related to the time-interval between the 2 ligations; the greater the interval the smaller the difference between the right and the left sides of the brain. These results demonstrate an almost complete redistribution of blood flow between the right and the left hemispheres if sufficient time is allowed between the 2 ligations (4–5 days). Initially, there was a marked reduction in blood flow in those structures supplied by the carotids e.g. the cortex, striatum, hippocampus, mid-brain and hypothalamus. In contrast, there was an increased blood flow to the brain stem (table). The blood supply to the cerebellum was greatly reduced, but not as dramatically as in other areas of the brain; this suggests that the supply to the cerebellum is not entirely carotid-dependent.

Our results show the role of the carotid and vertebral arteries in supplying blood to the brain. In Long Evans rats the circle of Willis cannot ensure immediate redistribution of the blood flow to all areas of the brain after bilateral ligation of the carotid arteries. However, when a time interval is allowed between the clampings, an apparent redistribution occurs via the circle of Willis and probably via anastomoses of other arteries of the vertebrobasilar axis.

Two months after ligation of the carotids, the surviving rats still showed differences in the distribution of the cerebral blood flow. In particular, it was noted that animals surviving carotid ligatures made at an interval of 24 h had a low blood flow to the cortical areas but a good supply to the brain stem (fig. 3). A similar but less marked result was obtained for animals in which ligatures were made at an interval of 3 days. The distribution of blood flow in rats where the interval between ligatures was 5, 7 and 15 days was very similar to that found in control rats. It is suggested that in some animals rapid redistribution can occur, leading to a new equilibrium which is then maintained.

The results here show that in Long Evans rats the cerebral trunk is supplied by blood from the vertebral arteries. In some rats this system can compensate for carotid failure. However, it is necessary for a certain time to elapse before there is optimal function of the system.

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Aortic fibrous components in exercised rats

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Summary. In exercised female rats, the elastin content of the thoracic and abdominal aorta decreased by 4–8% ($p < 0.05$). The collagen content in the thoracic aorta, was unchanged but in the abdominal aorta was reduced by 5.2% ($p < 0.05$). These results are discussed in connection with physical training.

The metabolism of collagen and elastin in mammalian arteries is affected by various factors. Collagen production in rabbit aorta is enhanced by simultaneous administration of epinephrine and thyroxine[2] and by hypoxia[3]. In addition, collagen and/or elastin synthesis in the rat aorta and/or mesenteric artery is augmented by hypertension[4,5]. Thus, sympathetic activation and augmented secretory activity of the adrenal medulla during exercise appear to have an effect on the metabolism of arterial fibrous protein.

We estimated the amounts of collagen and elastin in rat aorta after forcing the rats to run on a treadmill, and the role of physical exercise in the metabolism of these components was considered.

5-week-old female Wistar rats weighing approximately 100 g before training were housed in 2 groups (C and R) and maintained on laboratory chow (Oriental Yeast Co. MF., Japan) and water ad libitum. The room temperature was kept at 19±1 °C. Group C (27 animals) served as the control (sedentary rats), while group R (24 animals) was forced to run on a treadmill. In the initial stage of the experiment, the rats ran at 10–26 m/min, on a slope of 10 °C for 1 h/day, 6 days/week. After 4 weeks, the same rats were made to run continuously at 30 m/min for 1 h/day. The entire experiment lasted 12 weeks. The animals were decapitated 24 h after the last exercise and the thoracic and abdominal aortae were immediately removed and perfused with a chilled solution of physiological saline (4 °C, pH 7.2–7.4). After blotting to remove the adherent fluid, the aortae were frozen in dry ice-acetone and stored at −80 °C until assay. The aortic materials were dried for 48 h at 100 °C and then immersed in acetone for 24 h and in ether for 18 h. Subsequently, they were dried for 24 h at 100 °C to prepare the dry defatted tissue. The separation of collagen and elastin in the dry defatted tissue and hydroly-

Effects of daily forced treadmill running on the contents of collagen and elastin in the rat aorta

	Collagen (%)	Elastin (%)	
Thoracic aorta			
C	28.6±0.7	54.9±0.6	n=27
R	28.7±0.4	52.4±0.6*	n=24
Abdominal aorta			
C	42.2±0.7	38.1±0.4	n=27
R	40.1±0.7*	35.4±0.5*	n=24

C, sedentary rats; R, trained rats. Average of 24–27 tests. ±SE. *Statistical significance ($p < 0.05$).

sis to hydroxyproline in the fibrous proteins were carried out by the method of Fischer and Llaurado[6]. These samples, containing hydroxyproline, were adjusted to pH 6-7 and hydroxyproline was estimated by the method of Woessner[7]. The hydroxyproline content thus obtained was multiplied by a factor of 7.46 for collagen estimation and by a factor of 43.4 for elastin determination[8]. These estimated values for collagen and elastin were expressed in terms of percentages of the dry defatted weight of the aorta. The statistical significance of the differences between the 2 groups (C and R) was determined by Student's t-test.

The results are summarized in the table. Percentages of the dry defatted weight represented by these components varied in different areas of the aorta. With respect to sedentary rats, the percentage of collagen was 28.6% in the thoracic aorta and 42.2% in the abdominal aorta, while that of elastin amounted to 54.9% in the thoracic aorta and 38.1% in the abdominal aorta. These control values for collagen and elastin were comparable with those found in the ascending and the abdominal aorta in mongrel dogs, as reported by Fischer and Llaurado[6]. After forced running, the estimated values for collagen were unchanged in the thoracic aorta, while the amount of this fibrous protein in the abdominal aorta decreased by 5.2% of the control ($p < 0.05$). In this case, elastin in the thoracic and abdominal aorta was also reduced by 4-8% ($p < 0.05$).

Reduction of collagen in the abdominal aorta indicates that the distal area of the aorta is more distensible in the trained animals, at a high transmural pressure in the aortic region, because collagen is stiff and resists further stretch. We also noted a decrease in elastin in the thoracic and the abdominal aorta after training. As elastin, unlike collagen, stretches freely to accomodate a given blood pressure, free energy developed in this elastic component in the process of the aortic expansion is probably released during diastole in order to facilitate blood flow against the resistance of circulation. Accordingly, the decrease in the aortic elastin may indicate that the diastolic flow in the trained animals cannot be enhanced during exercise, unless the peripheral vasoconstriction is less pronounced.

In studies on trained rats, Harri[9] found an increased sensitivity of $beta_2$-adrenoceptors and/or decreased sensitivity of alpha-adrenoceptors in the peripheral vessels. This would suggest that in the trained animals, sympathetic activation during exercise augments vasodilation and accelerates the diastolic flow with less free energy expenditure of the aortic elastin. It has been shown for rabbits that the circulation in the capillary beds of skeletal muscle increased as a result of training[10], thereby suggesting an elevated vasodilation of arterioles in the muscle during exercise. Moreover, resting diastolic pressure in healthy men was reduced after training, and was ascribed to a decrease in the peripheral vascular resistance[11].

In our experiments, we detected no increase in the aortic fibrous proteins after exercise, this being in contrast to the overproduction of collagen and/or elastin induced by epinephrine and thyroxine[2], by hypoxia[3] and by hypertension[4,5].

Bassler[12] speculated that marathon runners may have an immunity to fatal atherosclerosis, on the bases of overproduction of collagen and of accumulation of lipid materials. As described above, we used only female rats in the running exercise. Estrogen is a known antiatherosclerotic agent, as explained by Kishi and Numano[13] and suppresses the reduction of cyclic AMP levels in blood vessels and inhibits vascular permeability to protect the walls from lipid intrusion. Thus, estrogen must be considered in evaluating such protective effects of exercise. However, as trained female subjects often have irregular menstruation[14], the question arises as to whether estrogen activity would be stimulated by physical training. When taking into account overproduction of collagen (collagen α B chain)[15] in an early stage of atherosclerotic plaques and considering a reduction of collagen in the distal area of the aorta in the trained rats, daily running for a long period seems to inhibit the proliferation of this stiff component in the arteries. Further studies are warranted on the effect of physical training with regard to metabolism of lipid and connective tissue, including collagen and elastin, in peripheral blood vessels.

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Strain differences in responses of the circadian system to light in the Syrian hamster[1]

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Summary. The circadian systems of 2 strains of the Syrian hamster responded differently to single short light pulses. The differences in the amplitudes of the phase response curves were associated with different ranges of entrainment of the circadian rhythms to periodic light pulses.

The Syrian hamster *(Mesocricetus auratus)* has been one of the most extensively used mammalian species in the research of daily (circadian) and annual periodicities[2-8]. Valuable models have been proposed that describe ex-