deep sleep. The peak latency averaged 10.25 ± 1.22 msec during light sleep and 9.82 ± 0.58 msec during deep sleep. Both changes (initial − 0.30 msec and peak − 0.43 msec) observed during deep sleep are highly significant ($P < 0.01$).

With medial lemniscus stimulation (Figure B): (a) The initial latency of the radiation spike (which is thalamic in origin) recorded in the somatosensory cortex of 9 animals averaged 1.39 ± 0.16 msec during light sleep and 1.31 ± 0.13 msec during deep sleep; the peak latency averaged 1.88 ± 0.15 msec during light sleep and 1.78 ± 0.18 msec during deep sleep; the decrease in latency observed during deep sleep (initial − 0.08 msec and peak − 0.10 msec) is highly significant ($P < 0.01$). (b) The peak latency of the surface-positive wave of the response evoked in the somatosensory cortex of 9 animals averaged 4.83 ± 0.66 msec during light sleep and 4.59 ± 0.38 msec during deep sleep. The latency decrease (− 0.24 msec) observed during deep sleep is highly significant ($P < 0.01$).

With somesthesic radiation stimulation the peak latency of the surface-positive wave of the response evoked in the somatosensory cortex of 7 animals averaged 2.18 ± 0.39 msec during light sleep and 2.11 ± 0.27 msec during deep sleep. The latency decrease observed during deep sleep (− 0.07 msec) is not significant ($P > 0.05$).

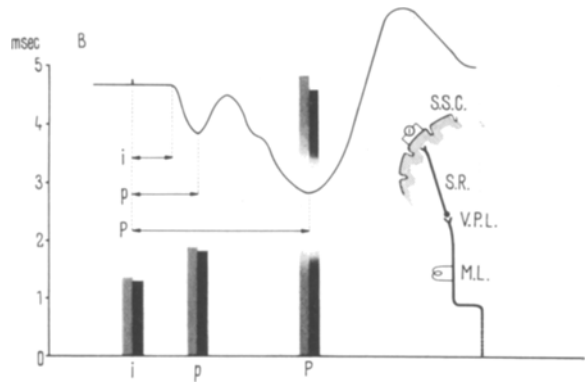

Fig. B. To show latency changes of different components of the response evoked in the somatosensory cortex by medial lemniscus stimulation. Histograms represent initial (i) and peak (p) latencies of the radiation spike; peak latency of the slow surface-positive wave (P). Same symbols as in A.

Conclusion. It is clear that during deep sleep the response evoked in the somatosensory cortex of the cat by shocks to the cutaneous nerve or medial lemniscus is not only higher[2] but has a shorter latency as well. Our experiments demonstrate that this decrease in latency occurs during transmission through the nucleus VPL. No change in synaptic transmission time was observed in nuclei gracilis and cuneatus or in the cortex itself.

Riassunto. La latenza delle risposte evocate nella corteccia somatica sia da stimolazione cutanea che del lemnisco mediale diminuisce significativamente con l'aumentare della profondità del sonno. Tale fenomeno è dovuto ad una accelerata trasmissione degli impulsi ascendenti a livello del nucleo ventro-postero-laterale del talamo.

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The Action of Iodoacetate on the Antigenic Power of Insulin

Confirmed evidence exists that the action of insulin depends, at least in part, on its disulphide group content[1,2] and on the fact that the biological effect of the hormone does not necessarily parallel its antigenic power[3]. This antigenic power of the hormone is, however, inhibited by iodoacetate, although differently from the way this compound inhibits the biological action of insulin in vitro[4-6].

In this work a study is made, using HALES and RANDLE's immunological method[7], of the action shown by iodoacetate on the antigenic power stability of bovine insulin.

Procedure. A test sample of insulin is treated with iodoacetate, according to the conditions explained in the text, and mixed with a constant amount of insulin ^{131}I. A limited quantity of anti-insulin serum is then added, and the insulin that consequently attaches itself to the antibody is precipitated quantitatively by the incorporation of a second antiserum obtained from the rabbit against the antigenic action of the guinea-pig serum. The precipitated complex is collected by the microfiltration in a millipore membrane and is recounted for radioactivity.

The antigenic power of insulin, as far as its reaction capacity with the specific antibody goes, may thus be assessed by comparison with a standard calculation curve prepared beforehand with pure commercial insulin.

Results. Stability of the antigenic power of insulin in the presence of iodoacetate: Table I shows how a sample of bovine insulin (100 μU/ml), kept at 0–4 °C and at pH 7.4 for 24 h, retains 64 and 85% of its original activity when incubated with $1 \cdot 10^{-4} M$ and $5 \cdot 10^{-5} M$ respectively of iodoacetate.

Action of the incubation time at 37 °C: Table II gives an illustration of the action of different concentrates of iodoacetate when incubated at 37 °C in the presence of a sample of insulin (200 μU/ml) at a pH of 7.4 for 120 min.

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The loss of hormone activity depends (though not to any proportionate extent) on the incubation time and the iodoacetate concentrate, a clearly inhibitive action becoming evident after 90 min and a concentration of $5 \cdot 10^{-5} M$ of iodoacetate.

Influence of the hormone concentrate with regard to its antigenic power stability: A study has been made of the effect of the insulin concentrate in relation to the stability of its antigenic power by exposing different samples of bovine insulin to various concentrates of iodoacetate. As will be seen from Table III, the increase in insulin concentrate to 300 μU/ml over an incubation time of 120 min fails to protect the antigenic stability of the hormone when placed in the presence of iodoacetate.

Table I. *Effect of iodoacetate on beef insulin stability*

Reagent present during incubation	Residual activity (μU/ml) at 24 h of incubation
None	100
$1 \cdot 10^{-4} M$ iodoacetate	64
$5 \cdot 10^{-5} M$ iodoacetate	85
$1 \cdot 10^{-5} M$ iodoacetate	98
$5 \cdot 10^{-6} M$ iodoacetate	97

Tubes contained 100 μU/ml of crystalline beef insulin and the indicated additions in phosphate buffer 0.040 M, pH 7.4. Incubation was carried out at 0–4 °C.

Table II. Effect of iodoacetate on beef insulin stability

Reagent present during incubation	Residual activity (μU/ml) Time (min)			
	0	30	90	120
None	200	190	175	160
$1 \cdot 10^{-4} M$ iodoacetate	–	139	70	65
$5 \cdot 10^{-5} M$ iodoacetate	–	154	92	82
$1 \cdot 10^{-5} M$ iodoacetate	–	159	106	90
$5 \cdot 10^{-6} M$ iodoacetate	–	180	170	150

Tubes contained 200 μU/ml of crystalline beef insulin and the indicated additions in phosphate buffer 0.040 M, pH 7.4. Incubation was carried out at 37 °C.

Inhibition of the antigenic power of the insulin by iodoacetate makes it clear that this reactive does not act exclusively as a blocker of the –SH groups, but also affects other structural groups necessary to the hormone's antigenic stability. MICHAELIS[8] reported in 1934 on the action of the iodoacetate on other non-sulphydric parameters of the proteins and showed the quick, easy action of this halogenated acid with the amino groups of the amino acids.

Nevertheless, HALIKIS and ARQUILLA[9], studying immunological aspects of insulin by using isothiocyanate fluorescein, came to the conclusion that the substitution of the amino groups inhibits the antigenic power of the hormone specifically.

Table III. Influence of iodoacetate on stability at several hormone concentrations

Concentration of hormone (μU/ml)	Residual activity (μU/ml) at 120 min of incubation				
	No iodoacetate	With iodoacetate $1 \cdot 10^{-4} M$	$5 \cdot 10^{-5} M$	$1 \cdot 10^{-5} M$	$5 \cdot 10^{-6} M$
150	112	68	80	84	88
200	160	65	82	90	150
300	220	100	148	180	230

Tubes contained the hormone concentration and the indicated additions in phosphate buffer 0.040 M, pH 7.4. Incubation was carried out at 37 °C.

Résumé. Nous avons observé que le iodoacétate en concentrations de $1 \cdot 10^{-4}$, $5 \cdot 10^{-5}$ et $1 \cdot 10^{-5} M$ et préalablement incubé à 37 °C, a une influence de l'ordre de 61, 50 et 44% respectivement sur la totalité du pouvoir antigénique de l'insuline de bœuf. L'effet inhibitif est proportionnel à la durée d'incubation et l'on ne constate pas de différences lorsque les concentrations de l'hormone se trouvent entre 100 et 300 μU/ml.

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Different Activation of the Two Types of the Pyramidal Tract Neurones Through the Cerebello-Thalamocortical Pathway

The motor area of the cat's cerebral cortex receives histologically a projection from the nucleus ventralis lateralis (VL) of the thalamus[1], which is innervated by the intracerebellar nuclei[2]. Activation of the pyramidal tract neurone (PTN) through this cerebello-thalamocortical pathway has been demonstrated by several authors[3–5]. Intracellular recording has further revealed that by stimulating VL directly EPSP's were induced from PTN's with short latency[6,7]. However, PTN's can be classified into two groups by the axonal conduction velocities in their antidromic activation: the faster group and the slower[8,9]. So we investigated the VL-evoked EPSP's with special reference to the axonal conduction velocities of each PTN, and a clear difference was found between the fast and the slow PTN's.

Cats were anaesthetized with pentobarbitone sodium (30 mg/kg). Bipolar concentric electrodes for stimulation were inserted to VL and to the brachium conjunctivum (BC) stereotaxically. After the removal of the oesophagus and the basilar bone, stimulating electrodes of enamel-