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.

compared the rates of O_2 consumption of stimulated and control homogenates and found them to be not significantly different. Therefore, we have not, as yet, determined what initiates this rise in catalase activity.

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Effects of inhibition and stimulation of Na^+-K^+ active transport on the resting membrane input conductance of the guinea-pig ventricle[1]

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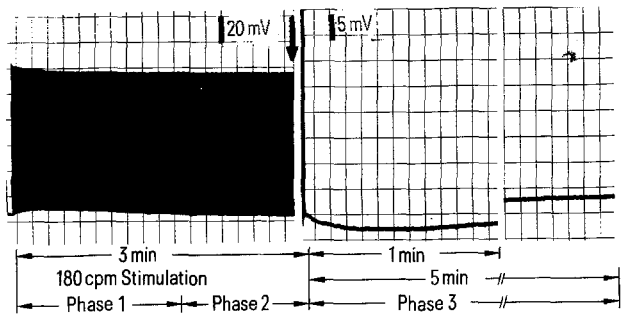
Summary. The effects of inhibition by ouabain and stimulation by high frequency drive of the sarcolemmal Na^+-K^+ active transport system on the resting input conductance (g_i) of guinea-pig ventricular muscles were determined. Although both pump inhibition and stimulation were associated with changes in electrophysiological properties of the muscles, neither had a significant effect on g_i.

The resting membrane potential of excitable cells is determined by the distribution of ions across the sarcolemma, the conductance of the membrane to these ions, and the net current produced by the Na^+-K^+ active transport system. It is generally accepted that Na^+ and K^+ active transport via the ouabain sensitive sarcolemmal Na^+-K^+ ATPase maintains the transmembrane concentration gradients for Na^+ and K^+ and that the Na^+/K^+ active transport coupling ratio and level of Na^+-K^+ ATPase activity determine the magnitude of the net current produced by the pump. Shanes[3] proposed that the Na^+-K^+ active transport system can also influence the bioelectric properties of a cell by preventing ions from penetrating the membrane and thereby influencing membrane conductance. This hypothesis has been supported by the results of experiments on frog skeletal muscle[4], cardiac Purkinje fibres[5], and crayfish giant axons[6]; inhibition of the pump by ouabain significantly increases the resting membrane conductance of these preparations while stimulation of Na^+-K^+ ATPase in the crayfish axon by papaverine produces a significant decrease in membrane conductance[7]. The purpose of this study was to determine the effects of inhibition and stimulation of the Na^+-K^+ active transport system on the resting input conductance of the ventricular myocardium.



Response of the transmembrane potential to 3 min of suprathreshold old stimulation at 180 cpm (Exp. No.121675). Note the changes in vertical scale and time base at the arrow.

Male guinea-pigs, weighing 200–400 gm, were stunned by a blow to the head and their hearts rapidly removed. A muscle strip was cut from the right ventricle and mounted in a bathing chamber through which a solution of the following composition flowed: 128 mM NaCl, 5.6 mM KCl, 2.5 mM $CaCl_2$, 1.2 mM $MgCl_2$, 10 mM Tris HCl, 2.2 mM Tris base, and 10 mM dextrose (pH = 7.41). Either gluconate or isethionate, 2 relatively impermeant anions, replaced 100% of the Cl^- in the Cl^--free media. The Ca^{++} activity of these solutions was titrated to control levels to compensate for Ca^{++} binding to the impermeant anions[8]. All media were saturated with 100% O_2 and maintained at 30 °C for the pump inhibition experiments and 37 °C for the pump stimulation experiments. One end of the muscle was attached to a fixed post while the other end was attached to an isometric strain gauge for recording mechanical activity. An isolated electronic stimulator excited the muscle strips through 2 platinum wires placed next to the preparation. The transmembrane potential was recorded from cells on the endocardial surface of the muscles by an electrometer through a glass microelectrode (15–20 M Ω resistance) filled with 2.5 M KCl. The electrical and mechanical signals were displayed on an oscilloscope and strip chart recorder for graphical analysis.

The input conductance (g_i) of the muscle was measured by the 2-electrode technique which has previously been described in detail[9]. A current-passing microelectrode (<3 M Ω resistance), positioned in the muscle within 30 µm of the voltage-recording electrode, injected a 30 msec duration square current pulse from an isolated stimulator. The magnitude of the current (averaging 8 µA) was chosen to produce a subthreshold steady-state voltage response of less than 8 mV at the recording electrode. g_i was calculated as the ratio of the injected current to the steady-state voltage change. Because cell geometry and the relative placement of the current and recording electrodes influence the value of g_i, data was accepted only from experiments in which the placement of the electrodes was maintained constant during the test and pre-test periods.

Partial inhibition of Na^+-K^+ active transport was produced by the addition of 1.2×10^{-6} M ouabain to the bathing media. This was the highest dose that could be consistently used without the induction of toxic side effects charac-