

or veins or of capillaries. Arteries were often constricted to less than 50% of their original diameter but the maximum venous constriction was only about 20% when the paravascular nerves were stimulated under these conditions. The constrictor effect of nerve stimulation was blocked by guanethidine ( $10^{-6}$  g/ml) or by phentolamine ( $10^{-6}$  g/ml). The response was restored after the wash out of guanethidine by the inclusion of D-amphetamine ( $2 \times 10^{-6}$  g/ml) in the bathing solution. All arteries and veins, including those not responsive to nerve stimulation, were constricted by noradrenaline and this constriction was blocked by phentolamine ( $5 \times 10^{-6}$  g/ml). Noradrenaline in concentrations up to  $10^{-5}$  g/ml had no effect on capillary diameter. A gradient in the sensitivities in the arteries was found; the smaller arteries being more responsive to noradrenaline. Veins were significantly less sensitive than arteries of comparable size.

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### **The influence of some centrally acting drugs on sympathetic nerve activity**

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Pharmacological agents have been used to investigate the possible involvement of descending monoaminergic pathways in the regulation of sympathetic outflow, since such pathways have been shown, by fluorescent histochemical studies, to terminate in the region of the lateral horn cells of the thoracic spinal cord (Dahlstrom & Fuxe, 1965).

In unanaesthetized, decerebrate cats with a spinal section at C1, L-dopa, 100 mg/kg i.v., administered with a peripheral dopa decarboxylase inhibitor, MK486 (1- $\alpha$ -hydrazino- $\alpha$ -methyl dopa) 50 mg/kg i.v. had little apparent effect on spontaneous activity but depressed reflex activity recorded from preganglionic (white rami communicantes) and post-ganglionic (renal) sympathetic nerves in response to stimulation of spinal afferent nerves.

In another series of experiments, reserpine, 1 mg/kg, was administered intraperitoneally on each of four consecutive days before the experiment was carried out. The animals were anaesthetized with a mixture of chloralose, 35 mg/kg, and urethane, 700 mg/kg. Spontaneous activity in pre- and postganglionic sympathetic nerves was indistinguishable from that in the normal animal.

However, following the administration of reserpine, 5 mg/kg, intraperitoneally, four hours prior to the initiation of anaesthesia (chloralose and urethane as above), the spontaneous activity recorded from preganglionic (splanchnic) and postganglionic (renal) sympathetic nerves had increased considerably, compared with the untreated animal. In addition, the effectiveness of baroreceptor stimulation in inhibiting sympathetic nerve activity was decreased, tested by means of a carotid sinus blind sac preparation. When reserpine, 5 mg/kg i.v., was given acutely during an experiment, similar changes were seen, the onset of the effect being approximately four hours after injection.

In the anaesthetized preparation,  $\alpha$ -methyl dopa, 30 mg/kg, infused slowly into a vertebral artery was found to have little effect on spontaneous activity recorded from pre- and postganglionic nerves.

These experiments support an hypothesis that there is an inhibitory monoaminergic link between the medulla and the lateral horn cells of the thoracic spinal cord.

#### REFERENCE

DAHLSTROM, A. & FUXE, K. (1965). Evidence for the existence of monoamine neurons in the central nervous system. II. Experimentally induced changes in the intraneuronal amine levels of bulbo-spinal neuron systems. *Acta Physiol. Scand.*, **64**, Suppl., **247**, 1–36.

#### **The iontophoretic release of aspartate and glutamate ions from multibarrelled micropipettes (T)**

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#### **The fractionation of brain tissue by zonal centrifugation with special reference to the synaptosomal populations (T)**

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#### **Effects of $\alpha$ - and $\beta$ -adrenoceptor blocking agents on the release of renin by renal nerve stimulation in the cat (T)**

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#### **Action of papaverine on spinal reflexes (T)**

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#### **Central actions of catatonia-inducing drugs with a structural resemblance to catecholamines (T)**

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#### **Effects of long-term motor nerve stimulation on the pharmacological responses of rabbit muscles (T)**

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