Blood flow in the neurohypophysis of the rabbit associated with hormone-releasing stimuli

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Stimuli which release hormones from the neurohypophysis in rats have been shown, by blood volume techniques, to be followed by an acetylcholine-mediated vasodilation in this organ (Sooriyamoorthy & Livingston, 1971).

In order to extend these investigations neurohypophyseal blood flow has been studied in adult rabbits anaesthetized with Inactin (Promonta) using ^{133}Xe clearance methods based on those of Cranston & Rosendorff (1971). The hormone-releasing stimuli used were central vagal stimulation, intra-carotid administration of $CaCl_2$ and haemorrhage.

Saline (2–5 μl) saturated with ^{133}Xe (Radiochemical Centre) was introduced into the neurohypophysis by a needle in a steel jacket inserted through the mouth and entering the sella tursica via a natural foramen. The position of the needle in the neurohypophysis was checked radiographically. The peripheral arterial blood pressure was recorded from a cannula placed in the femoral artery.

For these studies a tissue/blood partition coefficient (λ) was assumed to be similar to that for hypothalamus (0·74) as used by Cranston and Rosendorff (1971).

The experiments gave a control blood flow of $11{\cdot}8 \pm 0{\cdot}58$ (ml/100 g tissue)/min for the neurohypophysis and this flow was significantly increased by vagal stimulation to $23{\cdot}9 \pm 2{\cdot}1$ (ml/100 g tissue)/min and by $CaCl_2$ injection to $14{\cdot}7 \pm 0{\cdot}47$ (ml/100 g tissue)/min. After haemorrhage (20–25 ml blood) the blood flow still remained close to the control values despite a 40% drop in peripheral blood pressure. This may have been due to vasodilation associated with the release of hormones, or may have been the result of autoregulatory activity, or a combination of both.

The time course of the responses showed that vagal and $CaCl_2$ stimulation produced a maximal effect between 30 and 60 sec after the application of the stimuli. There was also a temporary drop in blood pressure of about 20% which persisted for 60 sec.

Studies with atropine sulphate (2 mg/kg, i.v.) showed that this blocked the response to vagal stimulation completely, but that $CaCl_2$ still caused a slight increase, possibly associated with the maintained raised peripheral blood pressure produced by $CaCl_2$ after atropine administration.

Methacholine (0·002 mg/kg, i.v.) gave an atropine-sensitive increase in blood flow of 112%, an effect which persisted for about 70 min after a single injection.

Vasoxine (0·2 mg/kg, i.v.) did not produce any significant changes in blood flow either alone or in combination with α-adrenoceptor blocking drugs. Both methacholine and vasoxine produced significant changes in the peripheral arterial blood pressure.

These experiments show that there is an increase in blood flow through the neurohypophysis after hormone-releasing stimuli which is associated with the vasodilation previously demonstrated.

REFERENCES

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