in cats. The lesions may have acted by eliminating tonic inhibitory or occlusive volleys originating in the retina, or both.

Since publication of the initial descriptions of the cortical response to electrical stimulation of the geniculo-striate pathway (*1*) there have been a number of studies of the anatomical substrate (*2*) and recovery cycle (*3, 4*) of this response. Recent studies of this recovery cycle (carried out in unanesthetized cats with chronically implanted electrodes) showed that a stimulus to the lateral geniculate radiations was followed by subnormality lasting 1 second or more (*5*). In recent studies of the factors underlying this prolonged subnormality, it was found that optic nerve section or destruction of the lateral geniculate nucleus markedly altered the recovery cycle of the cortical response to lateral geniculate radiation stimulation. The present report describes these observations.

Adult cats were anesthetized with pentobarbital, ether, or urethane and placed in a stereotaxic instrument. Stimulating electrodes in the lateral geniculate radiations delivered a pair of shocks every 5 seconds. The first (conditioning) stimulus of the pair preceded the second (test) stimulus by 3.2 to 1600 msec. The evoked responses were recorded from the surface of the lateral gyrus with a pore electrode. Lesions of the lateral geniculate were produced electrolytically. The optic nerve was interrupted by freezing or by clamping.

Interruption of both optic nerves or destruction of the ipsilateral lateral geniculate caused a decrease in variability and an increase in amplitude of the postsynaptic components of the cortical response to geniculate radiation shock. Such lesions also caused a marked decrease in the degree of subnormality of the surface positive components, though not of the surface negative component, of the test response (Fig. 1). Figure 2 presents a graph of a recovery cycle before and after optic nerve section. These effects could be demonstrated in cats anesthetized with each of the three anesthetics employed.

Several experimental variables modified the degree to which recovery was enhanced following interruption of the visual pathway. One of these was stimulus intensity, recovery being enhanced more for the responses to supramaximal than for those to near-threshold stimuli. A second variable was the relative position of the stimulating and recording electrodes. The responses in cortical positions outside the maximal cortical focus of the stimulating electrodes showed marked increase in amplitude, but the enhancement of the recovery cycle was relatively slight. On the other hand, at the maximal focus, responses showed less increase in amplitude but more enhancement of the recovery cycle.

Several hypotheses may be offered as to the mechanism by which interruption of the visual pathway exerts these effects. The lesions might act by eliminating the

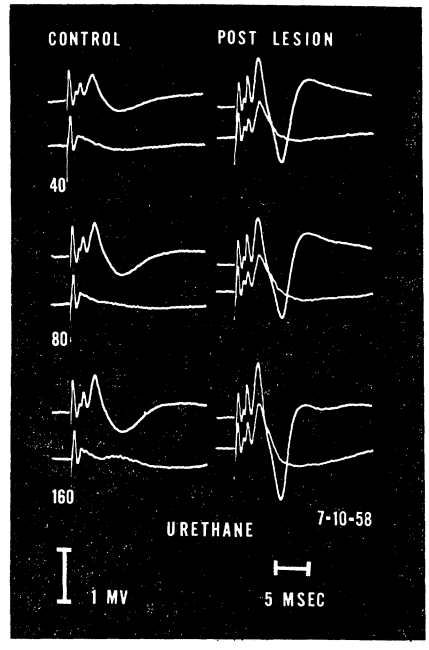

Fig. 1. Cortical responses to paired geniculate radiation shocks before (left) and after (right) optic nerve section under urethane anesthesia. Separations between the shocks of each pair are indicated at the left of each row. Following the lesion there is an increase in amplitude of all components of the response. Positive is up.

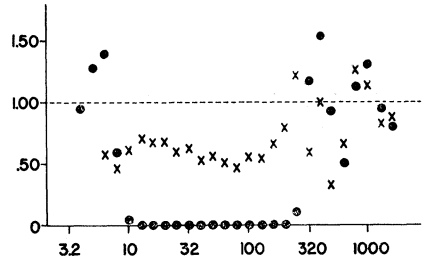

Fig. 2. The recovery cycle of the major positive component (C_4) of the cortical response to geniculate radiation stimulation before (dots) and after (crosses) optic nerve section. On the abscissa is plotted separation (in milliseconds) between paired shocks. On the ordinate is plotted the ratio of the amplitude of C_4 (the major positive component of the test response) to C_4 of the control. Following the lesion, depression of the test response between 10 and 250 msec was much less marked than it had been before the lesion.

background of spontaneous afferent impulses reaching the cortex from the retina. This background activity may be inhibitory or excitatory, or both, and the changes in the cortical response might thus result from elimination of tonic inhibitory or occlusive influences, or both. It is also possible that the lesions alter cortical excitability secondarily, as a result of an alteration of excitability of the central structures (*6*) to which the slower-conducting optic nerve fibers project. These structures may, in part, mediate the prolonged effects of the conditioning volley on the test response.

Regardless of what mechanism is ultimately shown to operate, the present experiments indicate the large extent to which the recovery processes of the primary visual cortex depend upon impulses reaching the central nervous system from the peripheral visual apparatus.

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Utilization of Porphobilinogen Carbon-14 in Biosynthesis of Vitamin B_{12}

The structural formula of vitamin B_{12} proposed by Hodgkin (*1*) and by Bonnet (*2*) and their colleagues focused attention on the probable presence of a cobalt-binding porphyrin-like moiety in the molecule. Despite its unique character, the tetrapyrryl ring structure indicated the likelihood that common pyrrol precursors for this vitamin and for the various naturally occurring porphyrins were present. In the George R. Minot lecture (*3*) in June 1956, mention was made of our observation that porphobilinogen, the monopyrrolic precursor of uro-, copro-, and protoporphyrin, is readily utilized in the formation of vitamin B_{12} by a bacterial culture. At about the same time Shemin *et al.* (*4*) reported the incorporation of δ-amino-levulinic acid into vitamin B_{12} under similar conditions.

The present report describes the conditions and results of the porphobilinogen C^{14}-vitamin B_{12} studies (*5*).

Carbon-14 labeled porphobilinogen was prepared as follows: Two rabbits were treated with allyl-isopropyl-acetylcarbamide (Sedormid) for 10 days. Each rabbit then received a total of 160 μc of glycine-2-C^{14} subcutaneously in five divided doses over a 36-hour period. The urine was collected during this period and for the next 6 days. The pooled 975 ml of urine contained 310 mg of porphobilinogen as determined by quantitative analysis of the Ehrlich aldehyde reaction. The pooled urine was subjected to the method of Cookson and Rimington (*6*) for the isolation of porphobilinogen; 102 mg of crystalline porphobilinogen were obtained.

Some crystalline material was dissolved in H_2O and evaporated on a planchet for radioactivity measurement. A specific activity of 6300 count/min per milligram was observed. Solubility properties and the intensity of the Ehrlich aldehyde compound were similar to those found with repeatedly recrystallized porphobilinogen isolated from urine of patients with acute intermittent porphyria.

Ninety milligrams of the crystalline compound were placed in an evacuated and sealed ampule and sent to the Merck Laboratory for further study. Here the crystalline porphobilinogen was dissolved in 0.1*N* sodium hydroxide and sterilized by filtration before being added to the sterile nutrient medium (*4*). It was incubated with the bacterial culture in three separate experiments. In the first study, 12.6 mg (56 μmole) of porphobilinogen was added at time 0; in each of the other studies, a total of 22 mg (97 μmole) was added to three divided portions, at time 0 and again after 1 and 2 days' incubation, respectively. The broths were all harvested after 4 days. The vitamin B_{12} content of the broth was assayed microbiologically. Carrier B_{12} was then added to facilitate isolation. Radioactivity measurements were performed with a windowless gas-flow counter, with suitable correction made for self-absorption of the samples evaporated on a steel planchet.

Table 1. Recovery of vitamin B_{12}-C^{14} from a bacterial culture incubated with porphobilinogen-C^{14}. Background count, 22.0 ± 0.6 count/min for experiment No. 1 and 26.4 ± 0.7 and 27.6 ± 0.7 count/min, respectively, for experiments No. 2 and 3 (60 min each). A correction factor was applied to correct for self-absorption of NaOH used as solvent and for differences in background counts.

	Expt. 1	Expt. 2	Expt. 3
Porphobilinogen added (mg)	12.6	22.5	22.2
Vitamin B_{12} produced (mg)	0.15	1.29	1.47
Carrier added (mg)	10.1	10.2	8.83
Carrier + B_{12} recovered (mg)	6.91	5.41	2.98
Amt. in planchet (mg)	0.33	0.216	0.119
Duration of count (min)	120.	300.	300.
Count/min above background	23.1 ± 0.6	20.4 ± 0.4	7.6 ± 0.4
Count/min corrected	38.4	28.9	10.2
Count/min mg of B_{12} produced	8000.	1200.	600.
Count/min μmole of B_{12} produced	10800.	1630.	810.
Count/min μmole of B_{12} per C^{14} carbon atom	1540.	233.	116.

The observed radioactivity of the porphobilinogen at the Merck Laboratories was 6900 count/min per milligram or 1560 count/min per micromole. This corresponds to an activity of 780 count/min per micromole for each of the two labeled carbon atoms in the molecule (*7*).

As is shown in Table 1, the calculated activities of the vitamin B_{12} produced ranged from 810 to 10,800 count/min per micromole of vitamin B_{12} produced. If one assumes labeling of seven carbon atoms in the vitamin corresponding to the two labeled carbon atoms in porphobilinogen (*7*) (one alpha carbon atom in each of the four pyrrole rings and three bridge carbon atoms), it would appear that approximately one-third and one-half of the porphyrin-like moieties were derived from the added porphobilinogen-C^{14} in experiments 2 and 3, respectively. In the first experiment, however, the radioactivity recovered is greater than can be accounted for on the basis of the observed production of only 0.15 mg of vitamin B_{12}. It is evident that the many possible additive errors inherent in such a calculation do not permit precise estimation of the dilution factor involved in the incorporation of the added porphobilinogen C^{14} into vitamin B_{12}.

The present result, coupled with that of Shemin and his coworkers with δ-aminolevulinic acid, clearly reveals that the biosynthesis of the porphyrin-like moiety of vitamin B_{12} is along the same primary pathway as that of other naturally occurring porphyrins. The