end of the transected pyramidal tract, effectively activating all recurrent collaterals of these neurons, had no measurable effect on the cortical neurons tested. However, intentional involvement of the adjacent medial lemniscus fibers profoundly altered the responsiveness of all neurons tested. During such conditioning input, no responses could be obtained from the cortical neurons by adequate stimulation of their excitatory receptive fields during stimulation, but complete responsiveness returned within 1 second upon cessation of stimulation. Such involvement was obtained either by sufficiently increasing the strength of stimulation at the ventral surface of the brain stem (pyramidal bundle) to produce orthodromic events in the cerebral cortex (*6*) or by penetrating the pyramidal bundle and inserting the electrodes into the medial lemniscus. However, during weak stimulation confined to the pyramidal tract the receptive fields of coronal neurons were enhanced. It is thus evident that the excitatory effects, and perhaps the inhibitory effects, result from pyramidal tract facilitation of a particular set of neurons in the cuneate nucleus.

Evidently the pyramidal tract—a uniquely mammalian possession that connects the cerebral cortex directly with so many brain stem and spinal neurons, both sensory and motor—constitutes one route by which the cerebral cortex can modify its own afferent input. Suggestive as the findings are, the role that this system of fibers plays in perception and attention remains to be demonstrated.

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7. Supported by PHS grants B 396, NB 5136, and GM 666.

17 March 1966

Hormones as Allosteric Effectors

In his article, "Plant hormones and regulators" (*1*), van Overbeek makes the cautionary point that certain responses to both plant and animal hormones occur much too rapidly to be mediated through an action at the gene level. He suggests that there may be "several sites of primary hormone action, just as there are several doors that can be opened by one key." However, it is important to emphasize that different sites of action (such as at the DNA template and at the cell membrane) need not imply fundamentally different methods of action. To carry van Overbeek's analogy further, doors that can be opened by one key presumably have identical or similar locks. The concept of hormones as allosteric effectors, propounded by Monod, Changeaux, and Jacob (*2*), provides a plausible common denominator among apparently divergent locking mechanisms. As these authors have stated, "it seems difficult to imagine any biochemical mechanism other than allosteric which could allow a single chemical signal to be understood and interpreted simultaneously in different ways by entirely different systems." Thus rapid manifestations of hormone action could result from direct allosteric modification of extranuclear enzymic or structural proteins. Such an action would not differ in essence from hormonal control of enzyme biosynthesis through allosteric interaction with repressor proteins on operator genes. A similar view, as specifically applied to auxins, has recently been expressed by Südi (*3*) in the suggestion that "similar allosteric sites of a great number of functionally different proteins make up auxin receptor sites."

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1 June 1966

Signal versus Noise in the Evoked Potential

Various methods have been developed over the last two decades which allow the detection of cortical evoked potentials with scalp electrodes. The most widely used method summates electrical activity occurring over the same time interval following a repetitive stimulus, and a number of specialized computers which do this are commercially available. One assumption in this method is that potential changes evoked by each stimulus presentation (the signal) will be time-locked and will summate with repetition. A second and corollary assumption is that remaining potential changes (the noise) will be random and cancel out with enough repetitions.

The second assumption is met with practical sample sizes only within a certain variance (or standard deviation) of error, and therefore the signal must to some extent be composed of noise. Where it is assumed that the signal and noise are independent, and that the noise remains the same under conditions of stimulus or no stimulus, the amount of noise in the signal is related to the obtained ratio of signal to noise; thus, 2 : 1 ratio would mean that approximately half the signal was noise. Larger ratios would produce corresponding decreases in the amount of noise in the signal. Therefore, it seems incumbent upon investigators to present data regarding the degree to which the signal exceeds the noise or, at least, to acknowledge that this has been examined. In fact, failure to use noise, or control, data (summation over the same temporal interval but with the light or other stimulus occluded) makes it difficult to determine whether a cortical event related to the stimulus did indeed occur. The ease with which the simple presence or absence of a signal may be determined, even with low ratios of signal to noise, probably accounts in part for the omission of noise data in some reports [*Science* **150**, 1162 (1966); **148**, 980 (1965); **145**, 180, 182 (1964); **141**, 1285 (1963)]. Failure to present noise data is even more serious when attempts are made to interpret variations in small components of the signal. For example, conclusions regarding differences between earlier and later components of potentials in aged subjects [*Science* **151**, 1013 (1966)] must be considered tentative until it is demonstrated that such differences cannot be attributed to variations in the noise. Thus disregard of noise in summation techniques weakens an otherwise impressive research tool.

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26 May 1966