Responses to thalamic stimuli were more susceptible than those to stimulation of the pericruciate cortex, and responses to nigral stimulation were usually least affected. The observation that responses to stimulation of the CN are more sensitive to GABA and picrotoxin than are responses to stimulation of major afferent pathways suggests that these agents act preferentially on the output of caudate interneurons and not on the input to these neurons from major afferents. This action was clear in spite of factors which could bias results in the opposite direction, namely: a) while direct stimulation of the CN principally excites interneurons, it also excites some afferent fibers traversing the CN, and b) stimulation of afferent pathways secondarily activates some interneurons and may account for the weaker effects of GABA and picrotoxin seen in potentials evoked by stimulation of the afferent pathways.

The effects of picrotoxin further support the concept that intrinsic GABA is involved in the production of the response to stimulation of caudate interneurons. Picrotoxin antagonized the effect of extrinsic GABA on the evoked potential, as would be expected of an antagonist of any agent affecting the evoked potential. More importantly, picrotoxin alone antagonized and inverted those components of the evoked potential which were facilitated by extrinsic GABA; the inversion is probably due to components of opposite polarity normally masked by the GABA-sensitive components. The block of GABA-sensitive components by picrotoxin makes it likely that picrotoxin interacts with an intrinsic GABA receptor which is responsible for the production of the late components. Because these components were elicited only by strong stimuli and had a longer latency than the early peak produced by weak stimuli, they are presumed to be due to the excitation either of interneurons of relatively high threshold and slow conduction velocity or of polysynaptic interneuronal connections. While GABA may thus be the synaptic transmitter of interneurons of this description, another transmitter is probably liberated by a set of interneurons with a low threshold producing the early peak in the evoked potential. The GABA receptor is probably at or near the soma of interneurons, because picrotoxin altered evoked potentials only when applied at a site where neuronal action potentials could also be recorded. This is consistent with electronmicroscopic evidence showing that afferent fibers terminate on dendrites and somata of interneurons while terminals of interneurons have synapses on the initial segment as well[18].

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The effect of vasoactive agents on nutritive collateral circulation in rat muscles

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Summary. The relationship between the collateral and control nutritive blood flow (^{86}Rb) of the triceps muscle was not influenced by vasoactive agents (phenylephrine, angiotensin II, vasopressin) in the rat. Isolevine caused vasodilation only in the control muscle.

Immediately following ligation of an artery, the oxygen and metabolic supply of the area fed by that artery is determined by vessels called primary nutritive collaterals. Both from physiological and clinical points of view, it is important to know the sensitivity and reactivity of the nutritive collaterals. In our experiments, the effect of vasoactive agents on the accumulation of ^{86}Rb by m. triceps surae, following closure of the femoral artery, has been studied.

Methods. Male rats after 12–16 h of fasting were anesthetized with 50 mg/kg b.wt pentobarbital i.p. Arterial blood pressure was continuously recorded from the carotid artery with a mercury manometer. Cardiac output was determined with the Evans blue dilution method, and the m. triceps surae fraction of the cardiac output was measured using the isotope fractionation method of Sapirstein[1]. Circulatory resistance was calculated. Nutritive collateral resistance refers to resistance of the whole area accumulating Rb and supplied by the collaterals. Since we do not know the pressure drop inside individual vessel segments, partial resistances cannot be calculated from our data. Details of these methods are found in our previous papers[2,3].

The left femoral artery was ligated under the inguinal ligament. In previous experiments[2,3], it was found that the resistance of the collaterals of the m. triceps surae does not change during the first 10–120 min following ligation. Therefore, we started the infusion of drugs into the jugular vein 10 min after ligation. The infusion lasted 5 min, at a rate of 0.02 ml/min, after which the cardiac output was determined by sampling blood from the carotid artery; then about 10 μCi of ^{86}Rb was injected into the jugular vein. At 60–80 sec after Rb injection, the animals were killed by i.v. administration of KCl solution. The cumulative doses of the vasoactive agents for 100 g b.wt were as follows: vasopressin: 16 and 32 mU; angiotensin II: 0.10 and 0.20 μg; phenylephrine: 9.2 and 19.4 μg; isolevine: 0.11 and 0.19 μg. Cardiac output and TPR were calculated for 100 g b.wt, blood flow and resistance of the triceps muscle for 100 g muscle weight. Changes in the experimental animals were compared to those in the non-treated control group. Data are shown in the table with common standard deviations.

Results and comments. As illustrated in the table, the nutritive blood flow to the left triceps muscle decreased to 55% and its resistance increased to 206% of the contralateral side following ligation of the left femoral artery. Both concentrations of vasopressin, angiotensin II and phenylephrine increased the blood pressure and increased the resistance of the triceps muscle on both the ligated

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		Vasopressin		Angiotensin II		Phenylephrine		Isolevine		Common SD
	Control	16 mU	32 mU	0.10 μg	0.20 μg	9.2 μg	19.4 μg	0.11 μg	0.19 μg	
n	20	11	11	10	10	10	10	9	9	
Total body										
Body weight	198.5	202.3	205.4	192.0	195.5	218.0	203.5	185.0	207.8	34.8
Blood pressure	123.2	152.3[b]	160.1[b]	142.0[b]	152.5[b,c]	173.0[b]	172.5[b]	93.9[b]	77.8[b]	18.1
Cardiac index	20.6	17.8	18.7	16.3	13.6[b]	18.8	17.2[b]	23.1	27.4[b]	4.8
TPR	503.2	713.7[b]	730.2[b]	736.7[b]	961.0[b]	749.0[b]	805.5[b]	338.1[b]	227.0[b,d]	150.7
M. triceps surae										
Blood flow l	2.89	2.77	2.45	2.34[a]	1.85[b,c]	3.05	2.31	2.21	1.54[b]	0.85
Blood flow r	5.64	4.78	4.93	4.66	3.49[b]	5.00	3.61[b,c]	6.72	16.42[b,c]	3.42
left/right; percent	55.2	59.3	51.6	53.9	54.3	60.8	63.5	39.8[a]	11.0[b,d]	15.4
Resistance l	3722	5133[a]	5757[b]	5021[a]	7634[b]	5061[a]	6628[b]	3753	6127	2340
Resistance r	1930	2819[b]	2762[b]	2741[b]	3776[b,c]	2891[b]	4127[b,c]	1508	483[b,c]	904

Body weight: g; blood pressure: mm Hg; cardiac index: ml/min 100 g b.wt; TPR: 10^3 cm dyn sec^{-5}/100 g b.wt; blood flow: ml/min 100 g weight of m. triceps; resistance: 10^3 cm dyn sec^{-5}/100 g weight of m. triceps. l = left, r = right.
Differences. Control versus treated: [a]$p < 0.05$, [b]$p < 0.01$; treated versus treated: [c]$p < 0.05$, [d]$p < 0.01$.

and contralateral sides. Except for the larger dose of phenylephrine, these drugs did not change the blood flow and resistance on the 2 sides relative to each other.

Isolevine, especially the larger dose, significantly increased the cardiac index and decreased the TPR. The blood flow of the triceps muscle increased, while its resistance decreased compared to the control non-treated group. On the ligated side, these changes were opposite in direction. Thus, the ratio of blood flows on the 2 sides decreased and that for their resistances increased depending on the dose.

In our experiments the relationship between the ligated and control sides for nutritive blood flow and resistance did not change following administration of vasoconstrictor agents. Thus, the 'relative' sensitivity of nutritive collaterals in the muscle to vasoconstrictor agents can be regarded as similar to that of the contralateral, non-ligated side. On the contrary, isolevine caused dilatation of the muscle vessels on the control side while on the ligated side – probably due to a decrease in perfusion pressure – it resulted in a decrease in blood flow and an increase in resistance. In respect of total blood flow, there are published data reporting decreased sensitivity of muscle collateral vessels to vasodilating agents[4]. Similar observations were made for cardiac muscle[5]. On the basis of our experiments, it can be stated that the decreased reactivity is valid for the nutritive collaterals of the striated muscle of rats as well.

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Amphetamine induced turning behavior as an index of stroke in the mongolian gerbil

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Summary. The effects of i.p. injections of amphetamine and apomorphine were determined before and after unilateral ligation of the carotid artery in the gerbil. Significant increases in turning behavior were observed in the absence of neurohistological evidence of infarction.

Dopaminergic drugs induce turning behavior in animals that have been subjected to unilateral lesions of the nigrostriatal system[1]. Amphetamine which causes the release of (DA) gives rise to turning towards the lesioned side (ipsilateral) and apomorphine, a DA receptor stimulant, induces turning away from the lesion (contralateral). The apomorphine effect may be due to denervation supersensitivity of DA receptors on the lesioned side and only develops after a delay. The initial effects of apomorphine, like amphetamine, cause ipsilateral turning due to the more active dopaminergic system in the unlesioned side. These drugs in higher doses also produce turning behavior in some normal animals, implying an inherent imbalance in DA systems[2].

Uniquely, in the case of the Mongolian gerbil (Meriones unguiculatus) ligation of the carotid artery on one side will usually induce ischaemia in only that hemisphere[3]. This is due to the absence of significant connections between the basilar and carotid circulation in this species. Unilateral carotid ligation is fatal in a proportion of gerbils but at least 50% recover with no visible functional deficit and no histological signs of cerebral infarction[4]. In order to test the residual effects of this procedure in

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