fiber with radiosodium. The result of such an experiment, recorded in Figure 2, shows a 59.5% rise in the Na efflux. This value turned out to be almost thrice that obtained with a control fibre involving a single injection of TN-C. In the second experiment, however, the Na efflux showed a 29.6% rise, a value which was not very different from the control value of 20.5%. Figure 2, in addition, provides evidence indicating that double injection of TN-C does not interfere with the sensitivity of the fibre to external acidification. It is, however, worth remembering that since the affinity constant of TN-C for Ca^{2+} is pH-dependent[8], it is not unlikely that some Ca^{2+} bound to the protein had been liberated into the myoplasm following injection. In other experiments 5×10^{-4} TN-C was injected only once, followed by lowering of the external pH from 7.8 to 6.3. The results obtained in 4 experiments showed a $44.2 \pm 13\%$ rise in the Na efflux, and that all fibres were markedly sensitive to external acidification.

The next question coming to mind was whether the effect caused by TN-C involved the ouabain-sensitive or the ouabain-insensitive Na efflux. As shown in Figure 3, external application of 5×10^{-5} *M* ouabain caused a large fall in the Na efflux, while internal application of 5×10^{-4} *M* TN-C caused a small rise in the remaining efflux. The magnitude of the stimulation averaged $23.3 \pm 1.8\%$ ($n = 5$). This result was not wholly in accordance with expectation. This is because the effect of TN-C on the Na efflux in unpoisoned fibres was thought to be due to reduced suppression of the Na^+-K^+ ATPase as the result of reduced $[Ca\ ATP]^{-2}$ formation.

It seems, then, as if TN-C has the ability to stimulate the Na efflux in barnacle fibres. Although the mechanism of this stimulation remains far from clear, there are reasons for supposing that it resembles the mechanism underlying the stimulation obtained by injecting low concentrations of EGTA (BITTAR and SCHULTZ, unpublished data). Since TN-C has a high affinity only for Ca^{2+}, it is tempting to speculate that both TN-C and EGTA, when used in low concentrations, stimulate the Na efflux as the result of removing Ca^{2+} from the myoplasm. Whether EGTA, when applied in high concentrations, inhibits the Na efflux by removing Mg^{2+} from the vicinity of the transport sites, is a question which forms the subject of experiments already in progress.

Zusammenfassung. Nachweis, dass eine Microinjektion von Troponin-C den Na-Ionen-Ausfluss aus Einzelfasern des Entenmuschel-Muskels steigert.

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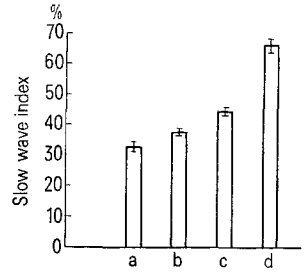
Electroencephalographic Studies in Toad (*Bufo melanostictus*) Following Prolonged Exposure to Heat During Hibernation and Non-Hibernation

Electroencephalographic (EEG) studies have been done exhaustively in hibernating mammals but the studies of the same in hibernating poikilothermic animals is inadequate. EEG studies during hibernation and arousal have been made by CHATFIELD et al.[1] and CHATFIELD and LYMAN[2]. DE, BORAL, DEY and DEB[3] found marked variation in the electrical activities of brain in hibernating toads over the non-hibernating ones. The principal feature observed during hibernation was slowing of the brain potential, which, however, could be replaced by low voltage fast activity (LVFA) following sensory activation.

It has been observed (unpublished) that hibernating toads can withstand an exposure of 56°–58 °C for 8 days, while the non-hibernating animals can tolerate up to 48 °C only for 8 days. This fact aroused the curiosity of the authors to know the electroencephalographic response to this variance in heat tolerance according to season.



Slow wave indices of brain in toads: a, non-hibernation exposed group; b, non-hibernation control group; c, hibernation control group; d, hibernation exposed group.

Materials. Male toads (*Bufo melanostictus*) weighing 45–65 g were taken during hibernation (May to August). These toads were placed in an incubator set at 56 °–58 °C for 8 days with the supply of water being maintained. The rectal temperature of the hibernating toads was 19 °–21 °C, which after the exposure rose to 30 °–33 °C. The rectal temperature of the non-hibernating toads was 29 °–31 °C. After an exposure to 48 °C for a period of 8 days, the rectal temperature was found to vary from 33 °–35 °C.

Methods. After 8 days exposure the animals were taken out of the incubator one by one and made spinal by inserting steel probe downwards through the vertebral column. Steel needle electrodes were fixed on the scalp at 4 points over the right and left cerebral hemispheres and connected to the machine. Electrographic recordings of the brain potential was taken by a Grass Model III-D, 8-channel Electroencephalograph with ink writing pens. The paper was run at a speed of 30 mm/sec.

Brain waves with a frequency of 12 c/sec or less have been designated as slow waves and those ranging above 12 c/sec are designated as fast waves. The slow wave index has been calculated according to the method of

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Group	[a]LVSA		[b]LVFA		[c]HVSA	
	Frequency (c/sec)	Max. amp. (μv)	Frequency (c/sec)	Max. amp. (μv)	Frequency (c/sec)	Max. amp. (μv)
Non-hibernating control (10)	9.9 ± 0.41	29 ± 1.54	25.8 ± 0.41	12.5 ± 0.63	Absent	
Non-hibernating exposed (10)	12.7 ± 0.68 $P < 0.01$	40.4 ± 0.87 $P < 0.001$	31.2 ± 0.66 $P < 0.001$	12.5 ± 0.81	Absent	
Hibernating control (10)	9.8 ± 0.51	34.6 ± 0.99	25.8 ± 0.866	17.9 ± 0.75	8.9 ± 0.11	84 ± 3.48
Hibernating exposed (10)	9.9 ± 0.69	47.2 ± 1.31 $P < 0.001$	24.8 ± 0.94	21.7 ± 0.82 $P < 0.01$	5.1 ± 0.525 $P < 0.001$	134.5 ± 5.65 $P < 0.001$

[a] LVSA, low voltage slow activity. [b] LVFA, low voltage fast activity. [c] HVSA, high voltage slow activity. Figures in parentheses indicate number of animals. The results are Means $\pm$ S.E.

DAVIS[4] as modified by PINEDA and ADKISSON[5]. The maximum amplitude was calculated according to the method of DEY et al[6].

Results. When compared with non-hibernation control group, the characteristic feature of EEG of hibernation control group consists of an increase in the sequence of occurrence of slower waves and in the slow wave index. Mild high voltage slow activity (HVSA) was also evident. The EEG of the non-hibernation exposed group revealed extreme low voltage fast activity (LVFA) with complete absence of HVSA (Table). The hibernation exposed group demonstrated EEG features, characterized by random to regular HVSA (4–7 c/sec) with about 58% increase in the amplitude and 50% increase in the slow wave index in comparison with the hibernation control group (Figure).

Discussion. ECCLES[7] ascribes the cause of synchronised theta activity in nonprimates to the inhibitory neurones acting as pace-makers by periodically inhibiting the ascending afferent pathways. According to GASTAUT and FISCHER-WILLIAMS[8], this inhibitory system is 'branched off' in a side chain from the thalamo-cortical projection, a system which may actively inhibit the reticular formation of the thalamus (rostral) as well as the caudal brain stem and prevent the discharge of cortical spikes resulting in exclusive HVSA. Thus reticular release may be equally responsible for putting into action the inhibitory system. Naturally the high voltage slow waves do not represent a convulsion wave but a veritable state of neuronal depression linked to a phenomenon of active inhibition. From the present study it appears that, in toads during non-hibernation, the tone of the ascending afferent pathways can be increased by sensory activation (e.g. heat exposure) as revealed by extreme altering response in EEG. Whereas, during hibernation, there is a possibility that some cortical and subcortical inhibitory mechanisms operate even during prolonged heat exposure, resulting in HVSA in EEG. Moreover, the heightened activity of these inhibitory neurones, according to ECCLES[7], might be responsible for the conditions of accommodation[9] so as to withstand such a high environmental temperature for a prolonged period.

Résumé. L'électroencephalogramme des grenouilles qui ont été exposées à 48 °C pendant 8 jours durant la non-hibernation révéla une extrême «low voltage fast activity» avec une absence complète de la «high voltage slow activity» (HVSA). L'hibernation (à 58 °C pendant 8 jours) montra un HVSA irrégulier à régulier avec 100% d'augmentation en longueur et quelques méchanismes corticaut et subcorticaux restent actifs même lors d'une prolongation de la chaleur.

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Depressed Synthesis of DNA in Regenerating Rat Liver after Spinal Cord (C_7) Transection

Spinal cord transection results in the increase of tryptophan oxygenase and tyrosine aminotransferase activities in rat liver by a process independent of adrenal secretion[1–3]. The enhancement was observed only after C_7 level section, i.e. above the segments which innervate the liver. In an effort to get more information on the possible mechanism of this phenomenon, we studied the effect of spinal cord transection on the synthesis of liver DNA.

Material and methods. Groups of 3–4 male albino rats (175 g) kept under standard conditions were used throughout the experiments. Spinal cord transection[1] and partial hepatectomy[4] were performed under light ether narcosis. In sham-operated animals the spinal cord was exposed only. Synthesis of DNA was measured after i.p. administration of thymidine-2-^{14}C (1.5 μCi/0.5 μM

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