CIRCULATION DURING CONTROLLED LOCOMOTION IN THE MESENCEPHALIC CAT

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During controlled locomotion of a mesencephalic cat, the cardiac output increases on the average by 70-95%, the arterial pressure rises by 20-35%, and the heart rate increases by 15-20%. The total vascular conductance increases mainly on account of dilatation of vessels of the working limbs through the action of a local factor and to an increase in the conductance of the remainder of the vascular system evoked by afferent influences from the working limbs.

Hemodynamic changes during brief muscular activity have been adequately studied [5-7]. They may be due to a change in the state of the vasomotor centers, to reflexes from working muscles, to dilatation of their vessels by the action of a local factor (changes in local metabolism, effects of intramuscular pressure on the tone of the musculature of the vessels [2]) and to the movement of metabolites from the working muscles into the general blood stream, where they can exert a direct or reflex action on the nervous centers.

Hemodynamic changes arising during controlled locomotion in a mesencephalic cat are described in this paper [3, 4].

EXPERIMENTAL METHOD

Under ether anesthesia and after tracheotomy, a thermoelectric transducer [1, 8] was introduced through one of the carotid arteries into the aorta to record cardiac output (F) by the thermodilution method [9]. A catheter for injecting cold Ringer's solution was introduced via the jugular vein into the inferior vena cava toward the atrium. The catheter for recording the pressure (P) (by an external tensometric manometer) was introduced into the left brachial artery; the left brachial plexus was divided. After precollicular decerebration [4], the anesthesia was stopped, the cat's head was fixed in a stereotaxic holder, and the limbs were placed on the belt of a treadmill. To produce locomotion, the "locomotor area" (LA) in the mesencephalon was stimulated [3]. The duration of each walk was about 1 min and intervals between them at least 5 min. From the recorded values of P, F, and the heart rate (f), the peripheral resistance ($R = P/f$), the conductance ($I = 1/R$), and the stroke volume (V) were calculated. The venous pressure was ignored.

Three series of experiments, with 6, 4, and 3 tested respectively, were carried out. The strength of stimulation to evoke locomotion was less then 200 μA.

EXPERIMENTAL RESULTS

Series I. The pressure P increased after walking for 5-7 sec, but then fell slightly and remained at a constant level (Fig. 1). After the 30th-50th sec of walking, a marked increase in F and a small increase in P and

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TABLE 1. Mean Values of Hemodynamic Parameters for Different Series of Experiments

Hemodynamic parameters	Series of experiments	Rest before walking	Walking	Rest after injection of curare	Stimulation of LA under curare	Rest after denervation of all limbs	Stimulation of LA after denervation of all limbs	Stimulation of peripheral ends of divided sciatic nerve	Rest after compression of abdominal aorta	Walking during compression of abdominal aorta
P (in mm Hg)	I	98	112	87	96	—	—	—	—	—
	II	89	119	—	—	92	127	84	—	—
	III	91	130	—	—	—	—	—	137	181
F (in ml/sec)	I	7,8	13,5	6,8	7,6	—	—	—	—	—
	II	7,3	12,4	—	—	6,4	7,9	7,9	—	—
	III	7,2	16,3	—	—	—	—	—	7,8	12,8
f (beats/sec)	I	3,5	4,0	3,9	3,9	—	—	—	—	—
	II	3,8	4,4	—	—	3,5	3,5	3,6	—	—
	III	3,3	4,0	—	—	—	—	—	3,2	3,6
R (mm Hg/ml/sec)	I	14,3	9,2	16,0	18,7	—	—	—	—	—
	II	12,7	7,7	—	—	15,0	19,2	10,8	—	—
	III	15,9	8,3	—	—	—	—	—	17,8	13,7
V (in ml)	I	2,2	3,4	1,7	1,9	—	—	—	—	—
	II	2,0	3,0	—	—	1,9	2,1	2,2	—	—
	III	2,3	4,2	—	—	—	—	—	2,6	3,7

[1]Rest after compression of aorta: P=115; F=8.2; *f*=3.3; R=14.3; V=2.5.

[2]Rest after walking with aorta compressed: P=118; F=7.8; *f*=3.4; R=16.1; V=2.5.

f were observed (Table 1). The increase in F was mainly due to an increase in V: R decreased by 30-40%. After curarization (1-2 mg tubocurarine or 8 mg flaxedil per cat, intravenously) stimulation of LA caused only a slight increase in P and a corresponding increase in R. Consequently, it was the locomotion and not the stimulation of LA itself which was responsible for the decrease in R and the increase in F, V, and *f*.

Series II. In these experiments, the right brachial plexus also was divided, the right brachial artery was ligated, and the sciatic, femoral, and obturator nerves were dissected. After denervation of all the limbs, stimulation of LA evoked the same increase in P as during walking, but only a small increase in F; *f* was unchanged. R increased more strongly than in response to stimulation of LA under curare. Since these two methods of immobilization differ in the fact that after division of the nerves the limb vessels also are denervated, it can be postulated that stimulation of LA itself dilates the vessels of the limb muscles to some extent and constricts the rest. The overall effect consists of a small increase in R.

During alternate (in the rhythm of walking) stimulation of the peripheral ends of the divided left and right sciatic nerves (with volleys of pulses of adequate strength, 0.3-0.5 sec in duration, pulse duration 0.1-0.3 msec, frequency 30/sec) for 1 min, after the 30th-50th sec of stimulation F increased but much less so than during locomotion, and no increase was observed in P and *f*. The increase in F and decrease in R in the absence of afferent impulses from the working limbs and of LA stimulation were evidently due to vasodilatation in the working muscles under the influence of a local factor (or of the entry of metabolites into the general blood stream). As mentioned above, the decrease in R and increase in F and *f* during walking were not connected with the stimulation of LA itself, and were more marked than during muscular work resulting from direct stimulation of the nerves. Consequently, afferent impulses from the working limbs play an essential role in the decrease in R and increase in F and *f*.

Series III. Both forelimbs were denervated and both brachial arteries ligated. The abdominal aorta was dissected above its bifurcation into the iliac arteries. Movements of the hind limbs during walking with a normal blood supply and during walking with the abdominal aorta compressed were compared (using

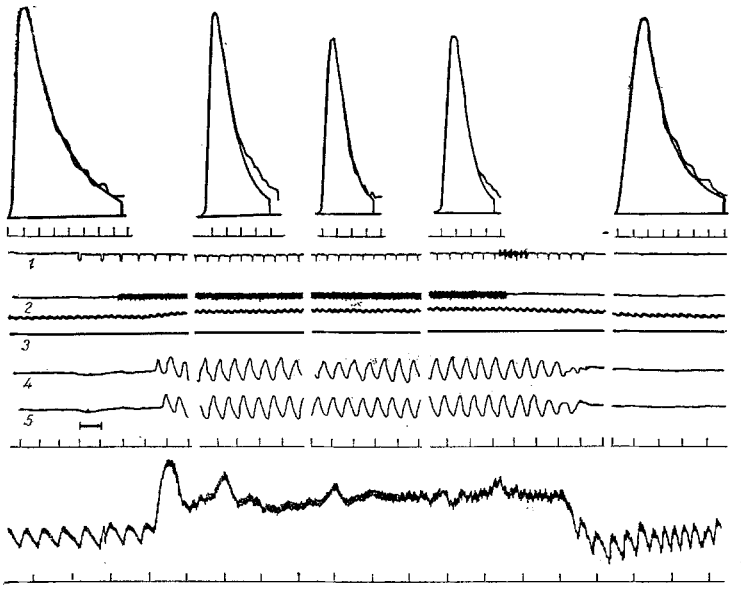

Fig. 1. Hemodynamic changes during controlled locomotion in a mesencephalic cat. From top to bottom: arterial pressure, longitudinal movements of right and left hind limbs, pulse rate, marker of LA stimulation, movement of treadmill belt (between adjacent marks the belt moves 0.5 m), thermodilution curves (6.8, 11.1, 15.0, 14.0, and 7.8 ml/sec). Time markers for records of arterial pressure, limb movements, and thermodilution curves are 5, 1, and 2 sec respectively.

values of the hemodynamic parameters 30-50 sec after compression of the aorta at rest for reference). The increase in P during walking with a normal blood supply was the same as during walking after compression of the aorta, but the increase in F and f was almost twice as great. During walking with the aorta compressed, R fell by almost twice as much as during walking with a normal blood supply.

Consequently, the increase in P during locomotion was unconnected with entry of metabolites into the general blood stream. It is assumed that in the case of locomotion for 1 min this factor can be disregarded by comparison with the rest of the hemodynamic parameters. The experiments with compression of the aorta also showed that the increase in total vascular conductance during locomotion is due approximately equally to an increase in vascular conductance in the working limbs and in the rest of the body. Exclusion of vessels of the working limbs may account for the smaller increase in F during locomotion with the aorta compressed. Since stimulation of LA by itself is unaccompanied by a perceptible increase in f, afferent impulses from the working limbs may play an essential role in its increase during locomotion.

During locomotion with the aorta compressed, a decrease in R can take place only through an increase in conductance of vessels outside the limbs. Since stimulation of LA itself can only constrict these vessels, and the action of metabolites from the working muscles in this case was excluded, it is evident that afferent impulses from the working limbs increased vascular conductance outside the limbs more strongly than it was reduced by stimulation of LA itself.

Comparison of the results of all series of experiments suggests some additional conclusions, although their reliability is problematical. Accepting that the effect of metabolites entering the general blood stream can be disregarded, it can be concluded that during locomotion the vascular conductance of the limbs increases several times, but that of the remaining vessels by less than 50%. The proportion of the total conductance due to vessels of the limbs is about 1/9 at rest and about 1/3 during walking. The contribution of LA stimulation to vasodilatation in the limbs was several times less than that of the local factor. Afferent impulses from the working limbs had no appreciable effect on their own vessels, but substantially increased conductance of vessels outside the limbs.

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