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THE ACTION OF ADRENALINE, NORADRENALINE AND ACETYLCHOLINE ON BLOOD FLOW THROUGH JOINTS

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It was shown by Cobbold & Lewis (1956*a*) that, when measured by the bubble flowmeter, adrenaline decreased the arterial inflow to the dog knee joint. The action of adrenaline on the venous outflow has now been studied, and, since joint blood vessels show sympathetic vasoconstrictor tone (Cobbold & Lewis, 1956*b*), this action has been compared with that of noradrenaline and of acetylcholine.

METHODS

The measurement of blood flow in the knee joint of the anaesthetized dog has been described by Cobbold & Lewis (1956*a*, *b*). For the study of drug action the articular vascular bed in eight such dogs was isolated, all vessels being tied off except the highest articular branches of the femoral artery and vein. A polythene cannula was inserted into the stump of the saphenous artery and placed so that intra-arterial injection could be made as close to the joint as possible. The responses to the drugs were observed in the normally innervated and in the sympathectomized joint blood vessels. Blood pressure was recorded from a carotid artery using a mercury manometer.

RESULTS

Adrenaline, when administered by close intra-arterial injection, caused a marked vasoconstriction in both the innervated and the denervated joint. Constriction was found as the response to all doses, being transient in doses below 0·2 μg, but at this level and above the flow was reduced to, or nearly to, zero and the constriction lasted for some minutes (Fig. 1).

Noradrenaline, like adrenaline, caused a vasoconstriction of joint blood vessels in all doses, but its effect was more powerful and constriction was more prolonged (Fig. 2). With doses greater than 0·2 μg the constriction lasted for some minutes.

Acetylcholine was a powerful dilator of joint blood vessels and a dilator effect was still found in the sympathectomized joint, even when the blood flow was greater than that of the innervated joint (Fig. 3).

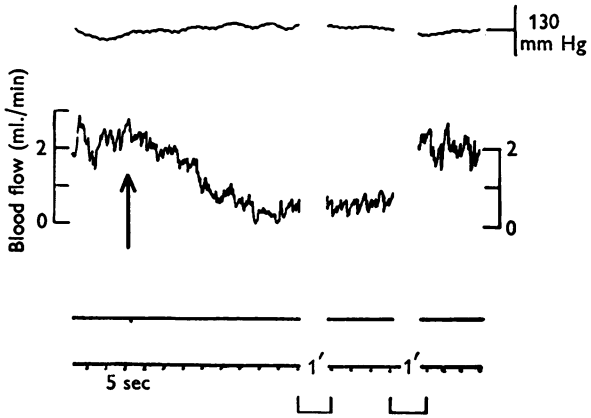

Fig. 1. Effect of adrenaline on joint blood flow. At the arrow intra-arterial injection of adrenaline 0·2 μg. The kymograph was stopped for intervals of 1 min. at the times indicated. In this and subsequent figures the upper record is of blood pressure in mm Hg and the lower is of venous outflow from the joint in ml./min. Time marker 5 sec.

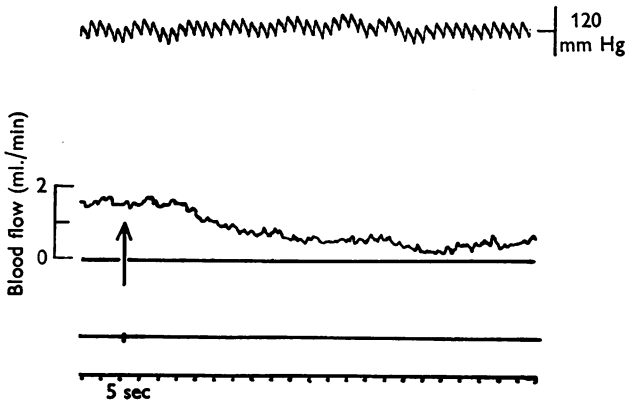

Fig. 2. Effect of noradrenaline on joint blood flow. At the arrow noradrenaline, 0·2 μg, was given by close intra-arterial injection.

DISCUSSION

The vasoconstrictor action of adrenaline on joint vessels previously reported has been confirmed by a method in which the venous outflow from the joint was measured by the electromagnetic flowmeter. The dilator response described by Bonney, Hughes & Janus (1952) has never been observed to occur with the dog knee joint. The fact that intra-arterial doses of adrenaline and of noradrenaline caused vasoconstriction of denervated joint blood vessels was in accord with the observation that they are under the control of sympathetic

vasoconstrictor fibres and show vasoconstrictor tone (Cobbold & Lewis, 1956*b*).

In this vascular bed, as elsewhere, acetylcholine had a marked vasodilator activity, and the fact that this persisted after the joint blood vessels had been deprived of their sympathetic innervation suggests that they possess a considerable basic and inherent constrictor tone which was not removed by denervation.

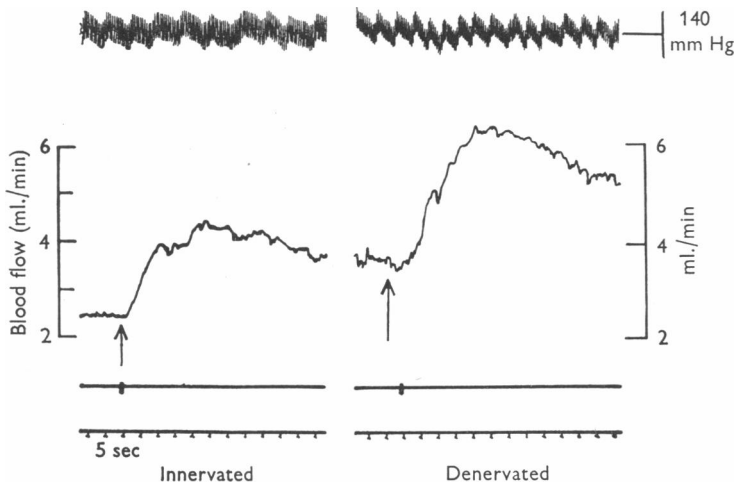

Fig. 3. Effect of acetylcholine on venous outflow from the innervated and denervated joint. At the arrows acetylcholine, 0·5 μg, was given by close intra-arterial injection.

SUMMARY

1. The venous outflow from the knee joint of the anaesthetized dog has been measured using an electromagnetic flowmeter.

2. Adrenaline and noradrenaline caused constriction of joint blood vessels.

3. Acetylcholine caused dilatation of joint blood vessels.

REFERENCES

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