Muscle–nerve sympathetic activity in man. Relationship to blood pressure in resting normo- and hyper-tensive subjects

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Summary

1. Simultaneous recordings of multi-unit muscle nerve sympathetic activity and arterial blood pressure were made in 29 subjects, 17 healthy and 12 hypertensive. The neural activity, quantified by counting the number of pulse-synchronous sympathetic bursts in the mean voltage neurogram (burst incidence), was plotted against blood pressure. The effect of spontaneous temporary blood pressure fluctuations was studied by correlating different pressure parameters of individual heart beats to the occurrence of a sympathetic burst.

2. Between subjects there were marked differences in burst incidence but no correlation was found to interindividual differences in blood pressure level.

3. When for each heart beat the occurrence of a burst was correlated to different pressure parameters there was a close negative correlation to diastolic, a low correlation to systolic, and an intermediate negative correlation to mean blood pressure.

4. In a given subject, when comparing heart beats with the same diastolic pressure, the occurrence and the amplitudes of the sympathetic bursts were higher during falling than during rising pressure. This directional dependence of the muscle–nerve sympathetic activity was slightly more pronounced in the hypertensive group, but this was considered secondary to the hypertension.

5. The findings of an intimate correlation with dynamic variations in blood pressure and the absence of correlation to the static blood pressure suggest that the sympathetic outflow to skeletal muscle is of importance for buffering acute blood pressure changes but has little influence on the long-term blood pressure.

Key words: blood pressure, burst incidence, muscle–nerve sympathetic activity.

Introduction

In a previous study no qualitative differences were found between the sympathetic outflow to the extremities of normo- and hyper-tensive subjects (Wallin, Delius & Hagbarth, 1973). Recently methods have been developed to quantitate both the amount of sympathetic activity in human muscle nerves and its relationship to the arterial blood pressure (Sundlöf & Wallin, 1977, 1978). In the present report these results will be compared with similar data obtained from recordings of muscle–nerve sympathetic activity in patients with arterial hypertension.

Material and methods

The comparison was based on simultaneous recordings of muscle–nerve sympathetic activity and intra-arterial blood pressure in 17 healthy, normotensive volunteer subjects, aged 18–54 years, and 12 hypertensive patients, aged 18–58 years. The investigation was made at rest in the recumbent posture. After a thorough clinical investigation eight patients were considered having essential, two borderline and two renovascular hypertension. Most of them were classified as WHO stage I. The nerve recordings were made with tungsten micro-electrodes from muscle.
branches of the peroneal nerve at the fibular head or the median nerve at the elbow level. Blood pressure was monitored via a catheter in the brachial artery. The recording technique and the methods of identifying sympathetic activity were the same as described previously (Wallin et al., 1973; Sundlöf & Wallin, 1977, 1978). During the experiment both original and mean voltage (time constant 0.1 s) neurograms were stored together with other variables on a tape-recorder. For analysis the signals were fed into a computer and the records were divided into 3 min periods. For each period the amount of activity was determined by counting the number of pulse-synchronous bursts and their amplitudes in the mean voltage neurogram. The number of bursts was expressed as burst incidence, i.e. number of bursts/100 heart beats. For each rest period the computer also calculated systolic, diastolic, pulse and mean blood pressure. For further details of the methods of analysis see Sundlöf & Wallin (1978).

Results

Relationship between sympathetic activity and transient blood pressure variations

Although there were marked interindividual differences in the incidence of pulse-synchronous bursts there was always an inverse relationship between the occurrence of bursts and spontaneous blood pressure fluctuations, so that bursts tended to occur during reductions and tended to disappear during transient increases of blood pressure. This relationship was quantified by correlating different blood pressure parameters of individual heart cycles to the probability of occurrence of a sympathetic burst. The experimental points of these threshold variability diagrams were fitted to straight lines and correlation coefficients were calculated. A close negative correlation to diastolic blood pressure was regularly found implying that low diastolic pressures usually and high diastolic pressures rarely were associated with a burst. To systolic blood pressure, on the other hand, there was no systematic correlation and to mean blood pressure an intermediate negative correlation was found. For the hypertensive subjects the threshold variability diagrams were situated higher up on the blood pressure axis, but there was no difference in the degree of correlation between normal and hypertensive subjects. Mean correlation coefficients for the whole material (calculated from 255 rest periods in 29 subjects) were −0.80 for diastolic, −0.69 for mean and −0.26 for systolic pressures. The corresponding value for pulse interval was +0.72.

In addition to burst incidence, burst amplitudes also increased during temporary blood pressure reductions and the quantitative analysis showed a close negative correlation between burst amplitude and diastolic blood pressure but a low correlation to systolic pressures. Again there were no differences in the degree of correlation between normotensive and hypertensive subjects.

Direction of the blood pressure changes

In some recordings, especially if there were pronounced regular blood pressure fluctuations, it was apparent that sympathetic bursts occurred more frequently during the periods of falling than during rising pressure. To test whether the difference was systematic or not each rest period was divided into two fractions, one consisting of heart beats preceded by beats with lower diastolic pressure (rising pressure) and one of the beats preceded by beats with higher diastolic pressure (falling pressure). In virtually every rest period burst incidence and mean burst amplitude was higher during the fraction of falling pressure and this could not be explained by the small differences in blood pressure between the fractions. Consequently the analysis confirmed that for a given diastolic blood pressure stronger sympathetic activity is likely to occur if pressure is falling than if it is rising.

The degree of directional dependence was quantified in terms of the difference of diastolic blood pressure between the fractions that would be expected to give the observed difference in burst incidence or burst amplitude. When comparing normo- and hyper-tensive subjects the degree of directional dependence was slightly greater in the hypertensive group both when calculations were based on burst incidence and burst amplitude (6.5 against 4.8 mmHg, P < 0.01, based on burst incidence and 8.5 against 4.9 mmHg, P < 0.01, based on burst amplitude).

Relationship between mean burst incidence and the static blood pressure

For each subject mean burst incidence and mean values of different blood pressure parameters were calculated for the whole experiment. Marked interindividual differences were seen both for burst...
incidence and blood pressure, but no correlation was found between the two measurements and there was no significant difference in mean burst incidence between normo- and hyper-tensive subjects.

Discussion

An important finding in the present study is the lack of significant relationship between the static blood pressure and the 'level' of muscle–nerve sympathetic activity. Consequently there is no indication that the elevated blood pressure in hypertension is maintained by a permanently increased strength of the sympathetic outflow to the muscles.

The dynamic interplay between transient variations in nerve activity and diastolic blood pressure (indicative of baroreflex modulation of the muscle–nerve sympathetic activity) was qualitatively similar both in normo- and hypertensive subjects. In the hypertensive subjects, however, the fluctuations of nerve activity occurred at higher diastolic blood pressure levels, corresponding to the well-known resetting of the baroreflex operating range secondary to the hypertension (cf. McCubbin, Green & Page, 1956). The slight increase in the degree of directional dependency of the nerve activity seen in the present study may also be secondary to the hypertension. In animal studies it has been well established that, at a given blood pressure, arterial baroreceptors discharge more if pressure is rising than if it is falling. Angell-James (1973) found this hysteresis to be more pronounced in rabbits with experimental hypertension than in normotensive controls and if the situation is similar in man it could explain our results.

In conclusion, the present findings with a lack of correlation between the static blood pressure and the 'level' of muscle–nerve sympathetic activity in the face of an intimate relationship between transient variations in blood pressure and sympathetic activity suggest that the sympathetic outflow to the vascular bed of skeletal muscle is of importance for buffering acute blood pressure changes but has little influence on the long-term blood pressure.

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References


