In vitro Oxygen Consumption of Slices from Kidney, Brain, Cortex and Liver in Hypoxia

Most authors assert that the acute renal insufficiency observed in shock, or after haemorrhage, is due to the damage caused by hypoxia.

Munck's *in vivo* experiments showed that in haemorrhagic shock the oxygen saturation of renal cortex capillary and of renal venous blood was scarcely decreased. He therefore doubted the importance of hypoxia in his cases¹. The degree of hypoxia is only one of the possible injurious factors; the other factor could be the sensitivity of different tissues to hypoxia.

In different mixtures of gases containing oxygen and nitrogen, we compared the *in vitro* oxygen consumption of renal cortex, brain cortex and liver slices.

White female rats (body-weight 130–150 g) were used. The animals were killed by decapitation. The rapidly removed organs were placed into Ringer phosphate buffer (pH 7.2; 0° C). The thickness of the slices was checked by Warburg's method. The respiration of 300 mg fresh wet tissue placed into 3 ml. buffer was measured in a Warburg apparatus. 0.1 ml. 20 per cent sodium hydroxide solution was used for absorbing carbon dioxide. Oxygen-nitrogen mixtures of different compositions were streamed through the flasks. Measurements were made at 30° and 37° C.

The oxygen consumption of renal cortex slices at 37° C was so strong that the deviation due to the frequent reading of the manometer was bigger than usual (see Tables 1 and 2).

Table 1. OXYGEN CONSUMPTION OF 300 MG FRESH TISSUE/H, 30° C

	100% O ₂	50% O ₂	20% O ₂	10% O ₂
Kidney	476	416	254	164
n = 12	± 37	± 9	± 43	± 3
Liver	172	140	126	97
n = 12	± 33	± 4	± 15	± 10

Table 2. OXYGEN CONSUMPTION OF 300 MG FRESH TISSUE/H, 30° C

	100% O ₂	50% O ₂	20% O ₂	10% O ₂
Kidney	442	323	247	148
n = 16	± 45	± 17	± 13	± 9
Brain	251	241	157	124
n = 16	± 37	± 11	± 12	± 9

Our experiments show that the oxygen consumption of the renal cortex is greatly influenced by hypoxia: (1) The oxygen consumption *in vitro* of renal cortex, brain cortex and liver slices is directly proportional to the logarithm of the oxygen content of the gas mixture (see Figs. 1–3). (2) The oxygen uptake of renal cortex slices decreased relatively more in gas mixtures with falling oxygen content than did that of the brain cortex and liver slices.

It can be assumed that the renal cortex is unable to take up the needed amount of oxygen even if the oxygen

Fig. 1. Relationship between *in vitro* consumption of oxygen by the renal cortex slices and the log oxygen content of gas mixture. $y - 321.9 = 293.7(x - 1.55)$

Fig. 2. Relationship between *in vitro* consumption of oxygen by brain cortex slices and the log oxygen content of gas mixture. $y - 175.07 = 171.4(x - 1.34)$

Fig. 3. Relationship between *in vitro* consumption of oxygen by liver slices and the log oxygen content of gas mixture. $y - 139.2 = 79.90(x - 1.55)$

tension is relatively high. This means that a degree of hypoxia which is too small to damage other organs may produce severe alterations in the kidney.

J. SKOLNIK*
L. TAKACS
E. SZENDE

Second Department of Medicine,
University of Budapest,
Budapest, 8.

* Present address: Department of Neurology, University of Budapest, Budapest, 8.

¹ Munck, O., *Absence of Renal Hypoxia in Haemorrhagic Shock* (Second Int. Congr. of Neph., Prague, 1963).

Time Course of Failure of Neuromuscular Transmission after Motor Nerve Section

AFTER transection of a motor nerve, the severed part of the axon undergoes progressive changes known as Wallerian degeneration. But before the degenerating axon loses its ability to conduct action potentials, function and structure of the motor nerve terminals are impaired and neuromuscular transmission fails¹. Several workers have noted that this failure occurs at a time which depends on the length of the degenerating nerve segment^{2,3}. This communication describes experiments which make it possible to relate quantitatively the length of the degenerating axon to the time at which neuromuscular transmission fails.

The experiments were made on the phrenic nerve-diaphragm preparation of female albino rats (140–170 g). Operating under anaesthesia, the left phrenic nerve was sectioned, either near its emergence from the spinal cord in the neck, or in the thorax a few mm from the entry of the nerve into the diaphragm. In the former case, 3.5–4.0 cm of extramuscular nerve remained connected to the