Recordings of renal and splanchnic sympathetic nervous activity in normotensive and spontaneously hypertensive rats

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Summary

1. Recordings have been obtained from single sympathetic fibres to the left kidney in anaesthetized adult spontaneously hypertensive (SH) rats and Wistar–Kyoto normotensive (WK) rats.
2. Under control conditions SH rats had a mean sympathetic activity of 3.3 ± SE 0.45 Hz and WK rats 1.6 ± 0.23 Hz. During noradrenaline infusion single fibre discharge decreased gradually to low levels in both SH and WK rats. The absolute decrease in fibre activity/mmHg rise of arterial pressure was greater in SH rats than in WK rats, but the reverse was true when the discharge reduction was expressed as a percentage change.
3. Recordings have also been obtained from the left splanchnic nerve in awake SH and WK rats. Upon volume load with blood, in SH rats the sympathetic activity decreased significantly more than in WK rats.
4. Thus the sympathetic discharge to the kidneys is higher in SH rats than in WK rats. Upon volume load SH rats show an exaggerated reflex response, probably due to an augmented volume-receptor response.

Key words: renal sympathetic activity, splanchnic sympathetic activity, sympathetic activity.

Abbreviations: SH, spontaneously hypertensive; WK, Wistar–Kyoto strain.

Introduction

The role of the sympathetic nervous system in the pathogenesis of essential hypertension is a contro-
versial issue. Thus recordings of the integrated or rectified nervous activity in splanchnic nerves in anaesthetized (Okamoto, Nosaka, Yamori & Matsumoto, 1967; Judy, Watanabe, Henry, Besch & Aprison, 1976; Judy, Watanabe, Henry, Besch, Murphy & Hookel, 1978) and awake spontaneously hypertensive (SH) rats (Judy et al., 1976) indicated an elevated nervous discharge in these rats compared with the normotensive control rats. Moreover, Coote & Sato (1977) concluded that cardiovascular pressure receptors are less able to induce sustained sympathetic inhibition in SH rats than in the normotensive rats, indicating a higher sympathetic activity in SH rats.

In contrast Lais, Shafter & Brody (1974) reported no increase in sympathetic traffic in the lumbar sympathetic nerves in SH rats. By more indirect means Iriuchijima (1973) observed an elevated splanchnic sympathetic activity in SH rats.

The aim of the present study was to record single fibre activity in post-ganglionic sympathetic fibres close to the kidney and compare the firing rates in SH rats with those in their normotensive Wistar–Kyoto (WK) controls. With this approach it is possible to quantify the sympathetic outflow.

In another series of experiments we have recorded splanchnic nervous activity in awake adult SH and WK rats via implanted electrodes during normal conditions and during volume load with blood.

Methods

Single fibre recordings

During a brief enalynmalnatrium (Brietal) anaesthesia a catheter was placed in the tail artery and chloralose (50-100 mg/kg body weight) and...
urethane (500 mg/kg body weight) were then given intra-arterially. The right femoral artery and vein were cannulated. The left renal nerves were reached via a retroperitoneal approach and single fibre recordings were obtained by splitting of the renal branches. The acid–base balance was checked at intervals and any tendency towards acidosis was corrected by injection of small amounts of NaHCO₃.

Splanchnic nerve activity in awake SH and WK rats

Under pentobarbital (Nembutal) anaesthesia the left splanchnic nerve was dissected free distal to the adrenal branches and placed on a small bipolar silver electrode. Silicon rubber (Wacker Sil Gel 604) was applied around the nerve in order to isolate and stabilize the electrode. The recordings were then performed 36–48 h later.

Results

Single fibre recordings

Recordings were obtained from seven single fibres in seven adult WK rats and 11 single fibres in nine adult SH rats. The mean spontaneous activity was 1.6 ± 0.23 Hz in WK rats and 3.3 ± 0.45 Hz in SH rats. These differences were significantly different (P < 0.01, Student's t-test). The baroreceptor reflex arc was also tested by slow infusions with noradrenaline (0.6–1.2 μg/min) in order to increase arterial pressure by 50–75 mmHg. The mean arterial pressure was then plotted against the average discharge. Fig. 1 shows the mean pressure–discharge curves for seven fibres in seven WK rats and 11 fibres in nine SH rats.

Recordings in awake animals

Satisfactory recordings were obtained in nine SH rats and eight WK rats. During volume load the splanchnic nervous activity decreased markedly. Thus at 10% increase of blood volume the splanchnic outflow decreased 33% in WK rats and 43% in SH rats. This difference was significantly different (P < 0.001). This augmented reflex response was not caused by a more marked inhibition from the arterial baroreceptors in SH rats because the arterial pressure increased less in these rats (2.1 mmHg) than in WH rats (8.0 mmHg) at 10% volume load.

Discussion

This is the first study to use single fibre recordings in order to quantify sympathetic outflow in WK and SH rats and the data clearly indicate a marked elevation of the sympathetic activity to the kidney in SH rats. The average number of impulses was twice as high in SH rats than in WK rats. These data are in accordance with earlier results by Judy et al. (1976, 1978). However, these authors used whole nerve recordings and with such techniques it is not possible to quantify the nerve activity since it is markedly dependent on the electrode position (Coote & Sato, 1977), which is not the case with single fibre recordings. A drawback with the present study is, of course, that the recordings must be performed on anaesthetized animals and after some surgery (i.e. retroperitoneal incision).

From the present data the baroreceptor reflex 'gain' can be expressed as the absolute or percentage reflex reduction in traffic per mmHg pressure rise during infusion with noradrenaline (Fig. 1). When expressed as absolute reflex reduction, the baroreceptor 'gain' is higher in SH rats and when expressed in relative terms the 'gain' is lower. The reason is that the control discharge is higher in SH rats than in WK rats.

Our experiments with recordings of sympathetic activity in awake rats upon volume load show an exaggerated reflex response to volume load in SH rats. The reason is not due to a more marked baroreceptor reflex inhibition. The most likely explanation is an augmented low pressure receptor reflex in SH rats. One possible reason for this might be alterations on the capacitance side.
Acknowledgments

This research was supported by grants from the Swedish Medical Research Council (no. 14X-4764 and 14X-00016) and from Magnus Bergvalls Stiftelse.

References


