The optimal and maximum frequency of a motor unit can be assumed to be related to the fusion frequency and the contraction time of the motor unit. Consequently, the motor unit first recruited in tonic activity should have a long contraction time and the later the motor units are recruited in tonic activity, the shorter should be the contraction time of the motor units. On the other hand, the motor units first recruited in phasic activity should have a shorter contraction time than that of the motor units first recruited in tonic activity. It should thus be possible to recruit slow and fast twitch motor units in a different order in different types of activity.

Zusammenfassung. Bei willkürlichen tonischen Muskelkontraktionen werden motorische Einheiten mit niedriger Maximalfrequenz zuerst rekrutiert. Dagegen können bei willkürlichen phasischen Muskelkontraktionen motorische Einheiten mit hoher Maximalfrequenz zuerst rekrutiert werden.

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The Effect of Temperature and of Relative Acidity on the Concentration of Lactate in Cardiac Muscle

The physiological importance assigned to pH values obtained in biological material such as plasma at various temperatures, has to account for the effect of temperature on the ionic product of water or the ratio OH^-/H^+ as suggested by WINTERSTEIN[1]. Actually the arterial pH in poikilothermic animals increases, thereby keeping the ratio OH^-/H^+ constant when the animals are acclimatized to a lowered temperature (RAHN[2]). Instead of using this ratio, RAHN introduced the term 'relative alkalinity' which he defined as the ratio H_N^+/H^+, where H_N^+ is the hydrogen ion concentration of pure water and H^+ the hydrogen ion concentration of e.g. plasma, both at the same temperature. Mathematically H_N^+/H^+ is the square root of the ratio OH^-/H^+. A line chart for the evaluation of both terms as a function of pH and temperature was given by ALBERS[3]. As pointed out by REEVES[4], the constancy of the OH^-/H^+ ratio implies also a constancy of the fractional dissociation of imidazole which in turn provides for optimal enzyme activities and protein conformation. However, though RAHN's experiments clearly demonstrated that in lower vertebrates the relative alkalinity or the fractional dissociation of imidazole appears as a regulated variable, up to now no physiological reaction has been found which is correlated to the relative alkalinity rather than to the hydrogen ion concentration itself. In what follows the well-known increase of the lactate concentration caused by hyperventilation is shown to be possibly such a reaction. The concentrations of lactate and pyruvate in blood and cardiac muscle of rats were determined enzymatically (TFELT-HANSEN and SIGGAARD-ANDERSEN[5]) at a body temperature of 37.9°C (group I) and after cooling the rats to 22.3°C (group II) and 21.2°C (group III). The ventilation in the hypothermic rats in group II was adjusted to give the same OH^-/H^+ ratio of about 15 as was obtained at 37.9°C. In group III the ventilation was increased to give an OH^-/H^+ ratio 3 times as large. As shown in the Table, no change in the OH^-/H^+ ratio is observed if the arterial pCO_2 in the hypothermic rats is lowered to 17 torr and the arterial pH increased to 7.63 (group II) which corresponds to a reduction in the hydrogen ion concentration to 58% of the control value. There is no difference in the blood and tissue lactate between the rats under these conditions and the rats at 37.9°C. If, however, the ventilation in the hypothermic rats is increased, thereby lowering the arterial pCO_2 to 7.9 torr and increasing the pH to 7.90 (group III), the OH^-/H^+ ratio is increased to about 47, the blood lactate is doubled and the lactate in cardiac muscle is increased by more than 300%. The pyruvate concentration in blood and muscle does not change during hypothermia at both levels of ventilation. If at a normal body temperature the arterial pCO_2 is lowered and the arterial pH is increased to values compar-

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Blood gases and concentration of lactate and pyruvate in blood and cardiac muscle of artificially ventilated rats at normal and hypothermic body temperatures

	Group I	Group II	Group III	S.D.
Rectal temperature	37.9	22.3	21.2	± 0.8
pCO_2 (torr)	38.7	17.3	7.9	± 1.6
pH	7.398	7.632	7.895	± 0.051
$[H^+] \times 10^8$	4.00	2.34	1.29	± 0.23
$[OH^-]/[H^+]$	15.1	14.9	47.2	± 7.8
Lactate (blood) meq/l	1.26	1.05	2.36	± 0.33
Pyruvate (blood) meq/l	0.042	0.043	0.058	± 0.013
Lactate (muscle) meq/kg	1.30	1.68	4.38	± 1.00
Pyruvate (muscle) meq/kg	0.062	0.068	0.067	± 0.016

Each value is the mean of 5 animals. S.D. = standard deviation obtained by analysis of variance.

able with the values of the hypothermic rats of group II, the lactate concentration of the blood invariably increases to about 300 to 400% of the control values (HUCKABEE[6]). Since no increase occurs in the hypothermic rats at a pH of 7.60, the hydrogen ion concentration itself is apparently not correlated with the changes in lactate concentration in hypothermia. However, the lactate concentration increases also in hypothermia if the ventilation is enhanced and the OH^-/H^+ ratio is raised. It therefore seems conceivable that the increase in lactate caused by hyperventilation is correlated with the relative alkalinity rather than with the absolute value of the hydrogen ion concentration.

Zusammenfassung. Der durch Hyperventilation ausgelöste Milchsäureanstieg bleibt bei vergleichbarer Senkung des pH in Hypothermie aus, solange dabei der OH^-/H^+-Quotient bzw. die relative Alkalinität nach RAHN[2] konstant bleibt. Stärkere Hyperventilation, die zu einer Zunahme des OH^-/H^+-Quotienten führt, löst dagegen auch in Hypothermie einen Milchsäureanstieg aus.

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Binding of γ-Aminobutyric Acid and other Amino Acids by Particulate Fractions of Developing Rat Brain[1]

In previous reports it has been shown that γ-aminobutyric acid (GABA) 'binds' rapidly to subcellular particles of brain by a Na^+-dependent mechanism which does not require enzymatic action or cellular energy[2–7]. It has also been shown that Na^+-dependent GABA binding is maximal at about pH 7.3[4,8] which is approximately the isoelectric point (pI) for GABA[9]; i.e., GABA binding is maximal at physiological pH when it is present as a 'zwitterion' and hence this binding may require two oppositely-charged membrane sites. Some evidence has been provided that other amino acids may be bound by Na^+-dependent mechanisms to brain particles[4], but no studies have been carried out on these processes in developing brain. In the present study, the Na^+-dependency of binding of GABA has been compared with that of other amino acids in subcellular particles prepared from rat brain during development.

Male, Sprague-Dawley rats, 1–75 days old (Indianapolis Lab. Supply Co.) were purchased as littermates except for those which were 35–36 or 74–75 days old. After decapitation, cerebral hemispheres were excised rapidly, weighed and homogenized immediately in 10 volumes of ice-cold isosmotic sucrose solutions containing 0.1 or 0.2 μCi/ml of U[^{14}C]D-sucrose (New England Nuclear Corp.: 505 mCi/mmole) + 0.25–0.45 μCi/ml of the radioactive amino acids [2,3-^{3}H]γ-aminobutyric acid, 2 Ci/mmole; [^{3}H]L-glutamic acid, 1.9 Ci/mmole; [2-^{3}H] glycine, 11.1 Ci/mmole,

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Table I. Differences between percentage distributions of ^{3}H-amino acids and ^{14}C-sucrose in P_1 fractions of developing rat brain

Post-partum age of rat (days)	(% ^{3}H-amino acid) minus (^{14}C-sucrose) in P_1 fraction GABA	Glutamate	Glycine	Arginine
	Na^+-free homogenizing fluid			
1	0.6 ± 0.18	0.5 ± 0.15	0.2 ± 0.11 ^{b}	1.9 ± 0.34 ^{c}
11	0.4 ± 0.16	0.7 ± 0.10 ^{a}	0.3 ± 0.06	1.6 ± 0.10 ^{c}
15	0.3 ± 0.10	0.6 ± 0.13 ^{c}	0.3 ± 0.10	1.6 ± 0.13 ^{c}
21–22	0.5 ± 0.11	0.3 ± 0.13	0.2 ± 0.08 ^{c}	1.4 ± 0.16 ^{c}
35–36	0.5 ± 0.16	0.1 ± 0.10 ^{c}	0.3 ± 0.09	2.2 ± 0.27 ^{c}
74–75	0.5 ± 0.08	0.04 ± 0.10 ^{c}	0.2 ± 0.08 ^{c}	2.7 ± 0.45 ^{c}
	Homogenizing fluid containing 40 mM NaCl			
1	2.4 ± 0.29	1.2 ± 0.21 ^{c}	1.7 ± 0.26 ^{a}	2.7 ± 0.35
11	5.4 ± 1.02	1.9 ± 0.39 ^{c}	2.9 ± 0.29 ^{c}	4.7 ± 0.43
15	5.2 ± 0.40	2.7 ± 0.44 ^{c}	2.9 ± 0.16 ^{c}	5.6 ± 1.33
21–22	7.5 ± 1.08	2.9 ± 0.50 ^{c}	2.9 ± 0.14 ^{c}	3.9 ± 0.69 ^{c}
35–36	8.1 ± 0.54	3.2 ± 0.13 ^{c}	2.7 ± 0.47 ^{c}	3.5 ± 0.61 ^{c}
74–75	6.7 ± 0.35	2.7 ± 0.30 ^{c}	2.4 ± 0.22 ^{c}	3.2 ± 0.34 ^{c}

Means ± standard deviations of individual differences between ^{3}H and ^{14}C found in each pellet fraction; 4 pellets from 4 rat brains in all cases. $^a p < 0.02$; $^b p < 0.01$ and $^c p < 0.001$, for comparisons between values for other amino acids versus GABA.