Resetting of the baroreceptor in hypotension in rats

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Summary

1. A progressive but incomplete adaptation of the aortic baroreceptor was observed after 1 and 6 h of hypotension in rats subjected to controlled bleeding. Administration of phenoxybenzamine produced a greater downward displacement of baroreceptor activation range.

2. Complete adaptation was observed after 48 h of maintained hypotension in rats treated with reserpine.

3. The range of the aortic baroreceptor activation shifted upward as pressure returned to normotension, indicating the reversibility of baroreceptor adaptation in hypotension.

Key words: baroreceptor adaptation, baroreceptor resetting, haemorrhagic hypotension.

Introduction

Studies performed in different species extended the earlier observation of McCubbin, Green & Page (1956) and conclusively established that baroreceptors reset to operate at higher pressure in chronic hypertension. In the rat the time-course of baroreceptor adaptation during the onset of acute hypertension lasts 1–2 days (Krieger, 1970) whereas the readaptation process during the reversal of chronic renal hypertension takes only 6 h (Salgado & Krieger, 1973). In the present experiment the effect of reduction of arterial pressure on baroreceptor function was studied. Two series of experiments were performed. In the first series the process of adaptation was studied in rats subjected to haemorrhagic hypotension lasting 6 h. In the second series the baroreceptors were analysed after 48 h of maintained hypotension in rats treated with reserpine.

Methods

Male Wistar rats weighing 200–250 g were used. The procedures for recording neural activity in the aortic baroreceptors during rapid changes in pressure were the same as used in previous studies from this laboratory. The systolic and diastolic pressures necessary to obtain a 'normal' firing pattern, the pressure threshold for baroreceptor activation and the pressures producing continuous discharge (saturation) were used to indicate the firing range of baroreceptors. Control arterial pressure was measured in conscious rats directly from the femoral artery by means of a cannula implanted under ether anaesthesia. Serial measurements of blood pressure were performed in rats injected with reserpine (2 mg/kg intraperitoneally). The a-receptor blockade produced by injection of phenoxybenzamine (5 mg/kg intravenously) was tested frequently by means of the pressor effect of noradrenaline.

Results and discussion

Progressive downward displacement in the range of the aortic baroreceptor function was observed 1 and 6 h after the reduction of blood pressure produced by controlled bleeding. However, the process of adaptation was not complete since the systolic and diastolic pressures necessary to obtain 'normal' firing discharge was higher than the pressures exhibited by the rats, and the pressure threshold for activation of baroreceptors was close to the
control pressure. This finding is similar to that observed during the onset of acute hypertension, when the process of adaptation was still incomplete after 6 h (Krieger, 1970). The downward shift of the baroreceptor function during haemorrhagic hypotension was more intense in those rats that received an α-receptor-blocking agent. Sympathetic inhibition in the control period also produced a small effect on baroreceptor function. After injection of the blocking agent approximately 10% less pressure was necessary to produce the same pattern of firing.

The time-course followed by the process of adaptation was studied in rats maintained in a hypotensive state for 48 h after receiving reserpine. Complete adaptation of the baroreceptors was observed after 48 h of hypotension and the process was proportional to the severity of the pressure decrease. Therefore the time taken for the baroreceptor to adapt to hypotension was almost the same as when the pressure was increased to hypertensive levels (Krieger, 1970).

Reversibility of the process of adaptation was observed in the rats treated with reserpine during the period when pressure was returning to normal. Since the upward shift of the baroreceptor activation range closely correlated with the change in blood pressure, this seems to indicate that the process of readaptation takes place more rapidly than the process of adaptation, as was observed in the reversal of chronic renal hypertension (Salgado & Krieger, 1973).

The observation of Judy, Murphy, Guleff & Selkurt (1975) that mean aortic nerve activity gradually recovered to 30% of control values at the end of 60–90 min of haemorrhagic hypotension in cats agrees well with the present findings that a 20% downward displacement of aortic baroreceptor activation occurred after 1 h of hypotension in the rat.

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References


