Rates of responding for heat and/or intracranial stimulation (ICS), and post-test rectal temperature

Subject-electrode	Current (μA)	Non-choice baseline				Choice			
		Heat reinforc. per min ⌀-5 °C	Tre (°C)	ICS per min ⌀ 25 °C	Tre (°C)	Rate/min ⌀-5 °C Heat	ICS	Tre (°C)	Test duration (min)
A) Posterior hypothalamic electrodes									
1–5	225	5.1	38.4	108	38.9	0.04	51	21.4	110
3–3	624	4.3	38.5	87	38.0	0.12	37	24.4	52
3–5	624	–	–	65	38.8	0.62	35	25.6	58
13–2	386	6.0	38.0	78	38.6	0.99	27	22.0	112
17–5	295	4.0	38.4	72	38.1	0.38	43	24.3	64
Group mean		4.85	38.32	82	38.36	0.43	38.6	23.54	79
B) Rostral hypothalamic electrodes									
2–4	545	4.9	37.5	57	39.2	1.8	17	34.2	60
3–2	995	4.3	38.5	24	39.4	3.3	21	38.2	60
4–1	995	4.3	38.2	23	39.1	3.0	21	38.2	60
15–5	545	5.4	38.0	67	39.3	1.9	16	36.8	60
18–2	545	5.9	38.4	103	38.2	2.9	61	37.2	60
18–4	545	–	–	63	38.4	4.2	26	37.6	60
Group mean		4.96	38.12	56.8	39.0	2.85	27.0	37.0	60

zero, and feeble attempts to press the heat lever or escape from the cage followed. Average posttest Tre was 23.5 °C, a value that is quite close to the critical temperature for self-stimulation of 22–23 °C[9]. Figure A shows the cumulative record of an ICS-persistent rat. All animals with rostral hypothalamic electrodes alternated between the heat and ICS levers; the reduction in rate of obtaining heat varied between 29–65%. The lowest Tre for an animal in this group was 34.2 °C, while the mean was 37.0 °C.

Subsequent testing showed these results were replicable, and could not be accounted for by a fortuitous current selection or simple perseverative responding on the ICS lever. Animals with rostral electrodes alternated between the heat and ICS levers when current was increased, while subjects with posterior electrodes worked for ICS until current values near threshold were attained and then switched to continuous responding for heat. A compulsive response automatism on the ICS lever by subjects with posterior electrodes was ruled out by making ICS available only 50% of the time; the on-off intervals were 10, 15, or 20 min. The output of the stimulator was disconnected at the end of an ICS interval, while 2 free stimulations were given to signal the availability of ICS at the end of a time-out period. Heat was available at all times. The subjects worked for ICS when it was available, but promptly began to press for heat when it was not. Figure B shows an example of alternation obtained in this manner.

Briese et al.[5,6] have suggested an overlap of temperature and reward systems based on a dual-center model of the thermoregulatory system proposed by Benzinger[10]. Persistent responding for posterior ICS in the cold is consistent with this view, but hyperthermia with rostral ICS is not. The difficulties involved in a consistent application of the Benzinger model to ICS data could mean either that the model is inadequate or that the drive-interaction paradigm itself is inadequate[11].

Résumé, Placés dans un environnement froid, les rats qui avaient des électrodes implantés dans l'hypothalamus postérieur, préféraient la stimulation intracrannienne à un réchauffement de l'environnement; de ce fait, ils devenaient hypothermiques. Les rats qui avaient des électrodes implantés dans l'hypothalamus antérieur actionnaient alternativement les leviers de chauffage et ceux du courant électrique; ils évitaient ainsi de devenir hypothermiques.

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Vagus Pneumonia as Membrane Phospholipase Activation

Lissák et al.[1] pointed to a sympathetic predominance after vagotomy. We tried to approach the direct site of sympathetic neural control in the lipid metabolism of cytosomal fraction, obtained from the rabbit lungs after vagotomy.

We conducted the experiments on rabbits weighing from 1.6–2.4 kg. To avoid the aspiration the rabbits were tracheotomized after the bilateral cervical vagotomy. A similar procedure was carried out on sham operated rabbits without vagotomy. 5 h after vagotomy rabbits were exsanguinated and the lungs removed immediately. Cytosome fraction of lung was obtained by means of centrifuging with 20,000 *g*, according to procedure of Reiss[2] after preparing homogenates in 0.3 *M* sucrose

solution in a Potter-Elvehjem homogenizer. Extraction of the lipids was carried out with chloroform and methanol in a Waring Blendor according to Bligh and Dyer[3]. Total lipid was determined by weighing a dried aliquot of the chloroform portion. Total phospholipid was determined by quantitative analysis of phosphorus using Fiske and Subarrow's[4] procedure of perchloric acid digestion. To determine the free fatty acids we used the micromethod of Dole[5]. Protein contents were determined by the method of Lowry[6] and lipids were referred to 100 mg protein.

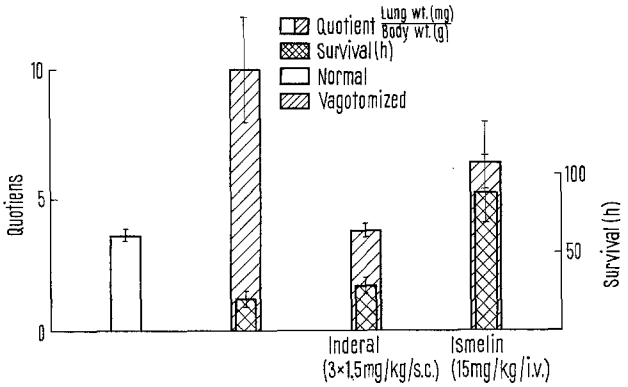

Fig. 1. Survival and lung quotient of normal and vagotomized rabbits.

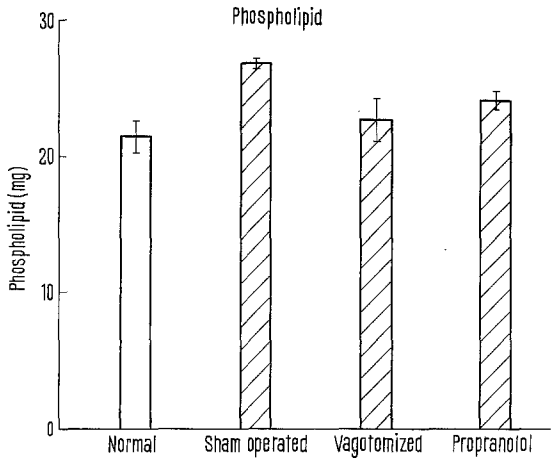

Fig. 2. Phospholipid content of cytosomal fraction from rabbit ungs, referring to 100 mg of protein.

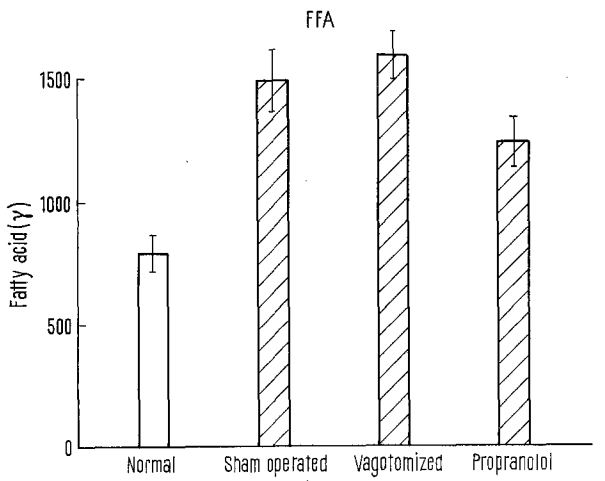

Fig. 3. The FFA level of cytosomal fraction from rabbit lungs, referring to 100 mg of protein.

In the first part of our experiments we measured the effect of different sympatholytics on the survival and lung quotient of vagotomized rabbits (Figure 1). The relatively standard lung quotient under normal conditions significantly increased postvagotomy. The inhibitory effects of sympatholytics appeared separately. Propranolol, a known type of β-receptor blocking agents, reduced the lung quotient without increasing the survival. Guanethidine, which deplets the catechol-amin content in the lungs, increased the survival but its effect on the quotient was not full (Figure 2).

The phospholipid content shows an absolute increase in the sham operated group and a limited increase also appears in animals treated with propranolol. The quantity of phospholipids in the vagotomized group remains about at the normal level. In Figure 3 we present the FFA levels of animals under normal conditions and in sham operated, vagotomized and propranolol treated group of animals. There is a very little difference between the values of sham operated and vagotomized group in relation to the normal level of FFA. The propranolol brought the elevated level of FFA nearer to the normal value.

The elevation of FFA level is followed by an increase of phospholipid content in the sham operated group but it is not followed by any change in the vagotomized group, however, indicates a remarkable reduction, and the higher value of FFA may come from this reduction in this group. This undoubtedly neurally-induced phospholipase effect can be diminished by propranolol, which decreases the FFA level and accordingly raises the quantity of phospholipids.

The vagotomized organism is basically characterized by an overbalanced vegetative regulation and the 'vagus pneumonia' by a sympathetic predominance resulting from the pathological regulation. The relative fall of phospholipid content and the elevated level of FFA indicates a phospholipase activation after vagotomy. Both of these changes can be inhibited by propranolol.

Zusammenfassung. Die relative Abnahme des Phospholipid-Gehalts und das erhöhte Niveau des FFA weist auf eine Phospholipase-Aktivität nach Vagotomie hin. Diese Befunde können durch Propranolol gehemmt werden.

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