frequency distribution of the entire cell population (type IM + IB) was symmetrical and that monophasic and biphasic secretory potentials could be found in the same cells (Figure 1) suggest that all cells studied in the present work are acinar. The finding that the latency for the stimulation-induced potential change was considerably longer for the type IM cells than for the type IB cells is of considerable interest especially in view of the finding that the decrease in membrane resistance preceded the type IM response but appeared concomitantly with the type IB response. The very marked decrease in membrane resistance seen during nerve stimulation leaves little doubt that ACh, which is the transmitter being released[10], increases the membrane permeability to ions. The fact that the secretory potential is often biphasic suggests that at least two ionic channels are opened by ACh. The results are thus consistent with the hypothesis of PETERSEN[5–7] that ACh acts by increasing the permeability to K^+ and Na^+.

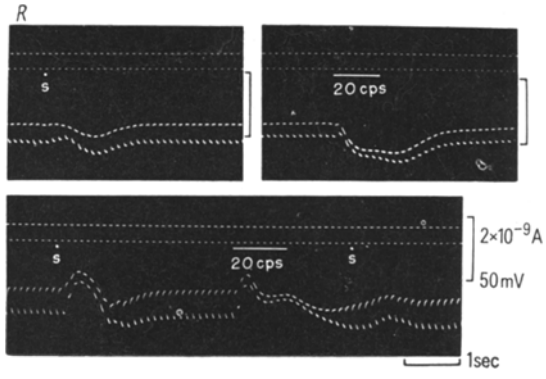

Fig. 3. Rabbit submaxillary gland: Measurement of membrane resistance during the secretory potential by application of hyperpolarizing current pulses through the intracellular recording microelectrode. s: single shock stimulation, 20 cps: repetitive stimulation.

Zusammenfassung. Die Membranpotentiale, in Azinuszellen der Submanibularisdrüse der Katze und des Kaninchens gemessen, waren höher als die in der früheren Literatur festgestellten. Die Stimulation erzeugte biphasische sekretorische Potentiale (Depolarisation-Hyperpolarisation), begleitet von einem Nachlassen des Membranwiderstandes. Es wird vermutet, dass Acetylcholin die Membranpermeabilität der Azinuszellen für zwei verschiedene Ionenarten erhöht.

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Reactivity of Myoglobin in Heart, Striated Muscle and Uterus in a Methaemoglobinaemia

Pathological processes during an anemic hypoxia of the methaemoglobin type can be characterized by the reactivity of two systems: 1. The detoxicating functions, i.e. the chemical transformation of methaemoglobin-forming agents, excretion and enzymatic reversibility of methaemoglobin formation; 2. The compensating capacity of the cardiovascular system: increase of cardiac output and reactivity of blood vessels. Whereas the complex of questions mentioned under 1. were thoroughly investigated during recent years, many physiological and biochemical problems connected with 2. remained without a clear pathophysiological answer. The results obtained from analyses of the rat heart proved that the changes of the percentages of the inactivated myoglobin components play an important role[1,2]; with respect to the microanatomical properties of various muscles, it was necessary to determine the extent of myoglobin inactivation under the same physiological conditions parallel in 3 preparations: the heart, the striated muscle and the uterus.

Materials and methods. 22 female rabbits were used for the experiments: 12 animals served as controls, and 10 were given a single injection of $NaNO_2$ solution. The animals were sacrificed 40, resp. 80 min after application of $NaNO_2$, and venous blood samples for haemoglobin and methaemoglobin determination[3] were taken. The concentration of myoglobin in the left heart ventricle, the triceps muscle and the uterus was estimated by the method of REYNAFARJE[4], and the myoglobin components (CO-myoglobin, NO-myoglobin and metmyoglobin) by the method previously described[1].

Results. In various types of muscles a differentiated reaction of myoglobin was observed. The amount of total myoglobin was not significantly changed in the $NaNO_2$-treated groups (Figure). A differentiated reactivity was observed in the determination of metmyoglobin formation. It is evident that in different types of muscles a different limiting rate can be observed. While in the heart the maximum amount was reached with 150 mEq $NaNO_2$ after 40 min, the maximum average value in the striated muscle was reached at the same time, but it was higher than in the heart. The highest rate of metmyoglobin formation was observed in the uterus, where also the speed rate was different: the maximum amount was found with 75 mEq $NaNO_2$ after 40 min.

Another characteristic can be seen during the myoglobin inactivation by forming NO-myoglobin. It could be proved that the higher dose and the longer treatment period increase the rate of NO-myoglobin formation. Therefore the total amount of inactivated myoglobin is after 75 mEq in 40 min greater than the inactivated part of haemoglobin. However, after 150 mEq in 40 min the amount of inactivated components remains in the same degree as that of inactivated haemoglobin in the erythrocytes.

Discussion. Myoglobin and its physiological properties remained, compared with haemoglobin, until today relati-

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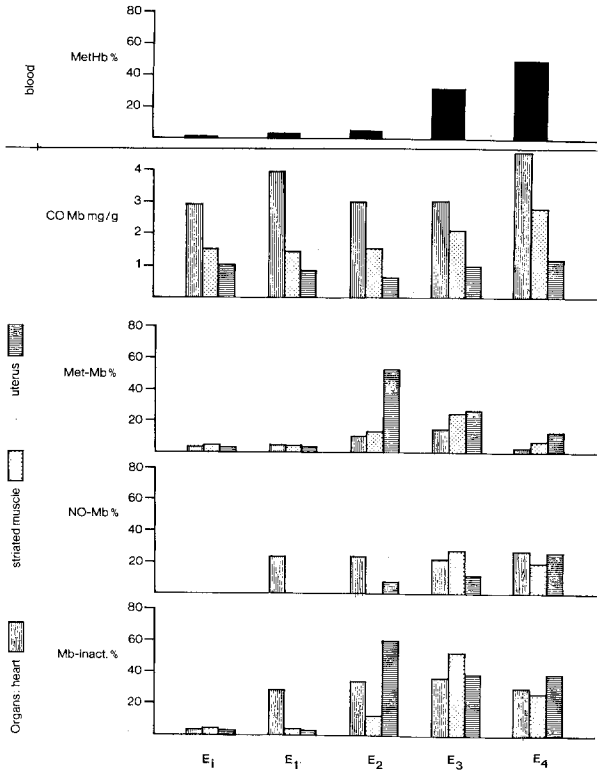

Experimental groups: E_i ($n = 12$) — control animals. E_1 ($n = 1$) — 75 mM $NaNO_2$/1 l solution; 3 ml/kg i.p. E_2 ($n = 2$) — 75 mM $NaNO_2$/1 l solution; 8 ml/kg i.p. E_3 ($n = 4$) — 150 mM $NaNO_2$/1 l solution; 5 ml/kg i.p. E_4 ($n = 3$) — 150 mM $NaNO_2$/1 l solution; 5 ml/kg i.p. The animals of the groups E_1, E_2, E_3 were sacrificed 40 min, those of the group E_4 80 min after the application on $NaNO_2$; controls remained untreated.

vely unclearly defined. Especially the situations where haemoglobin as the main oxygen transport system is either destroyed or inactivated, and the heart, striated and smooth muscle cells are involved in the cardiovascular compensatory mechanisms, have to be mainly studied. SCHOLANDER[5] emphasized in his experiments the possibility that haemoproteins may have an important influence on the facilitation of oxygen diffusion; this phenomenon would have a greater importance selectively in the processes of oxygen transport in the muscle cell. In the results referred to, it could be proved that the form of myoglobin and the rate of inactivation of ability for the intracellular oxygen transport are in a defined relation to the degree of incativation of haemoglobin. Last but not least it should be mentioned that all proved changes of the myoglobin molecule can be dependent upon the changing properties of myoglobin during the ontogenesis[6]; especially in human pathophysiology, the hypoxias of the methaemoglobin type occur during the early phase of life.

Zusammenfassung. Bei 21 Kaninchen wurde nach Injektion von $NaNO_2$ die Bildung von Methämoglobin, Metmyoglobin und NO-Myoglobin gemessen. Die Metmyoglobin-Bildung war am geringsten im Herzmuskel und am ausgeprägtesten im Uterus, während die Triceps-Muskulatur intermediäre Werte ergab.

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The Effect of Asphyxia and Re-Oxygenation on Bilateral Dorsal Root Potentials Produced by Stimulation of the Cutaneous Afferents

Asphyxia evokes profound changes in activity of the spinal cord. Both spinal reflexes and the dorsal root potentials (DRPs) rapidly decrease and finally disappear during the first min of oxygen deprivation[1-3]. Intracellular recording from motoneurones during acute asphyxia reveals that the arrest of reflex activity is due to the gradual decline of the excitatory postsynaptic potentials[4]. The failure of the motoneurone excitation is probably related to asphyxial potentials of the dorsal roots which depolarize the intraspinal part of the primary afferent fibres and thus produce their functional arrest[5]. The mechanism of this failure very much resembles the mechanism of presynaptic inhibition[6]. In the present investigation, the effect of acute asphyxia and re-oxygenation on presynaptic inhibition evoked by cutaneous volleys was studied. This inhibition was recorded as the DRPs produced by long-lasting unilateral stimulation on both sides of the spinal cord. It is known that after conditioning stimulation the DRP produced by the testing volley is depressed during a considerable period of time and this depression depends on presynaptic inhibition. In the experiments described below, this interaction was also investigated by determining the size of the testing DRP produced at fixed interval after each conditioning DRP.

Methods. The experiments were performed on 12 cats under light Surital anaesthesia. The spinal cord was severed at the first lumbar segment. The DRPs were led off from rootlets of the right and left L7 which entered the cord strictly at the same level. The potentials were produced by repetitive conditioning stimulation of 500 msec duration with frequency of 250 c/sec followed after 100 msec by the single testing pulse. This sequence of stimulations was applied every 7.5 sec to the superficial peroneal nerve. A stimulus strength of 4 times threshold was used. The spinal cord was asphyxiated during 3 min by clamping the thoracic aorta[4].

Results and discussion. The effect of acute asphyxiation and re-oxygenation on the DRPs are shown in Figures 1

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