An Insulin Inhibiting Agency in the Duodenum

HELLER[1] and Labarre[2] have reported that some of their preparations of the insuletropic hormone failed to demonstrate the usual increase in sugar tolerance in animals, and are unable to ascribe this to any definite cause. Others also report inconsistent results. Since 1929, using a method reported in conjunction with N. B. Laughton[3], I have noted that it is extremely critical, and deviation at one stage yields negative results.

The inertness of the preparations could not be ascribed solely to mere deficiency in the hormone content, since, while such preparations did not affect the blood sugar values of normal rabbits, these animals displayed a distinctly lowered sugar tolerance. Also such preparations were hyperglycæmic in their action on diabetic patients.

Striking effects were obtained where 'inactive' preparations were investigated on the insulinised rabbit. Controls were given 1 rabbit unit of insulin per 2 kgm. weight and the blood sugar values observed for 6–8 hours. These invariably gave the usual response—the sugar values dropping to 50 mgm. within 2 hours and remaining at this level for 3–4 hours longer. Afterwards, these same animals were treated with the defective preparations and then this standard amount of insulin administered. In some cases no fall in the blood sugar was observed, while in average cases the blood sugar values did not fall below 70 mgm. in 2 hours, and returned rapidly to 100 mgm., usually reaching 130–150 mgm. 4 hours after administration of insulin.

The failure to observe the customary effects of the insuletropic hormone was due to the preponderance of this insulin antagonistic element. After this had been separated, the preparations became insuletropically active, although extreme activity could be developed by further treatment.

The effect of the insulin inhibiting principle, like that of the insular hormone, persists for some weeks after its discontinuance, and a study of this factor in cases of hyperinsulinism is in progress.

The methods by which the above results were obtained will form the basis of a subsequent publication.

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[2] Labarre, *Bull. Acad. Roy. Med. Belg.*, **12**, 620; 1932.
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Experimental Deafness

CONTINUED investigation similar to that described elsewhere[1] has provided evidence for the view that the phenomenon designated 'auditory fatigue' by Ewing and Littler[2] and Rawdon-Smith[1] is in reality the result of the intervention of certain cortical factors. The marked decrease in sensitivity of the human ear following upon stimulation by loud pure tones for several minutes is, it has been found, not confined to the ear stimulated. The nominally unstimulated ear suffers a loss of sensitivity sometimes as great as that in the stimulated ear. Further, the loss of sensitivity in either may be temporarily removed or lessened in many cases by subjecting the observer to an unexpected stimulus (such as momentary darkness). This will be clear from Fig. 1.

It would seem, therefore, that the phenomenon of so-called auditory fatigue may more correctly be termed *inhibition*. The result of an unexpected stimulus is to produce momentary disinhibition.

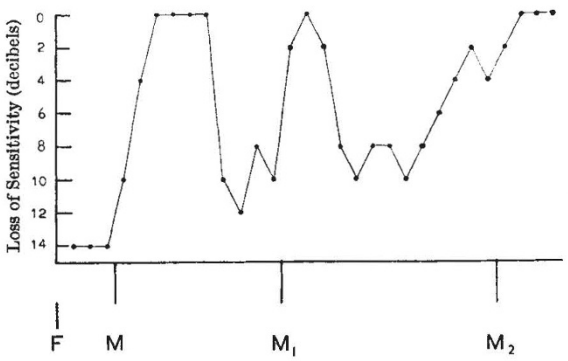

FIG. 1. Chart from actual record obtained from male subject. Each point represents one threshold reading. The points have been joined merely for convenience of interpretation—there is no indication that the threshold would fall on the line between readings. Frequency of stimulating and test tones = 2000 cycles per second. The stimulating tone ceased at *F*; *M*, M_1, M_2, represent points of application of momentary darkness in the sound-proof room in which the tests were made.

These effects find their nearest parallel in the phenomena of inhibition and disinhibition (or inhibition of inhibition) of conditioned reflexes described by Pavlov[3].

It is hoped shortly to publish these results in greater detail.

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The Band Spectrum of NH

IN the course of our investigation of the spectrum of discharges through streaming ammonia, it has been possible to record the λ 2530 band of NH, described by Hori[1], with a Hilger E.1 quartz spectrograph, using an improved type of hollow cathode. In addition, we have obtained four weaker bands at λλ 2730, 2835, 2885 and 2980 which appear to be due to the ion NH^+.

Our analysis with higher dispersion shows the necessity for revising Hori's interpretation of the λ 2530 band. The initial level is found to be $^1\Sigma^+$, while the final level is identified with the $^1\Pi$ level common to the λ 3240 band ($0,0\,^1\Pi \rightarrow {}^1\Delta$) and the λ 4502 band[2] ($0,0\,^1\Pi \rightarrow {}^1\Sigma^+$).

Full details will be published shortly.

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[1] *Z. Phys.*, V, **59**, 91; 1930.
[2] NATURE, **135**, 508, March 30, 1935. Cf. also forthcoming paper in *Proc. Roy. Soc.*, A.