The present results show that the effects of a specific NA-releasing tract can be modulated by the amount of circulating thyroid hormone, and reinforces the contention that this is one of the effects of this hormone in the CNS. At present it is not possible to decide if this effect is presynaptic, as has been proposed for the actions of TRH[6], or postsynaptic. However, iontophoretic application of NA onto PC (to be reported elsewhere), gave similar results to those described for brain stem cells[1], which suggests a probable postsynaptic site of action.

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Microsphere measurement of myocardial capillary bed in hypoxic rats

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Summary. Rats exposed to hypobaric hypoxia for 24 h showed a significant increase in the number of perfused capillaries and in the number of radioactive microspheres trapped in the coronary circulation.

It has been well established that the heart undergoes hypertrophy in mammals subjected to chronic hypoxia. In an attempt to clarify cellular events that occur in the heart during the early stages of exposure, previous work in our laboratory has shown that young adult rats exposed to a moderately severe level of hypobaric hypoxia (380 torr) during a 24-h sojourn in a decompression chamber exhibited a statistically significant increase in heart weight when compared with rats maintained at ambient pressure (725 torr)[1,2]. Ventricular tissue from hypoxia exposed rats weighed more than ventricular tissue from control animals on both an absolute and a relative (mg heart/100 g b.wt) basis. Body weight, on the other hand, decreased precipitously during 24 h of hypoxia exposure (mean: −33 g/rat) while control animals gained about 6 g per rat during the same time period. Cytophotometric measurements of RNA in cardiac myocytes showed slightly lower values for azure B-stained RNA in the cytoplasm of heart muscle cells from 1-day hypoxia exposed rats when compared with corresponding controls, an indication that augmented protein synthesis is probably not a factor in the observed increase in heart weight during the 1st day of hypoxia exposure[2]. Histological studies also revealed a significant increase in the number of capillaries per unit area of ventricular myocardium in heart sections from hypoxic rats[3].

Summary of experimental data

Parameter	24-h Hypoxia exposed (N = 12)	Control (N = 14)
Body weight		
at autopsy	295 ± 4 g*	323 ± 12 g
initial	328 ± 5 g	319 ± 13 g
change	−33 ± 2 g	+4 ± 2 g
Heart weight		
actual	807 ± 17 mg	805 ± 24 mg
mg heart/100 g b.wt	273 ± 7 mg	250 ± 6 mg
Capillaries/mm^2	1380 ± 47	902 ± 35
^{153}Gd microspheres		
total cpm injected	27×10^4	39×10^4
cpm/heart ventricles	2.5×10^4	2.1×10^4
cpm/mg heart	33 ± 24 (NS)	25 ± 15
% recovery	9 ± 5 (NS)	5 ± 2

* All values are expressed as mean ± SEM. ** Value is percentage of injected counts recovered in the heart ventricles.

The present study was designed to utilize the radioactive microsphere technique in an attempt to obtain a more accurate assessment of the extent of coronary vascularization resulting from exposure to hypobaric hypoxia. The validity of the microsphere technique for obtaining data of this type has been well established[4].

Methods. Male Wistar albino rats were maintained continuously at 380 torr in a ventilated decompression chamber; controls were maintained at an ambient pressure of 725 torr. At the end of 24 h rats were anesthetized with pentobarbital (35 mg/kg, i.p.) and a polyethylene catheter was inserted through the right common carotid artery and into the left ventricle, using the procedure reported by Sasaki and Wagner[5]. Approximately 50,000 microspheres, 15.3 μm in diameter, labeled with ^{153}Gd (New England Nuclear) and suspended in 0.05 ml of 10% dextran plus 0.01% Tween 80, were injected into the left ventricle, followed immediately by 0.3–0.4 ml of heparinized physiological saline. Total injection time was 1 min. Microsphere suspension was contained in a silastic tube (7.5 cm long, 1 mm inner diameter) which was attached to end of implanted catheter. Total amount of radioactivity injected was calculated from the difference between initial amount and that remaining in silastic tube after injection and saline flush. Ventricles, right lung, both kidneys were excised, fixed in 10% neutral buffered formalin for 24 h, transferred to fresh formalin and gamma radioactivity was counted using a Beckman Biogamma II. Ventricles were processed by the paraffin method, 8-μm sections stained with hematoxylin and eosin were used for capillary counts. An ocular reticle engraved with 1-mm^2 was used to delimit areas to be counted; capillaries were counted only in regions where muscle fibres were cut in cross section.

Results and discussion. Table 1 summarizes experimental data. Body weight changes were almost identical to those found previously but, unlike earlier studies[1,2], heart weights showed little difference between hypoxia-exposed and control animals. Relative heart weight (mg heart/100 g b.wt) was only 9% greater in hypoxia-exposed rats. Capillary counts in the ventricular myocardium showed 53% more perfused capillaries in hypoxic hearts (i.e., a change from 902 to 1380 capillaries/mm^2). Data on the number of microspheres trapped in the myocardial capillaries suggest an enhancement of coronary circulation in hypoxia exposed rats. This is supported by the finding that total counts in ventricular tissue and the percentage of injected radioac-

tivity recovered in heart tissue tended to be higher in exposed than in control animals. However, due to the high variability in the extent of microsphere entrapment, values for the amount of radioactivity recovered in the heart did not prove to be statistically significant. Thus, it would appear that, in our hands, myocardial capillary counts are a more reliable indicator of an expansion of the capillary bed than the use of radioactive microspheres.

Less than 1% of total counts injected was recovered in the right lung, indicating that microspheres lodged in the systemic capillary beds during the first circuit, which validates previous reports that very few 15-μm-diameter spheres pass through the capillary beds and reach the venous return of the heart[5,6]. Right and left kidneys showed a virtually identical percentage recovery of radioactivity (i.e., 5.6 ± 0.5 and $5.9 \pm 0.5\%$, respectively, in hypoxic rats and $6.7 \pm 0.7\%$ in right and left kidneys in controls), which provides validation of the adequate mixing of microspheres in blood flowing through the aorta[6,7]. Most workers agree that the rheology of microspheres 15 μm, or less, in diameter is comparable to that of erythrocytes[6,7].

The overall findings corroborate our belief that one of the earliest cardiac responses to hypobaric hypoxia is a recruitment of existing capillaries in an attempt to provide adequate amounts of oxygen to hypoxia-stressed myocardial tissue. At first glance, a hypoxia-induced expansion of about 50% in the capillary bed would appear to be an exceptionally large response. However, it is noteworthy in this regard that several workers (using measures of capillarity and intercapillary distances in myocardial tissue) have estimated capillary reserves ranging from 25 to 80% as a normal characteristic of the non-taxed heart[8,9]. These findings also support the generally accepted tenet that, in case of oxygen lack, blood flow to the heart and brain is maintained at the expense of other circuits[10,11].

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Catalase activity in electrically stimulated muscle

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Summary. 10 min of electrical stimulation resulted in a significant rise in gastrocnemius catalase activity.

It is well-known that a continuous supply of oxygen is required for skeletal muscles to maintain prolonged contractions. It is now known that superoxide radicals are formed during the biological reduction of oxygen to water[1]. Active cells are protected against the harmful effects of these metabolites by superoxide dismutases which catalyze the conversion of superoxide ions into H_2O_2[2]. Catalase and other peroxidases scavenge the lethal peroxides and thus maintain a low steady state of H_2O_2 and return some oxygen to the cell[3]. Although there has been a plethora of research related to catalase, and though catalase has been shown to be present in skeletal muscle[4], there is a paucity of research related to this particular tissue. Furthermore, the literature relating the effects of exercise on catalase activity is both sparse and confusing. For instance, catalase activity has been reported to rise[5], fall[6] or remain unchanged[7] after acute exercise. Therefore, the present study was conducted in order to determine the effect of controlled muscle contraction on catalase activity.

Materials and methods. 6 male Sprague-Dawley rats were used. The rats were housed individually under standard conditions. The animals were anesthetized with ketamine hydrochloride (60 mg/kg) and maintained at a surgical level with ether. The skin overlying both gastrocnemius muscles was retracted and 1 muscle was impaled with stimulating electrodes, while the control muscle received electrodes but no stimulation. The tendon of the stimulated muscle was connected by a thin wire to a linear core, isotonic myograph. Care was taken to keep the muscles moist with mammalian Ringer solution. Body temperature was maintained by placing a lamp over the preparation. The muscle was stimulated with supramaximal square-wave pulses of 0.5 msec duration, at a frequency of 30 Hz. At the termination of a 10-min stimulation period, the rate was increased to 100 Hz to fatigue the muscle. After decapitation and exsanguination, the muscles were prepared for analysis in the cold. Catalase was analyzed by the O_2 cathode method of Goldstein[8]. The enzyme unit is the amount of enzyme that releases 1 μmole of oxygen per min at 30 °C, pH 7 and 0.033 M perborate.

Results and discussion. Electrical stimulation resulted in a 66% increase in catalase activity. At first we thought this rise in activity might be a result of direct stimulation of the tissue since electrical stimulation has been shown to produce gasification[9]. However, when muscles were stimulated through their nerves, the same effect was observed. Since our analysis technique was based on the rate of O_2 evolution, the observed difference might actually have resulted from varying O_2 utilization rates of the homogenates. We

	Stimulated	Control
Catalase (units/g of tissue)	464 ± 4*	280 ± 4
O_2 consumption (μl O_2/min · g of tissue)	320 ± 8	312 ± 6

The data are means ± SE. *Significant at $p < 0.05$ versus non-stimulated control muscle.