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Subcortical Centers as Pacemakers of Cortical Activity.*

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In continuation of a previous investigation[1] in which the effect of anoxia and asphyxia on normal and convulsive potentials of the cortex was studied, experiments were undertaken to determine the hypothalamic-cortical relations under these conditions. All experiments were performed on curarized "Dial" cats (intocostrin, artificial respiration) in which after exposure of the brain cortical, hypothalamic, and thalamic potentials were recorded with mono- and bipolar silver electrodes. An Offner inkwriter and amplifier were used. Subcortical electrodes were inserted with the Horsley-Clarke apparatus. Strychnine pledgets served to elicit local cortical spikes; this drug was also injected either into the hypothalamus or intravenously.

In experiments in which the cortex was strychninized the results were similar to those discussed in the preceding paper. However, if strychnine was injected intravenously a series of very regular small spikes appeared synchronously in cortex and hypothalamus at a time when due to the asphyxia normal and convulsive potentials had either completely disappeared or were greatly reduced in amplitude and frequency. This burst of activity lasted in the majority of observations for 16 to 25 seconds although in some instances it was as brief as 6 or as long as 34 seconds. The frequency of discharge was variable. In general the amplitude declined greatly as the frequency increased, the maximum being about 14 to 18 per second.

Since the synchrony of discharge suggested a subcortical pacemaker strychnine was injected into the hypothalamus and in other instances into several nuclei of the thalamus. Although heavy strychninization of the hypothalamus may lead to appearance of spikes in the cortex[2] injection of this drug into a minute area of the hypothalamus induced local spiking but did not interfere with normal cortical activity and the asynchrony existing between different cortical areas. Asphyxia caused under these conditions likewise the appearance of synchronous spikes in various cortical areas and in the hypothalamus (Fig. 1). These potentials were larger in monopolar than in bipolar recordings. Cathode ray oscillograms showed that they consisted of bipolar spikes. This phenomenon was reversible and could be repeated many times in the same preparation. As time progressed the spikes appearing in the injected area became less in frequency and amplitude and then disappeared. Even at this stage the synchronous discharge could be induced in asphyxia. When some time later synchrony was no longer elicited it could be reinvoked after reinjection of strychnine into the hypothalamus.

The synchronous discharges discussed in this paper were recorded bilaterally in the cortex after unilateral injection of the hypothalamus or thalamus with strychnine. They were likewise found in contralateral thalamic nuclei. This phenomenon occurred in asphyxia as well as in anoxia (inhalation of nitrogen or nitrogen-air mixtures). It is probably related to the synchronous spindles which van Harreveld[3] observed in the cortex after prolonged asphyxia. It is suggested that:

1. Hypothalamic and closely related thala-

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[1] Gellhorn, E., and Heymans, C., *J. Neurophysiol.*, 1948, **11**, 261.

[2] Murphy, J. P., and Gellhorn, E., *J. Neurophysiol.*, 1945, **8**, 341, 431.

[3] van Harreveld, A., *J. Neurophysiol.*, 1947, **5**, 361.

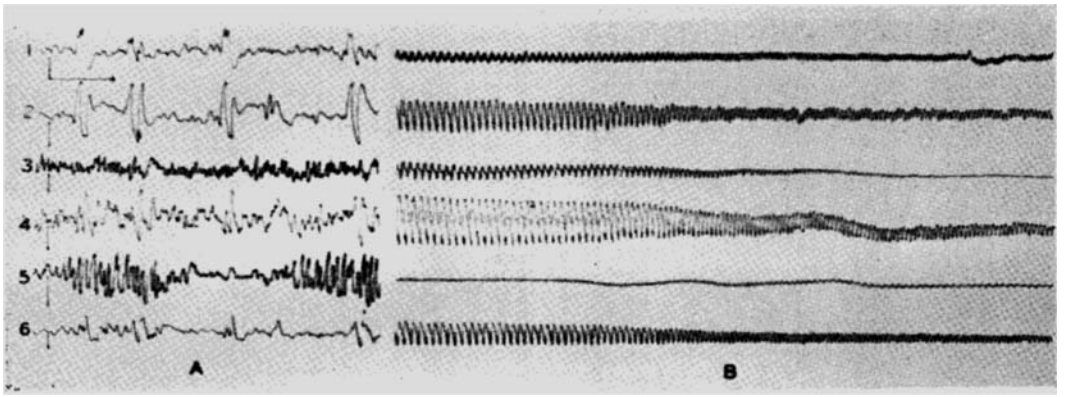

FIG. 1.

Dial cat. Right hypothalamus injected with 0.03 mg strychnine. A, control; B, during asphyxia, immediately after normal and convulsive potentials had been abolished. Note asynchrony in control and synchronous discharge in asphyxia.

1. Left ventrolateral thalamus (monopolar).
2. Right hypothalamus (monopolar) injected with strychnine.
3. Left motor cortex (bipolar).
4. Same site, monopolar.
5. Left auditory cortex (bipolar).
6. Same site, monopolar.

Calibration 100 microvolts and 1 second.

mic nuclei[2,4] do not act as pacemakers of cortical activity as long as the excitability of the cortex is unchanged.

2. In conditions such as asphyxia and anoxia which lead to a relative predominance of hypothalamic excitability the discharge of these subcortical nuclei determines the qualitative and quantitative activity in thalamus and cortex and leads to periods of complete synchrony of hypothalamic, thalamic and cortical activity.

[4] Gellhorn, E., and Ballin, H. M., *Am. J. Physiol.*, 1946, **146**, 630.

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Relation of Iodinated β-amylose (Tridine) to Some Thyroid Functions.

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The appearance of iodinated β-amylose, "Tridine",† a compound of carbohydrate and iodine, suggested a biological assay to determine whether it behaves like an inorganic salt of iodine or like other iodinated organic compounds such as thyroxin or thyroglobulin.

Methods. White albino rats were used throughout the experiment. All animals received ground dog biscuit‡ and water *ad libitum* for 5 to 7 days before any experiment was begun. During the experimental period the control group remained on this diet while

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† According to the manufacturer, The Clairmint Chemical Co., 7 Lackawanna Avenue, Newark 2, N. J. "Tridine" is iodinated β-amylose and contains approximately 20% of iodine by weight. The Clairmint Chemical Co., defrayed part of the expense of this investigation.

‡ Purina Laboratory Chow.