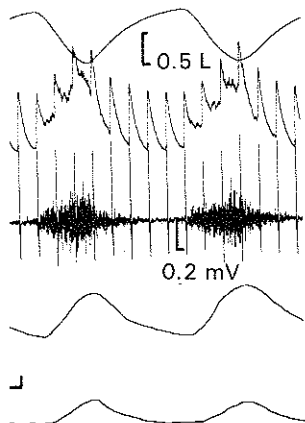


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phragm using fixed electrodes (1 figure).

oesophageal electrodes are poor reflect sture are changed (GANDEVIA & Mc KEN: needle electrode (DELHEZ, 1974). How- orax. We therefore undertook this study ding by « temporary myocardial electro-

dy. Informed consent was obtained from iratory disease. All these patients under- of the surgical procedure, two temporary sternal part of the right diaphragm (inter- amplified using a Medelec AA6M ampli- elec analogic integrator (RC : 0.5 s). The ferences were measured using two Schae- onned at umbilical and mamillary levels. ograph. All those parameters were simul- rt recorder. The patients were examined rdings were performed during quiet tidal vers.



aseline during each examination. Integra- So we were able to measure the peak and $r : 0.84 - 0.92$) is found between the inte- y this technique, diaphragmatic electrical ic and abdominal predominant maneuvers. itients undergoing thoracic or upper abdo- llows an accurate measurement of diaph- terns.

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Influence of tianeptine and clomipramine on the electrical activity of rat monoaminergic neurons and hippocampus pyramidal cells.

Tianeptine (T) is a new potential antidepressant (AD) active in classical pharmacological tests used for AD screening. Biochemical studies *ex vivo* indicate that T increases 5-HT uptake after acute and chronic administration without modification of NA uptake. This property differentiates T from classical tricyclic AD which are potent inhibitors of 5-HT and/or NA uptake.

Electrophysiological studies were performed in order to compare the influence of T and clomipramine (CIMI) on the firing rate of central locus coeruleus (LC) noradrenergic neurons, dorsal raphe (DR) serotonergic neurons and hippocampus CA1 pyramidal cells.

The interaction of T and CIMI with the response of CA1 cells to iontophoretically applied 5-HT or GABA was also investigated.

Multibarrel glass micropipettes were implanted stereotaxically in male Wistar rats anaesthetized with chloral hydrate. The central barrel filled with NaCl 2M saturated with fast green was used for recording action potentials. Side barrels were filled with a solution of acetylcholine chloride 0.02M in NaCl 0.2M pH 4, GABA 0.001M in NaCl 0.1M pH 4, 5-HT creatinine sulphate 0.02M in NaCl 0.02M pH 4, CIMI HCl 0.1M pH 4 or T 0.1M pH 9 and NaCl 4M for automatic current balance.

In systemic studies T or CIMI were perfused into the jugular vein by means of a perfusion pump (flow 6 ml/h). In iontophoretic studies, 5-HT or GABA were applied by pulses of 60 s at constant current before, during and after the iontophoretic application of T or CIMI.

In systemic studies the dose of drug required to produce a 50% inhibition (ID_{50}) or activation (ED_{50}) of the firing rate was calculated. In iontophoretic studies, the responsiveness of CA1 cells to 5-HT or GABA was evaluated by calculating the mean percent inhibition produced by the iontophoresis of 5-HT or GABA. Statistical analysis was performed using Student's *t* test for paired samples. The recovery after 5-HT or GABA was measured by calculating the percentage of recovery 10, 20 and 30 s after the iontophoretic application. Statistical comparison of the recovery curves was realized using Zerbe method.

The i.v. perfusion of CIMI decreases the firing rate of DR, LC and CA1 neurons. The i.v. perfusion of T does not modify the firing rate of DR neurons, decreases the firing rate of LC neurons and increases the firing rate of CA1 pyramidal cells. Quantitative values are given in Table I.

The iontophoretic application of CIMI (6-10 nA) on CA1 neurons selectively potentiates the responsiveness of these cells to 5-HT ($P < 0.01$) but not to GABA. The

TABLE I. Mean (\pm SE) effective doses (in mg/kg) of CIMI or T required to produce a 50% inhibition or activation of LC, DR or CA1 pyramidal cells after i.v. perfusion. *n* = number of animals.

	CIMI	<i>n</i>	Tianeptine	<i>n</i>
DR	0.34 \pm 0.02	6	no effect	6
LC	3.08 \pm 0.30	6	1.74 \pm 0.20	5
CA1	0.27 \pm 0.05	7	0.68 \pm 0.17	8

iontophoretic application of T (10-14 nA) does not induce a significant modification of the responsiveness of CAI cells to 5-HT or GABA. The iontophoretic application of CIMI significantly increases the recovery time after 5-HT ($P < 0.005$).

The iontophoretic application of T significantly decreases the recovery time after 5-HT ($P < 0.001$) and GABA ($P < 0.005$).

T and CIMI differ in their influence on the firing rate of monoaminergic neurons and CAI pyramidal cells.

The inhibitory effect of CIMI on the firing rate of DR and CAI neurons after i.v. perfusion is probably related to an increased availability of 5-HT in the synaptic cleft due to the uptake inhibition. The similarity between the ID_{50} values supports this interpretation. Furthermore iontophoretic studies with CIMI show a selective potentiation of the inhibitory effect of 5-HT on CAI cells.

The unselective effect of CIMI on the recovery time after 5-HT or GABA may be related to the inhibitory effect of CIMI itself on the firing rate of CAI cells after prolonged application.

The effect of CIMI on LC neurons may be related to its weak inhibitory effect on NA uptake.

T has no effect on the firing rate of DR neurons, decreases the firing rate of LC neurons and increases the activity of CAI cells. The activation of CAI cells seems in agreement with an increased uptake of 5-HT. However the only indication of an interaction between T and 5-HT mechanisms in our studies is the decreased recovery time after 5-HT and this effect is not specific as the recovery time after GABA is also decreased.

In conclusion : T appears to have an original electrophysiological profile in comparison with classical AD. Its mechanisms of action remain to be elucidated.

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Pain-related cerebral evoked

To the best of our knowledge quantitative assessment of the techniques for deriving the cerebral the first time a wave appearing nociceptive electrical shock applied by Fentanyl and enhanced

Following an early cortical later negative rounded wave progressively and is fully developed the tail and at 8 times the threshold over the parietal area. The wave and parietal areas. At its threshold Over the frontal area, where the "receptive" wave appears as the first

This wave disappears 5 minutes reappears progressively in 45 minutes restored by i.m. injection of morphine accurately and may provide a clue. Both the rather short peak-late suggest peripheral conduction block. A very similar wave has been reported (1984).

Finally, more subtle modifications Fentanyl administration. A second component derived over the frontal area accompanied by the first positive wave. In other words tanyl was accompanied by a morphine

It is hoped that this method of comparing the effect of different compounds

Reference

CHAPMAN, C. R. & JACOBSON, R. C. (1984) *of Pain* (BROMM, B., ed.) pp. 233-240.