Ascending Noradrenaline Neurons from the Pons and the Medulla oblongata

The existence of noradrenaline (NA), dopamine (DA) and 5-hydroxytryptamine (5-HT) neurons in the central nervous system have recently been described (for references see [1]). It has inter alia been discovered that practically all the NA, DA and 5-HT terminals in the diencephalon and the telencephalon belong to ascending neurons with the cell bodies in the lower brain stem[2–6]. However, it has as yet not been possible to demonstrate the exact localization of the NA cell bodies. Such a mapping out has been made in the present investigation using a combined histochemical and biochemical approach.

About 20 adult male Sprague-Dawley rats have been used. Electrothermic lesions[7,8] were made at the junction between the pons and the mesencephalon on the left side 7–10 days before killing. The lesions mainly occupied the entire lateral reticular formation. The NA and the DA on each side of the diencephalon plus the telencephalon were determined biochemically in 8 of the animals[9,10]. The rest of the animals were taken for histochemical demonstration of NA, DA and 5-HT in the telencephalon and the diencephalon[11,12].

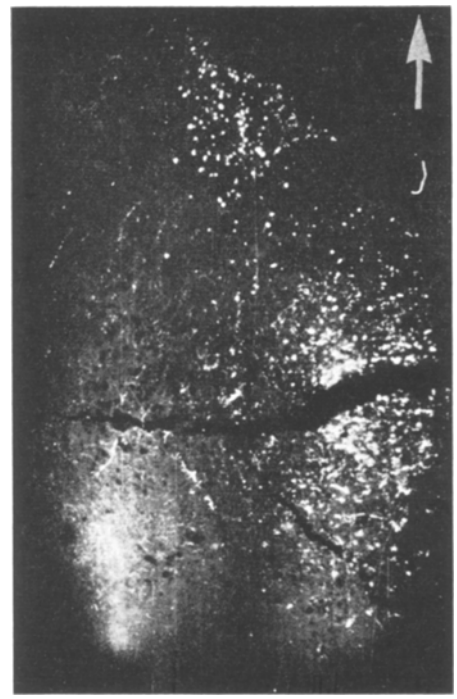
In the vast majority of the areas in the telencephalon and the diencephalon on the operated side, there was a marked to very marked decrease in the number of NA nerve terminals, e.g. in the neocortex, the limbic forebrain structures and the hypothalamus. However, the DA nerve terminals, e.g. in the neostriatum (the caudate nucleus-putamen), the tuberculum olfactorium, the nucleus accumbens and the median eminence, appeared intact on both sides. The histochemical results are in complete agreement with the biochemical data. The NA level in the telencephalon plus the diencephalon on the operated side was 0.13 μg/g (s.e.m. = 0.012, n = 8), whereas on the intact side it was 0.41 μg/g (s.e.m. = 0.083, n = 8), i.e. about normal. The difference is highly significant ($P < 0.001$). No obvious changes were found in the DA levels on either side. Thus, at least two thirds of the NA terminals in the telencephalon and the diencephalon must belong to neurons with the cell bodies caudal to the mesencephalon, whereas practically all the DA cell bodies are present mainly in the mesencephalon and partly in the diencephalon.

The monoamine nerve terminals in the pons, the medulla oblongata and the spinal cord were intact. Thus, most if not all of these terminals must derive from fibres originating from cell bodies in the pons and the medulla oblongata[13].

Immediately proximal to the lesion, a marked accumulation of catecholamines (CA) was observed in nerve fibres forming a large lemniscus in the lateral part of the mesencephalic reticular formation (Figure). The lemniscus was oriented mainly in a dorso-ventral direction. Such an accumulation proximal to a lesion has previously been found to be typical for monoamine neurons[5,14].

A marked increase in fluorescence intensity was observed in swollen CA cell bodies in the lateral reticular formation of the medulla oblongata (group A1[15]) and in all CA cell bodies of the pons (group A5–A7), e.g. in the locus coeruleus. Most of these retrograde cell body changes (cf. [6, 13]) were found on the side ipsilateral to the lesion. In the medulla oblongata such changes were observed also in some cell bodies on the contralateral side. However, most of the NA neurons appeared uncrossed.

The fibres from the medulla oblongata reached the mesencephalon via the dorsal part of the reticular formation in the medulla oblongata, and then the reticular formation of the pons just medial to the outgoing fibres of the facial nerve and joined the fibres from the NA cell bodies in the pons at the level of the nucleus motorius



Caudal part of the mesencephalon of normal rat just caudal to an electrothermic lesion made in the lateral reticular formation. Transverse section. A large lemniscus of strongly green-fluorescent fibres with a marked accumulation of CA is present. The fibres are transversely cut and appear as fluorescent dots. The ventro-dorsal direction is indicated by the arrow. × 100.

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nervi trigemini where all the fibres lay immediately medial and dorsomedial to this nucleus[16].

Zusammenfassung. Nach einseitigen Läsionen im Rattenhirn an der Grenze zwischen Pons und Mesencephalon, konnte auf Grund von anterograden und retrograden neuronalen Veränderungen gezeigt werden, dass mindestens $^2/_3$ der noradrenergenen Endigungen im Telencephalon und Diencephalon hauptsächlich zu den ungekreuzten, von Zellkörpern aus Pons und Medulla oblongata stammenden, Axonen gehören.

[16] Acknowledgments: This work has been supported by research grants from the National Institute of Neurological Diseases and Blindness (NB 05236-01, NB 04359-02), US Public Health Service and from the Swedish State Medical Research Council (14X-502-01, 12X-715-01). – For technical assistance we are grateful to Miss Birgitta Nilsdotter-Högberg.

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The Effect of some Neurohormones on the Heart Rate of Spiders

The authors of the present paper studied the effect of acetylcholine and adrenalin on the heart rate of the spider *Tegenaria atrica* C. L. Koch, by the method used by Mikulska and Kokociński[1] and using electroencephalographic recording.

Isolated weighed abdomens were immobilized on the stage of a stereoscopic microscope, after which two steel needle electrodes of 5–10 μ diameter, were introduced hypodermally at two symmetrical points with the help of a micromanipulator. The electrodes were connected to a Kaiser electroencephalograph and the actional potentials of the heart were recorded at a time constant of 0.6 or 1.0 sec, the instrument being calibrated at 50 μV/3.5 mm and the high frequency filter on.

The intracardiac injections of adrenalin and acetylcholine were made using an Agla microsyringe connected to glass needles controlled by means of a micromanipulator. The required concentrations of the neurohormones were obtained by diluting them in Jager physiological solution. Test injections did not produce any change in the action of the heart.

The results obtained so far are as follows: injections of adrenalin in a concentration of 10^{-4} caused a threshold effect in the form of a slight acceleration of the heart rate, but without any definite change in the amplitude of the curve. The threshold amount of adrenalin per 1 mg of preparation giving a positively chronotropic effect was $1.1 \cdot 10^{-6}$ mg. Dosed $5.2 \cdot 10^{-6}$ mg per 1 mg, adrenalin gave an acute effect characterized by a rapid decrease of the amplitude and an acceleration of the heart rate. The return to normal was comparatively slow (Figure 1). Further increase of the adrenalin dose led to a levelling of the amplitude and to a stop in the action of the heart.

The injections of acetylcholine gave a negatively chronotropic effect. Already in a concentration of

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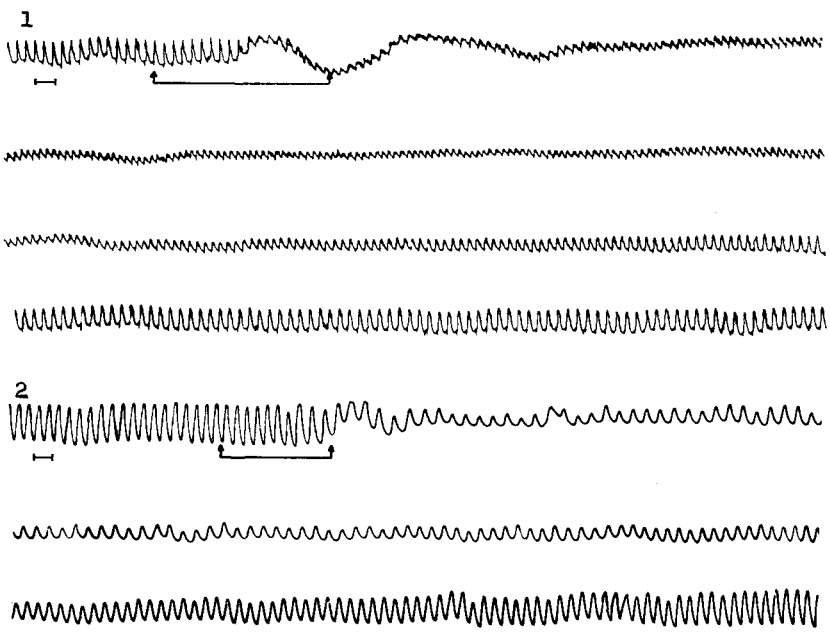

Fig. 1. Electrogram of heart in isolated abdomen. Injection of adrenalin $5.2 \cdot 10^{-6}$/mg of weight of abdomen. ▲—▲ = injection; |—| = sec.

Fig. 2. Electrogram of heart in isolated abdomen. Injection of acetylcholin $2.6 \cdot 10^{-6}$/mg of weight of abdomen ▲—▲ = injection; |—| = sec.