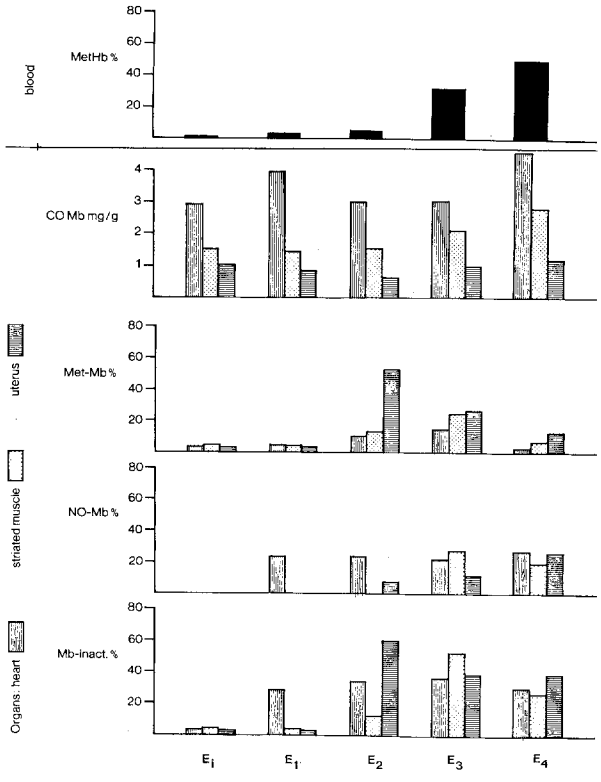

Experimental groups: E_i ($n = 12$) — control animals. E_1 ($n = 1$) — 75 mM $NaNO_2$/1 l solution; 3 ml/kg i.p. E_2 ($n = 2$) — 75 mM $NaNO_2$/1 l solution; 8 ml/kg i.p. E_3 ($n = 4$) — 150 mM $NaNO_2$/1 l solution; 5 ml/kg i.p. E_4 ($n = 3$) — 150 mM $NaNO_2$/1 l solution; 5 ml/kg i.p. The animals 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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and 2. The sample records are represented in Figure 1, while in Figure 2 the full time course of changes in size of these potentials is shown.

From records of Figure 1 it may be seen that in control conditions the prolonged stimulation of cutaneous afferents produces persistent depolarization of the dorsal root only on the ipsilateral side of the spinal cord. On the contralateral side, the potential increases to maximum and then it drops to zero despite continued stimulation. A testing DRP produced 100 msec after conditioning potential is depressed to about 49% of the control value on the ispsilateral side and to 8% on the contralateral side of the spinal cord.

Asphyxiation decreases the size of the conditioning and testing DRPs on both sides of the spinal cord and in 1.3–1.8 min all potential changes disappear. The testing DRPs are less resistant to circulatory arrest. On the ipsilateral side their surviral time is 1.3 min and on the contralateral side it amounts to 1.0 min. The conditioning DRPs disappear after 1.5 min on the ipsilateral and after 1.3 min on the contralateral side.

Re-oxygenation of the spinal cord is followed by prompt recovery of the ipsilateral DRPs which appear 2–3 min after the end of asphyxiation. In 58% of preparations at the beginning of recovery a hyperpolarization of the dorsal root is observed. In the experiment shown in the Figures, a slight hyperpolarization produced both

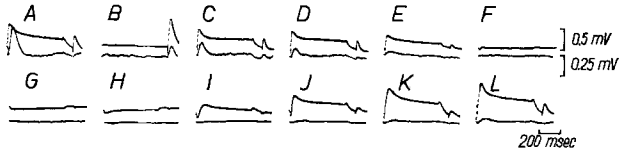

Fig. 1. The effect of acute asphyxiation on bilateral dorsal root potentials produced by long-lasting conditioning stimulation followed after 100 msec by single testing volley in the same cutaneous nerve. Upper traces of each record show ipsilateral and lower traces contralateral DRPs. A) and B) shows the control records. In B) the DRPs are evoked by testing volley. C–F) records of the DRPs taken 22, 30, 60 and 90 sec after the onset of asphyxia. G–L) recovery from asphyxiation. Records of the DRPs taken 1, 3, 5, 7, 9 and 11 min after the end of asphyxiation.

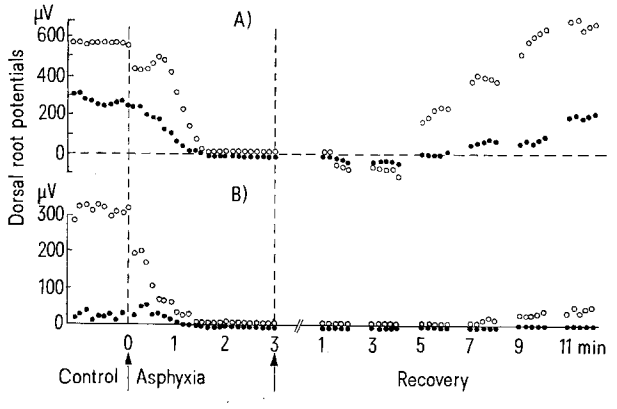

Fig. 2. The effect of asphyxia and re-oxygenation on the dorsal root potentials recorded on ipsilateral (A) and contralateral (B) side of the spinal cord. Open circles represent conditioning DRPs evoked by prolonged tetanization, filled circles represent the testing DRPs produced 100 msec after conditioning potentials by single volley in the same cutaneous nerve. Abscissa, time in min. Note contracted time scale during recovery. Ordinate, size of the DRPs in μV. In A) circles below zero line indicate hyperpolarization of the dorsal roots.

by conditioning and testing volleys occurs for about 3 min and then it disappears, being replaced by the negative DRPs. In other preparations, the reappearing potential from the very beginning has a form of depolarization. The ipsilateral conditioning DRP usually appears earlier and then, after about 7 min of recovery, the testing DRP is seen. The size of the conditioning DRP increases rapidly; after 9 min of revovery, it attains the control level and after another 2 min depolarization is larger then in control conditions. The growth of the testing DRP is much slower and up to the end of observation period it does not reach the initial value.

In contrast to the ipsilateral depolarization, the recovery of the contralateral DRPs is delayed and very slow. The conditioning DRP which appears as depolarization is visible 7 min after the end of asphyxiation, and at the end of observation period it only attains 14.8% of the control level. During all this period, the single testing volleys do not produce any contralateral depolarization. In several experiments the effects of a single testing volley are totally depressed during 15–20 min after the end of asphyxiation.

The changes in size of the DRPs observed during acute asphyxiation are most probably due to asphyxial potentials of the dorsal roots. Since it is known that depolarization decreases the presynaptic inhibition[7], it may be surmized that the gradual decrease of the DRPs occurring within a few min after oxygen deprivation depends on increase of the asphyxial potential. The total disappearance of the DRPs would indicate the block of transmission of the afferent impulses generating the primary afferent depolarization. BIERSTEKER et al.[5] found that the asphyxial potential increases during the entire duration of the asphyxiation, while the DRPs in our experiments disappear in the first 2 min of asphyxia. These data indicate that even small asphyxial potential may totally block transmission of volleys producing the DRPs. The recovery of the DRPs would depend on decrease of this potential. It is interesting to note that during recovery the depression of the testing DRP on the ipsilateral side is increased. This effect occurs when the conditioning DRP returned to its control value, suggesting the delayed differentiated action of asphyxia on the prolonged DRP and its after-effects. The slow recovery of the contralateral DRPs demonstrates that the oxygen deprivation is a potent factor counteracting the appearance of the primary afferent depolarization on the other side of the spinal cord. Since the synaptic organization of the pathway producing contralateral presynaptic inhibition is probably more complex than the ipsilateral one, this finding reflects its greater vulnerability to oxygen lack.

Résumé. Les potentiels conditionnants et les tests effectués sur la racine dorsale des deux cotés de la moelle montrent une sensibilité différenciée à l'asphyxie. Après le rétablissement de l'oxygénation la dépression du potentiel ipsilatéral testé est augmentée et les potentiels contralatéraux n'apparaissent qu'après un retard prolongé.

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