The present data indicate noteworthy sex differences with regard to food intake in rats lesioned at different ages. The unaffected absolute food intake during the first post-operative week in the male weanlings might be attributable to a decreased sensitivity of the lateral 'feeding center'[4] in the weanling male to the immediate effect of the lesion in the adjacent 'satiety center'[4]; the female weanlings[2] showed a depression of absolute food intake during this period. It is noteworthy, however, that already during this experimental period the oldest rats of both sexes showed gross hyperphagia.

It is of interest also that, while female rats lesioned at the age of 59 and 75 days, respectively, displayed hyperphagia throughout the experiment, their male counterparts began to plateau during the fifth week to control levels; in the same vein is the finding in the 140-day-old males which did not show the gross hyperphagia that was evident in the female rats of the same age when compared with their respective intact controls. This suggests that ventromedial lesions elicit greater hyperphagia in the adult female than in the adult male rat.

The data show also that, when food intake is expressed relative to body weight, weanling male rats consume more than corresponding females. The oldest rats with lesions, both male and female, however, show food intakes similar to that of their controls.

Expression of food intake relative to a seemingly more meaningful parameter, i.e. KLEIBER's 'metabolic size'[3] shows that there is little difference in whether food consumption is referred to this parameter or to body weight.

The data show that destruction of the ventromedial satiety neurons in the weanling male rat has no immediate effect on food intake and suggest a lesser sensitivity of the appropriate hypothalamic neurons[4] to the effect of the lesions. This is in contrast to the female rats. The data also reveal that, relative to body weight, the lesioned weanling rats ate more than older rats with lesions; female weanling rats ate similar amounts to older rats with lesions. Furthermore, male rats lesioned at later ages develop hyperphagia but this is not as pronounced as in correspondingly aged females. Male rats with lesions placed during adulthood plateau during the fifth week after the operation while corresponding females continue to show hyperphagia.

The sex difference in food intake may be related to a difference in body composition of males and females. That adipose tissue may play a role in the long-term regulation of food intake has been suggested by KENNEDY[5]. It is conceivable that the greater degree of adiposity in the lesioned female rats[2] than in the operated male animals did affect food intake[6].

Zusammenfassung. Im Gegensatz zu weiblichen Ratten zeigen männliche Tiere mit Läsionen des N. ventromedialis hypothalami im Alter von 26, 59, 75 und 140 Tagen kurz nach der Ablaktation keinen unmittelbaren Einfluss auf die Futteraufnahme, Erwachsene nie die in den gleichaltrigen weiblichen Ratten beobachtete Hyperphagie. Männliche Ratten erreichen das Kontrollniveau schon nach 5 Wochen, während die weiblichen Tiere noch ausgeprägte Hyperphagie zeigen.

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Hemodynamic Studies in the Circle of Willis in the Rat

During the last 20 years a great deal of biochemical and related research has been conducted using rats rendered anoxic by ligation of both common carotid arteries for varying periods of time[1,2]. This study was undertaken to aid in assessing the functional significance of the vertebral arteries in the rat and thus to elucidate the biochemical results.

Methods. Adult male albino rats (Wistar strain) averaging 450 g in weight and anaesthetized with interperitoneal pentobarbital sodium (4 mg/100 g) were studied. Artificial respiration was used when necessary. The lingual arteries served as the recording sites to measure the back pressures of the circle of Willis as reported by AYALA and HIMWICH[3] for the dog. Occipital, pharyngeal and superior thyroidal arteries were ligated near their origin and the carotid sinus baroreceptors denervated with 10% formalin. The catheters used were intramedic polyethylene tubing of 3 sizes telescoped together: I.D. 0.034 in. · O.D. 0.060 in., I.D. 0.023 in. · O.D. 0.038 in. and I.D. 0.011 in. · O.D. 0.024 in. Actual recording was accomplished from the right and left lingual and systemic (femoral) sites simultaneously on a type 'R' Offner Dynograph through Model P23 Gd Statham pressure transducers. A definite sequential pattern of clamping was followed throughout the experiment. The pressures were allowed to equilibrate between each clamping (45 sec) and at the end of each series (3 min). On each experiment 2 clamping series were followed: The first consisted of the occlusion in sequence of the left common carotid artery (LCC), the right common carotid artery (RCC), the left vertebral artery (LV) and, the right vertebral artery (RV); the second series was a reversal and consisted of occlusion of the RV, LV, RCC and LCC. Readings were taken immediately after occlusion and again approximately 40 sec later; thus 16 readings in addition to control values were taken for each animal.

Results. The results presented for each series (Table), can be compared only with other data in that series. In series I when the total effect following bilateral occlusion of both carotid and vertebral systems is considered the systemic pressure (femoral) had a decline of 23.1% in mean pressure immediately after the RV occlusion, which was followed in 30 sec in all cases by a pronounced rise in pressure to a level approximately 33% higher than the

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Pressures of lingual arteries after clamping the carotid and vertebral arteries in sequence as % drop[a] from control and heart rate

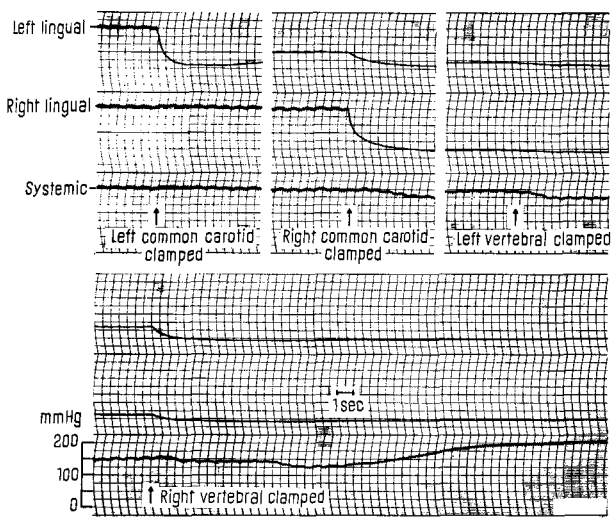
Artery clamped	Blood pressure recording						Heart rate	
	Left lingual Control 126/120 mm Hg		Right lingual Control 127/118 mm Hg		Systemic Control 126/115 mm Hg		Control 370/min	
	Initial	Recovery	Initial	Recovery	Initial	Recovery	Initial	Recovery
Series I								
LCC	− 74.1	− 68.2	− 8.2	− 6.4	− 9.8	− 6.4	365	368
RCC	− 77.0	− 75.3	− 80.4	− 78.8	− 14.8	− 9.5	370	360
LV	− 77.6	− 78.1	− 80.6	− 80.8	− 16.5	− 10.5	358	368
RV	− 83.5	− 80.2	− 86.8	− 82.6	− 23.1	+ 33.3	343	348
Series II								
RV	− 5.4	− 5.0	− 5.3	− 4.0	− 3.8	− 3.3	401	408
LV	− 16.3	− 15.5	− 15.6	− 15.0	− 15.6	− 14.8	406	406
RCC	− 18.1	− 15.2	− 70.3	− 64.6	− 16.6	− 12.4	406	400
LCC	− 82.0	− 80.7	− 84.5	− 83.7	− 7.4	+ 32.3	405	413

[a] In order to obtain the % drop the arithmetical average of the systolic and diastolic pressures at each stage of the procedure was taken, and the residual pressure after each occlusion calculated as 1% of the control. The difference between this figure and 100 is the *percentage drop* in pressure.

control values before the clamping was started. Actually there was a 56% rise from the lowest point (Table, Figure), all of which occurred only when the other 3 arteries were already closed. No increase in systemic pressure was noted after clampings of the common carotid arteries.

In series I the effect of clamping the ipsilateral common carotid artery on pressure at the lingual sites was approximately equal, the falls being 80.4% and 74.1% for the right and left sides respectively. These decreases were followed by slight recoveries within 40 sec. Successive clampings of the other arteries produced relatively small decreases. The final pressures after the last clamping showed a fall of 83.5% in the left lingual and 86.8% in the right. In series II the results were similar but the decline in pressure tended to be less than in the first series. The heart rate slowed slightly during the series I clampings, but at the beginning of series II was faster than that at the beginning of series I. This increase was maintained throughout series II.

Discussion. The lingual artery pressure, under the circumstances described in this study, has been shown by AYALA and HIMWICH[3,4] to reflect in a qualitative but not exactly quantitative fashion the pressures existing in a branch of the middle cerebral artery. It can, therefore, be assumed that the reductions in pressure noted were accompanied by similar ones on the arterial side of the cerebral circulation. The work of JILEK et al.[1] and also of KRIEGER[5] has shown that the rat is very susceptible to clamping of the carotid arteries. In the adult animal the ligation of both carotids for a period of 4 h results in marked pathological changes in the brainstem. On the basis of these data and the anatomical picture of the circle of Willis in the rat, which is complete apparently only by roundabout connections and not by direct communication as in the dog[6], it appeared to us that the vertebral arteries might make little contribution to the cerebral circulation in the rat. Although it is difficult on the basis of pressure measurements alone to determine the relative contribution which vessels make to the network at the base of the brain and hence to the total cerebral circulation, the data presented in this paper would suggest that in this species the vertebrals can make a considerable contribution. The failure of systemic blood pressure to respond to the clamping of the carotid arteries was probably due to complete denervation of the carotid sinus receptors. The final clamping of the 2nd vertebral artery, however, was followed not immediately but within 30 sec by a large increase in systemic pressure. This increase occurred in all animals studied, and suggested a central reflex mechanism which attempted to compensate for cerebral ischemia. The presence of such a mechanism in the rat has been suggested by KRIEGER[5].



Left and right lingual artery pressures and systemic blood pressures after the sequential closing of the common carotid arteries and the vertebral arteries.

Résumé. Les changements de tension artérielle dans l'artère linguale des Rats ont été mesurés après l'occlusion des artères carotidiennes et vertébrales. Les données obtenues nous font supposer que les artères vertébrales sont en grande partie responsables du maintient de la tension artérielle dans le cerveau.

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