ATRIAL RECEPTORS AND HEART VOLUMES

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THE FUNCTION OF ATRIAL RECEPTORS

The atrial receptors have reflex connections to the heart, such that on stimulation of the receptors there is an increase only in the heart rate; reflex connections to the kidney, causing an increase in water and sodium excretion and to the brain causing an inhibition of water intake.

Microscopically these receptors are unencapsulated nerve endings found in the subendocardial tissue at the junctions of the superior and inferior vena cavae and the right atrium, and the pulmonary veins and the left atrium. Methods of stimulating these receptors without interfering with the remainder of the circulation have been devised and result in an increase in heart rate (1,2) ranging from 10 to 90 beats/min. The afferent pathway is solely in the vagal nerves; a technique of cooling the vagal nerves showed that the increased activity in afferent vagal myelinated fibres, and the increase in heart rate, from the stimulation of atrial receptors could both be gradually blocked during stepwise cooling below 18 degrees celsius and completely blocked at 9 degrees celsius (3). The efferent limb of the reflex was solely in the sympathetic nerves to the heart and there was, surprisingly, no positive inotropic effect (1). No effect on efferent vagal nerves has been observed (2). The efferent sympathetic pathway of the reflex to the heart constituted a single bundle of nerves, and these afferent nerves were not involved in any other reflexes (4).

Stimulation of the atrial receptors was shown to inhibit the activity in efferent sympathetic nerves to the kidney, increase the activity in efferent sympathetic nerves to the heart but to have no effect on any other efferent sympathetic nerves (5). However distension of balloons in the left atrium resulted in an increase in urine flow. Using the stepwise cooling technique this reflex response was also shown to be a result of increase activity in atrial receptors and vagal afferent myelinated fibres (1). Right atrial receptors were shown to have the same effect.

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The efferent limb to the kidney of the atrial receptor reflex consists of three components. The nervous component, a depression of efferent sympathetic activity, causes an increase in urine flow and a small increase in sodium excretion; a haemodynamic component, resulting from changes in heart rate, causes a small increase in urine flow and sodium excretion; a hormonal component which causes an increase in excretion only of water and consists of a depression of the secretion of ADH and the increase in the concentration of a diuretic substance, as yet of unknown identity (2,6). There is also a decrease in renin concentration (7).

Some support for this concept that these receptors are volume receptors was supplied by the experiments of Gupta et al (8) who prepared two group of dogs with different blood volumes; one group had a mean blood volume of 95 ml.kg\(^{-1}\) and the other 57 ml.kg\(^{-1}\). The systemic blood pressure, atrial pressure, heart rate, plasma concentrations of sodium and plasma protein were not different between the two groups. Stimulation of atrial receptors resulted in greater diuretic and natriuretic responses in the dogs with high blood volume than in those with lower blood volume; results which support the hypothesis.

Stimulation of atrial receptors also inhibits drinking in the dog. Fitzsimons and Moore-Gillon (9) distended a small balloon in a pulmonary vein-atrial junction of the conscious dog which caused a reduced spontaneous water intake without affecting food intake. This inhibition of thirst was not observed when the left vagosympathetic trunk was blocked. The most likely receptors to be involved are the left atrial receptors. Similar effects on drinking have been observed by distending the junction of the right superior vena cava and the right atrium in the rat (10).

**Summary**

Stimulation of atrial receptors, discharging into vagal myelinated afferent nerves, has resulted in:

1. An increase in heart rate by efferent sympathetic nerves with no changes in inotropic state.
2. An increase in urine flow by hormonal mechanisms with no change in sodium excretion.
3. An increase in urine flow and sodium excretion by haemodynamic changes and by changes in renal efferent nerves.

**PROPOSED CONTROL SYSTEM**

As part of a control system the above responses can be included in only one hypothesis; that atrial receptors, attached to myelinated vagal afferent fibres, are the first link in a negative feedback mechanism controlling heart volumes. Such a mechanism has already been postulated (1,2,11,12,13). It is suggested that, without such a reflex control system, an increased inflow of blood to the heart, caused by an increase in venous return and/or blood volume, would cause the heart to increase its volumes and work over a range of larger volumes. This proposed control system would cause changes such that the heart would function over a range of small volumes. The heart would be smaller and this would be advantageous.