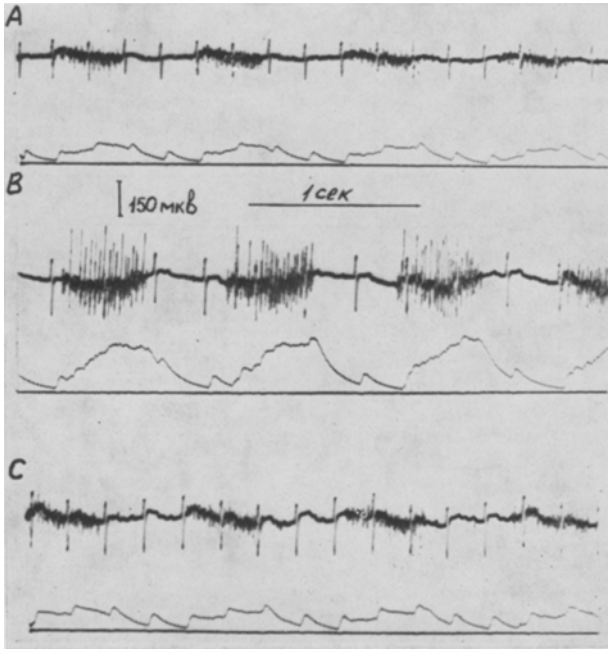
Table II. The influence of artificial ventilation on pH and Pa CO_2 in the phrenicotomized rabbits

	During the artificial ventilation which coincides with the respiratory frequency and tidal volume of the spontaneous breathing				The increase of the volume of the pump which was need to normalize pH and Pa CO_2			
	Volume of the pump (ml)	Frequency of the pump	pH	Pa CO_2 (mm Hg)	Volume of the pump (ml)	Frequency of the pump	pH	Pa CO_2 (mm Hg)
Mean	12.0	46.0	7.24	48.5	22.5	46.0	7.28	38.0
SD			±0.04	±5.6			±0.01	±3.5
$n = 10$								

See the glossary of abreviations in Table I.



The electrical activity from intercostal inspiratory muscles (the thin lines – integral activity of the same muscle). A) while the frequency and the volume of the respiratory pump coincides with the spontaneous breathing. Before phrenicotomy. B) the size of the artificial respiration remains the same as in A. After phrenicotomy strong increase of the electrical activity. C) while the volume of the respiratory pump was increased from 10 ml to 20 ml. The electrical activity is normalized.

basis of nomograms. Perhaps their demand depends on the decrease of the lung compliance and alveolar hypoventilation evoked by paralysis of the respiratory muscles.

2. There are some attempts to prevent breathlessness by means of a block of the phrenic nerves[2]. As is stressed by the authors there was only a partial diaphragmatic paralysis after the block. It may be thought that in the case of total paralysis of the diaphragm, this method may evoke a quite contrary result.

ВЫВОДЫ. Несмотря на искусственное сохранение нормальной лёгочной вентиляции, френикотомия вызывает у кроликов резкое увеличение напряжения CO_2 в крови. Для восстановления нормального напряжения CO_2 необходимо значительно увеличить лёгочную вентиляцию. Этим повидимому объясняется то, что больные с параличом дыхательных мышц требуют увеличения лёгочной вентиляции, значительно превосходящей нормальную.

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Age-Dependent Changes in (Na, K)-ATPase Activity in Brains of Mice Susceptible to Seizures

Mice of the DBA strain, susceptible to audiogenic seizures, have convulsions at the age of 30 days when they are exposed to heat, whereas mice of the C57 strain do not[1]. Since DBA mice do not have convulsions with elevated temperature at 20 or at 40 days, they seem to be a model of febrile convulsions in humans[1], and interest attaches to features that distinguish them from the seizure-resistant C57 strain. Curiously enough the findings of Abood and Gerard[2] have never been confirmed that brains of DBA mice have lower ATPase and a lower P/O ratio than those of C57 mice at the age of seizure-susceptibility but not before or after. The authors could not then differentiate the form of ATPase; it is now known that ATPase activity in brain is largely in the form activated by Na^+ and K^+, i.e. (Na, K)-ATPase[3].

For this reason I studied ATPase activity in microsomal membranes of brains of DBA mice at the age of susceptibility; 30 days; and before and after, at 20 and 60 days. The results were compared with those of non-

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ATPase activity of brain microsomes from DBA and C57 mice

Strain	Age (days)	(Na, K)-ATPase activity (μmole/h/mg protein)	P	Total ATPase activity (μmol/h/mg protein)	P
DBA	30	8.9 ± 0.8		19.1 ± 1.8	
C57	30	18.5 ± 1.2	< 0.001	27.5 ± 2.6	< 0.001
DBA	60	18.4 ± 0.8		27.6 ± 1.2	
C57	60	17.9 ± 2.8	N.S.	26.8 ± 0.9	N.S.

Mean values of 5–7 individual experiments are ± S.E. of the mean. P gives the significance of the difference between DBA and C57 mice. N.S., not significant.

susceptible C57 mice. The animals had never had a convulsion and had never been subjected to loud sound or to heat.

In brains of 30-day-old animals, activity of (Na, K)-ATPase in DBA mice was half that in mice of the C57 strain and total ATPase was less by a third (Table). At 20 days, (Na,K)-ATPase activity was nearly the same in the two strains, and there was no difference at the age of 60 days. There was no difference between the strains in Mg-ATPase.

The method of assay is described in full[1]. Briefly, whole hemispheres were homogenized in isotonic sucrose containing 5.0 mM phosphate buffer (pH 6.0) and 1.0 mM EDTA. The supernatant after centrifugation at 10,000 g for 20 min, was recentrifuged at 100,000 g for 30 min and the sedimented microsomal membranes were washed and suspended in distilled water. (Na, K)-ATPase activity was determined as total ATPase activity minus ouabain-insensitive Mg-ATPase activity. The former was measured in an incubation medium containing 1.0 mM $MgCl_2$, 120 mM NaCl, 20 mM KCl and 30.0 mM histidine, HCl buffer (pH 7.0), and the latter in a corresponding medium prepared without NaCl and KCl and containing 1.0 mM ouabain. 10 μg of microsomal membrane protein was preincubated (15 min, 30 °C), when (γ-P^{32})ATP (specific activity 950 mCi/mmole) was added to give a final substrate concentration of 1.0 mM. Inorganic P^{32} was extracted as previously described[4], and radioactivity counted by liquid scintillation. The specific enzyme activity is expressed as μmole of ATP hydrolyzed/h/mg protein.

Other neurochemical findings in brains of the same animals[1] may well be due to the deficit in (Na, K)-ATPase: lower concentration of K^+, of K^+-activated release of GABA, and of uptake of O_2 in brains of DBA mice at the age of susceptibility, but not before or after. The latter finding is also consistent with ABOOD and GERARD'S[2] report of a deficit in oxidative phosphorylation at the time of susceptibility to seizures.

In conclusion, the alterations in (Na, K)-ATPase activity point to a genetically determined age-dependent deficit in the brain with resultant ionic and metabolic changes that seem to account for susceptibility to seizures in mice[5].

Zusammenfassung. Nachweis, dass in Gehirnen 30 Tage alter für audiogene Krämpfe anfälliger Mäuse (Stamm DBA) die Aktivität der (Na, K)ATPase gegenüber nicht Anfälligen (Stamm C57) signifikant vermindert war.

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Heart Tissue Acetylcholine in Chronically Exercised Rats[1]

The two divisions of the autonomic nervous system controling the activity of the heart were shown to have a modified tonus in the chronically exercised rat. TIPTON and TAYLOR[2] reported vagotony in the athletic heart and HERRLICH et al.[3] found an increase of acetylcholine concentration in the auricles of trained rats. Moreover, several authors have shown that chronic exercise also decreases the sympathetic nervous activity of the heart[4–6]. While previous evidence was presented by us that long-term exercise is related to a reduced heart catecholamine concentration[7,8], it now seemed appropriate to investigate further whether in our chronically trained rats the decreased catecholamine concentrations was accompanied by an increase of the cholinergic transmitter.

Methods. Male Wistar rats were exercised intermittently for a 3 month period at 500 m/h as described previously[7]. At the end of the training period, the animals were sacrificed by decapitation. Within 30 sec, the heart was excized, washed in icecold saline, blotted and plunged in liquid nitrogen. The frozen heart was weighed and ground

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