Angiotensin II microinjections in the nucleus tractus solitarius has an inhibitory effect on the cardiac but not the non-cardiac sympathetic component of the baroreceptor reflex

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The nucleus tractus solitarius (NTS) is a major nucleus located in the dorsal medulla and is critical in the mediation of the baroreceptor reflex. It is also a site which contains a high density of high affinity binding sites for angiotensin II (AngII). Previous studies have shown that microinjections of AngII cause significant inhibition of the cardiac component of the baroreceptor reflex. However there is very little information on its effects on the non-cardiac sympathetic component, which is of critical importance in the regulation of blood pressure. Experiments were carried out in adult male Sprague-Dawley rats that were initially anaesthetised with pentobarbital sodium (60mg/kg, I.P.) and maintained by I.V. infusion of pentobarbital sodium (6mg/ml at 1-1.3ml/hr). The arterial pressure, heart rate (HR) and renal sympathetic nerve activity (RSN) were measured. The baroreceptors were stimulated by increases in mean arterial pressure (MAP) of 40-50 mmHg induced by a single bolus injection of phenylephrine, and the reflex decrease in HR and RSN measured.

Following bilateral microinjections of 40pmol AngII (50nl) into the NTS, the gain of the cardiac component of the reflex (measured as $\Delta HR/\Delta MAP$) was greatly reduced by 70 ± 8.7% compared with that before Ang II microinjections. In contrast, bilateral microinjections of AngII into the NTS had no significant effect (change of 1.4 ± 5.5%) on the gain of the renal sympathetic component of the reflex (measured as $\Delta RSNA/\Delta MAP$). In control experiments, bilateral microinjections of the vehicle solution into the NTS had no effect on the gain of either the cardiac or renal sympathetic component of the reflex.

We conclude from these experiments that the inhibitory influence of AngII microinjections may be restricted to the cardiac component of the baroreflex. It is possible that in conditions where endogenous AngII activity is increased (e.g. hypertension or heart failure) the cardiac reflex response to changes in blood pressure may be inhibited, whereas the baroreceptor mediated vasomotor response is largely unaffected.