of DRPs was evoked when the vibration alone was applied (Fig. 1*D*). These experiments indicate that a tonic pressure pulse to the cat's central pad does not evoke DRPs. Therefore, the steady DRP which had been observed when gently squeezing the cat's foot by hand[4] is most probably not evoked by the steady pressure applied to the foot but by the slight tremor superimposed on it.

Figs. 1*C* and *D* indicate that a conditioning pressure pulse reduces the amplitude of the DRP produced by a testing pressure pulse. Fig. 1*E* shows the full course of the depression of a DRP, which was evoked by the second of two identical 4 msec pressure pulses applied to the central pad in turn. Since peripheral interaction of pairs of mechanical pulses to the large pad does not exceed 10 msec[5], the observed reduction of the DRP must be of central origin, and presumably it is due largely to presynaptic inhibition[6]. Inhibitory curves of similar intensity and duration have been obtained by a short conditioning pressure pulse on the time integral of the flexor reflex or the monosynaptic and polysynaptic discharges in the ipsilateral dorsolateral cutaneous tract. A first topographical pattern of presynaptic inhibition has been detected on stimulation with two Rochelle salt crystals at different sites on the large pad; an increase in the distance between the stimulating styli reduced the effectiveness of presynaptic inhibition. There is also an interaction between the DRPs produced by stimulation of hairs of the paw and the DRPs evoked by pressure pulses on the pad.

The present experiments demonstrate that at least two types of peripheral receptors, namely hair follicle receptors[3] and touch receptors of the pad[5], produced presynaptic inhibition of primary afferent fibres of the spinal cord. This inhibition is similar to that found after synchronous electrical stimulation of cutaneous nerves. Excitability testing of single primary afferent fibres in the spinal cord is now being attempted to determine which types of afferent fibres are depolarized when stimulating these receptors.

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Hyperoxia and the Intravascular Volumes of Tissues in the Growing Rat

THE period of rapid myelination in central nervous development is immediately preceded by a rapid increase in oxygen utilization by the brain[1]. This is accompanied by a phase of rapid growth of capillaries and arterioles in the central nervous tissue[2–4]. By injecting dye Gyllensten[3] has recently shown that prolonged exposure to 90–100 per cent oxygen reduced the growth of new capillaries in the mouse brain during this stage of development.

The present communication describes an investigation of blood volumes in lung, liver, kidney and brain in adult and immature normal rats and in 9 day old rats which had been reared from birth in an atmosphere containing 90–100 per cent oxygen. The blood volumes of the organs were measured using red blood cells labelled with chromium-51 (refs. 5, 6) and the results expressed as grams of blood per gram of organ wet weight[6]. The animals were killed by light ether anaesthesia followed by immersion in liquid nitrogen.

Table 1 shows that there is a progressive increase in blood volume per gram of lung, liver, kidney and forebrain of normal rats from 2 days after birth to adulthood, but there are slightly higher volumes during the day of delivery compared with the second day of life.

In the adult rat the blood volumes were similar to those found by other workers[7,8]—with the exception of the kidney, which we found to have a lower blood volume than previously observed (Table 2).

The blood volumes of lung, kidney and brain for 9 day old animals reared in an atmosphere rich in oxygen for 8 days and then allowed to remain in air for 1 day are shown in Table 3.

Table 1. RADIOACTIVE BLOOD EQUIVALENTS OF ORGANS AND TISSUES IN NEWBORN, IMMATURE AND ADULT RATS (BLOOD EQUIVALENTS EXPRESSED AS G TAIL BLOOD/G WET WEIGHT)

Age in days	*n*	Lung	Liver		Kidney	Brain
			Right lobe	Left lobe		
1	6	0·423	0·192	0·194	0·071	0·012
2	6	0·329	0·144	0·167	0·060	0·011
4	8	0·447	–	–	–	0·011
9	6	0·426	0·161	0·186	0·076	0·011
16	4	0·431	–	–	–	0·016
27	3	0·562	0·265	0·297	0·097	0·019
Adult	4	0·494	0·324	0·270	0·127	0·031

Table 2. BLOOD VOLUMES OF ADULT RAT ORGANS ACCORDING TO THR DIFFERENT GROUPS OF WORKERS

	Lung	Liver		Kidney		Brain	
Oeff *et al.* (ref. 7) using phosphorus-32	0·43	0·25		0·37		0·024	ml. blood/g wet weight
Nair *et al.* (ref. 8) using iodine-131	–	Left lobe 0·82		Cortex 0·68	Medulla 1·41	Cerebral cortex 0·08	ml. blood/mg dry weight
Trappitt and Spector using chromium-51	0·49	Right 0·32	Left 0·27	0·13		Cerebral hemisphere 0·022	g blood/g wet weight

Table 3

Organ	Mean	*n*	*S.E.* of mean
Lung	0·401 g blood/g wet weight	9	0·029
Kidney	0·072 g blood/g wet weight	9	0·009
Forebrain	0·008 g blood/g wet weight	9	0·001

No significant difference occurred between the blood volumes of liver and kidney at this age in normal and hyperoxic rats (using *t* test, $P = 0{\cdot}3$–$0{\cdot}4$). The value for forebrain was reduced compared with that in controls, but the difference fell outside the accepted limits of statistical significance ($t = 1{\cdot}97$; $P = 0{\cdot}05$–$0{\cdot}1$). The observations of Gyllensten were made on the number and size of capillaries in the cerebral cortex while the method of blood equivalent used in this work measured total blood volume. Thus, although there is evidence that cerebral cortical capillary growth is markedly inhibited by chronic hyperoxia in growing animals, the present experiments suggest that the development of the total intravascular space in the entire forebrain is not greatly altered under these conditions. Similarly, no detectable difference in blood volume of lung and kidney tissues was observed. Nevertheless the hyperoxia was sufficiently severe to lower the haematocrit. In a group of control animals aged 6–7 days the mean haematocrit was 41·3 ($n = 14$), while in test animals in the same range of ages the mean haematocrit was 33·0 ($n = 5$).

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