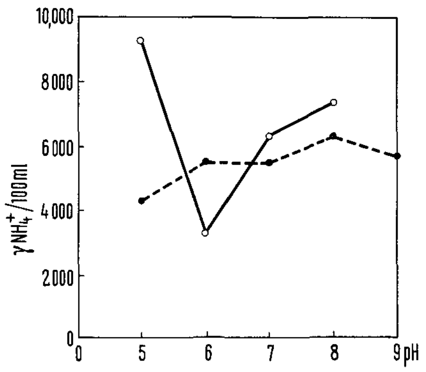
Discussion. The results presented here indicate that some factor which is also capable of hydrolysing glutamine could be dialysed out of the homogenates of both the normal kidney and the tumour. The presence of proteins in the dialysates and also the fact that this factor was heat-labile indicate the possibility of an enzymatic nature of this factor. This may be more clearly indicated by the high affinity of the dialysates towards the substrate and also by their pH optima.

The data presented here might indicate the possibility of the glutaminase being broken down into smaller molecules and the active core of the enzyme may be passing through the bag. Further work is, however, in progress to characterize this protein and study its properties.



Glutamine hydrolysing capacity of the dialysates obtained from kidney (normal) and tumour homogenates at different pH. ○—○, Kidney dialysate; ●—●, tumour dialysate.

Résumé. Dans les reins et dans les tissus de tumeur un facteur provoquant une glutaminase a été mis en évidance. Cette activité est plus faible dans les reins. Le dialysat du rein accuse deux pH optima de 5.0 à 8.0, tandis que celui de la tumeur n'a qu'un pH optimum de 8.0.

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Inhibition of Galvanic Skin Response by the Splanchnic Afferent

Preliminary experiments have shown that electrical stimulation of the somatic afferent nerve, as well as the splanchnic and the vagal afferent nerves, causes inhibition of galvanic skin response (GSR) in cats[1]. Very little is known, however, about the nature of the primary afferent concerned with these phenomena. In the present paper, the component of the splanchnic afferents participating in the reflex inhibition of GSR was investigated from the correlation between the neurogram and inhibitory effects of the splanchnic stimulation.

Material and methods. Under ether anesthesia, bronchial canula and saphenous vein canula were inserted into 16 cats. GSR was recorded from the paws of the anterior limbs by means of Ag-AgCl electrodes. EEG, femoral arterial pressure and heart rate were recorded simultaneously. The great splanchnic nerve was severed just proximal to the coeliac ganglion and was isolated from surrounding tissues up to the sympathetic trunk intrathoracically. The animals were immobilized with Flaxedil and maintained with positive pressure respiration. Stimulation electrodes were laid on the proximal portion of the nerve and the neurogram of the whole nerve was recorded at the distal end. All the nerves exposed were protected with warmed paraffin-oil. Rectangular pulses of 0.01 msec duration with variable intensities were applied repetitively, usually at a frequency of 5/sec.

In order to evoke GSR, the common peroneal nerve was stimulated every 45 sec for 200–500 msec with a train of pulses of 5 msec duration at a rate of 10/sec and an intensity adjusted to evoke GSR. In order to keep the amplitude of evoked GSR constant for a long period, body temperature was maintained constant and the depth of anesthesia (Urethane or Nembutal) was adjusted to a state in which evoked GSR was large and stable and spontaneous background activities were small.

Results and discussion. After securing constant amplitude of evoked GSR, splanchnic stimulation was applied for a few minutes, including 2 or 3 evoked GSR within the stimulation period. As shown in Figure A, evoked GSR was depressed almost completely during the period of splanchnic stimulation. In 30 out of 82 cases, depression of amplitude of GSR due to splanchnic stimulation was observed without changes in blood pressure; in 41 cases, it was accompanied by a fall of blood pressure and in 11 cases by a rise. Thus it can be concluded that the depression of GSR is not due to changes in the blood pressure. The neurogram of the splanchnics revealed 4 components, when increasing intensity of the stimuli. The first 3 components of the compound action potential of the splanchnic nerve are shown in Figure B. As was to be expected, threshold for the first component (TI) was lowest. Threshold of the second, the third and the fourth component was approximately 3, 8, and 25 times higher than TI. Relationship between intensity of stimulation and the inhibitory effect was examined in 9 cases, giving all similar results. Figure C shows one typical example. Mean amplitude of 2 GSR during splanchnic stimulation period and of 6 GSR in the control period (each 3 successive GSR of before and after splanchnic stimulation) were measured. In the ordinate, the degree of inhibition of GSR is expressed as

$$\left(1 - \frac{\text{mean amplitude of GSR during stimulation period}}{\text{mean amplitude of GSR in the control period}}\right) \times 100.$$

The abscissa shows the intensity of the stimulus expressed in multiples of TI. Stimulation of the first component fibers only was not accompanied by any changes on GSR. Depression of GSR set in when stimulus intensity was elevated to excite the fibers of the second component, the

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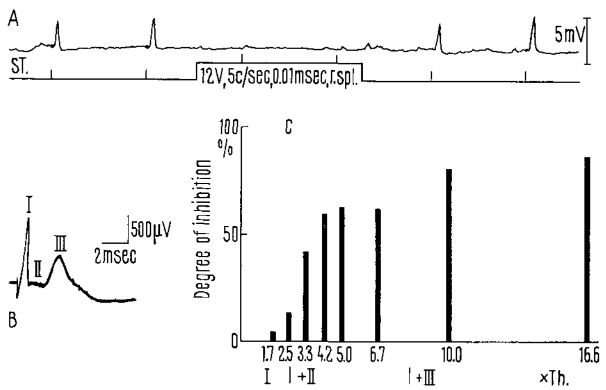
depressant effect thereafter increasing steeply with increasing magnitude of the second component. Further increase in depressant effect of the stimulation occurred in the realm of the stimulus intensity for the third component, however, the rate of increase was only slight in spite of the rapid augmentation of the amplitude of this third component. It can therefore be concluded that inhibition of GSR is due to activity carried in the fibers of the second component of the compound action potential. Mean value of the maximum conduction velocity of this component was 23 m/sec. Frequency characteristics of the splanchnic stimulation were investigated at frequencies: 0.3, 0.5, 1, 3, 5, 10, 50 and 100/sec, with stimulus intensity



(A) Amplitude of evoked GSR recorded from right paw of the anterior limb is depressed during splanchnic afferent stimulation. Stimulation-evoking GSR was applied to the common peroneal nerve at every 45 sec (signal on the baseline). The right splanchnic nerve was stimulated repetitively with rectangular pulses of 0.01 msec duration, 12 V intensity, at a rate of 5/sec, during the period shown by upward deflexion of the baseline. (B) Neurogram of the splanchnic nerve. 3 components are indicated by Roman numerals. The fourth component is not shown. (C) Relationship between degree of inhibition of the GSR (ordinate) and stimulation intensity expressed in multiples of the threshold of the first splanchnic component (Tl) (abscissa). The right splanchnic nerve was stimulated at a rate of 5/sec, with rectangular pulses of 0.01 msec, for 90 sec; threshold for the first component (Tl) was 1.2 V. Roman numerals at the bottom indicate the appearance of the respective component in the neurogram.

kept constant at a range of the second component. The inhibitory effect sets in at 0.5/sec; reaches a maximum at 5/sec or at 10/sec and decreases with further increase of the stimulation frequency.

Sympathetic reflex discharge elicited by splanchnic or somatic afferent stimulation was investigated intensively by PERL et al. and DOWMAN et al. Both of them reported marked depression of the sympathetic reflex discharge by prior conditioning of splanchnic or spinal afferent fibers[2,3]. The conduction velocity of these fibers lies between 15–35 m/sec[2]. DUDA[4] reported that the somatic polysynaptic reflex was inhibited by splanchnic afferent stimulation as well as by cutaneous afferent. In our preliminary experiment, it was found that GSR was inhibited by afferent fibers of the common peroneal nerve with maximum conduction velocity between 20 and 30 m/sec[1]. This value corresponds with that obtained on the splanchnic nerve reported here. These data mentioned above suggest that a common afferent system may operate in somatic and in visceral nerves to produce inhibition of a visceral and a somatic motor outflow. Further experiments are, however, still needed to clarify this problem[5].

Zusammenfassung. Der hemmende Einfluss wiederholter elektrischer Reizung der Afferenzen des N. splanchnicus auf den galvanischen Hautreflex (GHR) wurde an Katzen untersucht. Gleichzeitige Registrierung des Neurogrammes des N. splanchnicus zeigt, dass die reflektorische Hemmung des GHR durch Fasern der A-δ Gruppe hervorgerufen wird.

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Nachweis der Selbst-Erregung der circadianen Periodizität bei Carabiden (Coleoptera Ins.)*

Carabiden zeigen eine starke Abhängigkeit ihrer tageszeitlichen Laufaktivität vom zeitgebenden Licht-Dunkel-Wechsel, wie besonders die extrem schnelle Resynchronisation nach Phasenumkehr des Zeitgebers und die Synchronisation durch 8/8-Std-Tage zeigen[1]. Es war aus diesen Gründen fraglich, ob die auch bei Carabiden im Konstantversuch auftretende Circadian-Periodik selbsterregt (ungedämpft) ist oder lediglich ein gedämpftes Nachschwingen nach Erregung durch Aussenreize darstellt. Zur Entscheidung kann das Verhalten der Amplitude im Konstantversuch weniger gut herangezogen werden, da – im Falle der Fremd-Erregung – die Schwingung auch im Konstantversuch durch gelegentliche, aperiodische Reizveränderungen wieder aufgeschaukelt werden könnte. Hier soll zur Klärung der angeschnittenen Frage das Verhalten bei Zeitgebern verschiedener Frequenz herangezogen werden. Der Mitnahmebereich ist im Falle der Selbst-Erregung beschränkt; ausserhalb seiner Grenzen setzt sich eine endogene Circadian-Periodizität durch. Fremd-erregt schwingende Systeme lassen sich hingegen von beliebigen Zeitgeberfrequenzen mitnehmen[2].

Der Nachweis der Selbsterregung, der an Einzelorganismen durchzuführen ist[2], wurde auf diesem Wege bisher vor allem für Vertebraten erbracht, während bei Insekten positive Befunde für *Periplaneta* und *Leucophaea* (beide Blattoidea) vorliegen[3].

* Mit Unterstützung durch die Deutsche Forschungsgemeinschaft.
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